

Infectious Disease Manual

Infectious Diseases of Concern to Captive and Free Ranging Wildlife in North America

An official publication of the
American Association of Zoo Veterinarians
Animal Health and Welfare Committee

Updated March 2020

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Introduction:

This publication was designed as a starting reference point for information on the infectious diseases that affect zoo and wild animal species captively housed or free-ranging in North America. This volume complements a similar dynamic and routinely-updated version that exists for the same populations in Europe. While each sheet has been peer-reviewed, often by a topic expert, these fact sheets are not intended to be used as an exclusive source of information, but rather provide quick reference of basic disease properties and concerns. These fact sheets also highlight diagnostics, laboratories, specialists, and treatment recommendations for clinicians, pathologists, and wildlife biologists that encounter an infectious disease.

This compendium acts as a common resource and point of information for this discipline.

It is important to remember that these fact sheets are not to replace state or federal regulations. As such, they are not legally enforceable documents or required standards of care.

From the Editors

We hope that you find the information in this manual helpful. There have been some changes made since the last update of the Infectious Disease Manual. For example, the links to US state reportable diseases have been removed as this information is now easily searchable on the internet. We feel that it is impossible to maintain up-to-date information on these topics in such a quickly changing world. We encourage our readers to contact the specialists listed on each factsheet to obtain the most current information about a disease.

We would like to thank the many authors and reviewers who have contributed to this updated edition as well as the previous editions of this manual. We also thank Melanie Pearson, Class of 2021, who assisted with the formatting/technical editing of many of the updates. Without the hard work by so many people, this manual would not be possible.

Please note that we have elected to update this manual in stages so fact sheets have been updated between 2013 through 2020. The date of the most recent update is noted on the first page of each fact sheet. We will continue to update this publication regularly as new updates become available.

If you find any broken links within this document, please let us know so that we can attempt to correct the issue. If you would like to contribute as a new factsheet author, updating author, or reviewer, we would also love to hear from you. We can be reached at drbrockdvm@gmail.com (Paige Brock), GCole@okczoo.org (Gretchen Cole), and sim.richardr@gmail.com (Richard Sim).

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ACANTHAMOEBIASIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
-Primates -Dogs -Sheep -Cattle -Horses -Kangaroos -Birds -Reptiles -Amphibians -Fish -Invertebrates	-Source: soil, water -Gains entry via: breaks in skin; respiratory tract; corneal surface; hematogenous spread to central nervous system	-Cutaneous lesions -Sinusitis -Pneumonitis -Neurologic signs -Fever -Nausea -Vomiting	-Asymptomatic in immunocompetent individuals -Frequently fatal in immunocompromised individuals	Pentamidine isethionate; Sulfadiazine; Flucytosine; Fluconazole; Itraconazole Amphotericin B; Azithromycin	-Difficult due to ubiquitous nature of the organism -Limit exposure to dust, soil, and water	-Not directly transmitted -Can cause disease in humans

Fact Sheet compiled by: Laurie Gage

Sheet completed on: April 14, 2011; updated 19 March 2013.

Fact Sheet Reviewed by: Kimberly Rainwater, Ariana Finkelstein

Susceptible animal groups: Primates, dogs, sheep, cattle, horses, kangaroos, birds, reptiles, amphibians, fish, invertebrates.

Causative organism: Opportunistic protozoan parasites, *Acanthamoeba* spp. (*A. castellanii*, *A. culbertsoni*, *A. hatchetti*, *A. healyi*, *A. polyphaga*, *A. rhysodes*, *A. astronyxis*, *A. divionensis*)

Zoonotic potential: May infect cornea of contact lens wearers and cause disseminated infection in immunocompromised individuals.

Distribution: Ubiquitous worldwide. It may be found in soil; fresh and brackish water; bottled mineral water; cooling towers of electric and nuclear power plants; heating, ventilating, and air conditioning units, humidifiers; Jacuzzi tubs; hydrotherapy pools in hospitals; dental irrigation units; dialysis machines; dust in the air; bacterial, fungal, and mammalian cell cultures; contact lenses and ophthalmic saline flush; aural discharge; pulmonary secretions; feces.

Incubation period: 1 day to 2 weeks

Clinical signs: Granulomatous amoebic encephalitis: depression, nausea, vomiting, low-grade fever, lethargy, cerebellar ataxia, visual disturbances, hemiparesis, cranial nerve deficits, seizures, and coma. Cutaneous lesions: ulcers, nodules, and subcutaneous abscesses. Respiratory: sinusitis and pneumonitis. *Acanthamoeba* keratitis (reported in humans only): ocular pain, photophobia, corneal ulceration, loss of visual acuity, and blindness.

Post mortem, gross, or histologic findings:

Gross Findings: multifocal encephalomalacia and cerebral hemorrhage; nodular necrosis in the liver, kidney, lung, pancreas; multifocal granulomatous pneumonia; cutaneous granulomas

Histologic Findings: Focal areas of necrosis and granulomatous inflammation in affected tissues; necrotizing vasculitis; [resence of cysts (12-16 um diameter) and trophozoites (14-40 um diameter) in affected tissues

Diagnosis: Direct observation of amoebae in tissues stained with hematoxylin-eosin; indirect immunofluorescence staining using rabbit anti-amoeba sera; polymerase chain reaction to detect amoeba DNA in tissue and cerebrospinal fluid samples; computed tomography and magnetic resonance imaging

Material required for laboratory analysis: Serum, cerebrospinal fluid, formalin-fixed tissue samples, fresh tissue samples for culture (culture should only be done by accredited laboratories with the proper safety equipment)

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Relevant diagnostic laboratories: Centers for Disease Control and Prevention, Atlanta, Georgia
Treatment: Pentamidine isethionate, sulfadiazine and other sulfa drugs, flucytosine, fluconazole, itraconazole, amphotericin B, and azithromycin.
Prevention and control: Limit exposure to airborne soil particles that may carry cysts to the respiratory system; prevent exposure of open wounds to contaminated soil or water; preventative measures are especially important for immunocompromised individuals.
Suggested disinfectant for housing facilities: Chlorhexidine, isopropyl alcohol (20%), hydrogen peroxide
Notification Not required
Measures required under the Animal Disease Surveillance Plan None required
Measures required for introducing animals to infected animal None required
Conditions for restoring disease-free status after an outbreak: It is not possible due to ubiquitous nature of this organism.
Experts who may be consulted Centers for Disease Control
References: <ol style="list-style-type: none"> 1. Mehlhorn, H. 2008. Encyclopedia of Parasitology Volume 1, 3rd Ed. Springer-Verlag, New York. Pp: 2. 2. Schuster, F.L., and G.S. Visvesvara. 2004. Amebae and ciliated protozoa as causal agents of waterborne zoonotic disease. Vet. Parasitol. 126: 91-120. 3. Rutala, W.A., D.J. Weber, and the Healthcare Infection Control Practices Advisory Committee. 2008. Guideline for Disinfection and Sterilization in Healthcare Facilities, 2008. http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf. Accessed 14 April 2011. 4. Visvesvara, G.S., H. Moura, and F L. Schuster. 2007. Pathogenic and opportunistic free-living amoebae: <i>Acanthamoeba</i> spp., <i>Balamuthia mandrillaris</i>, <i>Naegleria fowleri</i>, and <i>Sappinia diploidea</i>. FEMS Immunol. Med. Microbiol. 50: 1-26.

ACANTHOCEPHALANS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All vertebrates	Requires ingestion of the intermediate host or a paratenic or transport host	Ill thrift, weight loss, anorexia, diarrhea, abdominal discomfort or colic; clinical signs compatible with peritonitis	Infections can cause clinical disease and mortality. Severity of disease may not be directly correlated with the number of adult parasites present.	There have been treatment difficulties noted in some species. However, ivermectin and doramectin have eliminated <i>Macracanthorhynchus</i> species in dogs and swine. Albendazole has eliminated <i>Moniliformis clarki</i> from cotton-topped tamarins.	Identify presence of infected animals. Removal of intermediate, paratenic, or transport hosts from environment.	Yes, if there is consumption of an intermediate or transport host

Fact Sheet compiled by: Peregrine Wolff

Sheet completed on: updated 9 July 2018

Fact Sheet Reviewed by: Rachel Marschang

Susceptible animal groups: Vertebrate species

Causative organism: Phylum Acanthocephala are commonly known as thorny headed worms and are highly specialized, unsegmented parasites of the digestive tract of mammals, birds, reptiles, amphibians and fish. Acanthocephala are characterized by their large size (up to 65 cm) and the presence of an anterior, retractable proboscis that is covered with rings of recurved hooks arranged in horizontal rows in almost all species. These hooks are used to attach the parasite to the intestinal mucosa of its final host while it completes its life cycle. Acanthocephalans do not possess a digestive tract and absorb all nutrients through their body wall. It is believed that over 1300 species of Acanthocephalan parasites exist. Within the four orders of Acanthocephalans:

- Noechinorhynchidea infect turtles, amphibians and fish.
- Echinorhynchidea infect primarily fish, amphibians, reptiles, birds and aquatic mammals. However, a few species are known to infect terrestrial mammals and birds of prey, such as *Corynosoma* reported in free ranging marine mammals and *C. polymorphus* reported in sea ducks.
- Within the order Aporhynchidea, the species *Apororhynchus* is a parasite of birds.
- Gigantorhynchidea contains families and species that infect mammals (*Macracanthorhynchus* [suids, carnivores], *Prosthenorchis* [primates], *Moniliformis* [rodents], and *Oncicola* [carnivores]) and birds.

Zoonotic potential: Yes. Human reports of infection with acanthocephalans are rare and are associated with ingestion of the intermediate host or the transport or secondary hosts and most commonly associated with the consumption of raw fish.

Distribution: Worldwide distribution in aquatic and terrestrial vertebrate species

Incubation period: Adult females lay eggs which contain a fully developed larva called an acanthor which passes into the environment. If the acanthor is ingested by a suitable insect (e.g., cockroaches can serve as intermediate insect hosts in zoos) or crustacean intermediate host it will first enter the acanthella stage before developing in 6-12 weeks into the infective stage larva or cystacanth, which encysts in the intermediate host.

ACANTHOCEPHALANS

If the intermediate host is ingested by a final host then the cystacanth attaches onto the intestinal lumen and matures into an adult in 8-12 weeks. If the intermediate host is ingested by a transport or paratenic host then the cystacanth will penetrate through the gut and encyst in the tissues or organs of this host only completing its life cycle if the transport host is ingested by a final host.

In experimental infections in swine, *Macracanthorhynchus ingens* worms were found embedded in the mucosa of the intestine 3 days after the pig was fed cystacanths contained in a gelatin capsule.

Clinical signs: Depending upon the host, the species of parasite and the parasite burden there may be sub-clinical to severe clinical signs. Diarrhea, emaciation, restlessness, abdominal pain (often severe) and poor weight gain has been described in pigs infected with *Macracanthorhynchus hirudinaceus*. Diarrhea, emaciation, lethargy, and ill-thrift have been noted in primates infected with *Prosthenorchis elegans*. If the parasite penetrates the serosa of the intestines then clinical signs consistent with peritonitis may be evident. Among birds, ducks, geese, swans, birds of prey, and some species of passerines are most commonly infected. Severe disease outbreaks have been reported in common eiders. All age classes can become infected. Acanthocephalans are actively being studied as bio-indicators of pollution. Parasites in fish have been found to have concentrations of heavy metals orders of magnitude greater than either the tissues of their hosts or the surrounding water. If captive species were fed fish that were heavily parasitized by acanthocephalans with high concentrations of heavy metals perhaps the potential for toxicity exists.

Post mortem, gross, or histologic findings: Attachment sites of the adult worms may be visible on the serosal surface of the intestine as circular flat areas of discoloration or as raised, firm, white nodules. The proboscis may penetrate the mucosa, submucosa, muscularis and serosal layers of the intestine with the body of the worm protruding into the lumen. Peritonitis occurs secondary to penetration of the parasite through the wall of the small intestine. Histological changes at the intestinal site of penetration include thickening of the submucosa, muscularis and serosa resulting from cellular infiltration and inflammatory exudates. Intestinal villi at and immediately surrounding the site of attachment may be absent, the cellular architecture may be disrupted and accompanied by leukocytic infiltration. Some species of Acanthocephala may move around the intestine prior to settling on a final attachment site. Gross and histological evidence of worm attachment without the presence of a parasite may be evident. Some individuals can have worm burdens numbering from hundreds to thousands. It is believed that this volume of large parasites within the lumen may cause mechanical blockage contributing to starvation of the host.

Diagnosis: Identification of adults often is based on the pattern of hooks on the proboscis, thus it is important that this portion of the worm is preserved and visible. If required for identification, and no adults are free floating, then the worm should be carefully removed from its attachment site within the intestine and placed in water which creates an osmotic turgor forcing the proboscis to evert. The worm is then fixed in warm alcohol-formaldehyde-acetic acid (AFA) a preservative which consists of 85 parts 85% ethanol, 10 parts stock formalin, and 5 parts glacial acetic acid.

Acanthocephalan eggs are large and heavy thus fecal sedimentation techniques utilizing formalin-ethyl acetate are felt to be superior to flotation techniques for identifying acanthocephalan eggs. The eggs are elongated with a thick outer wall and thin inner walls, often appearing to have 3 layers. Within the egg lies the acanthor larva. If the spines at one end of the larva are visible then a positive identification of acanthocephalan can be made. Although when laid by the female the eggs are clear, eggs of some species will appear brown due to fecal staining as they pass along of the intestinal tract of the host.

Material required for laboratory analysis: Feces to determine presence of eggs are needed and fecal centrifugation techniques are preferred. The whole parasite is necessary for species identification. Sections of the intestine with the parasite within the lumen or embedded within the wall to determine degree of pathology associated with the attachment site.

Relevant diagnostic laboratories: Before submission, confirm with your diagnostic laboratory that they can key out species of adult parasites.

ACANTHOCEPHALANS

Treatment: Albendazole (50 mg/kg b.i.d. x 16 days or 100 mg/kg b.i.d x 3 days and then repeated biweekly for a total of 4 treatments) has been used to successfully treat captive cotton-topped tamarins (*Saguinus oedipus*) infected with *Moniliformis clarki*. Swine and dogs have been successfully treated for *Macracanthorhynchus* species with ivermectin and doramectin at dosages typical for the species. Surgical removal of adult *Prosthenorchis elegans* from captive marmoset and tamarin species has also been an effective treatment.

Prevention and control: All incoming animals should be quarantined and receive comprehensive fecal exams. Animals infected with Acanthocephala should be treated. Species should not have the opportunity to infect or ingest possible intermediate or transport hosts that could be infected with acanthocephalan species. Although one species may shed the parasite, another taxonomically unrelated species (accidental host) may ingest the intermediate host and become infected. This transfer can have implications for mixed species exhibits and implementation of an effective pest control program.

Suggested disinfectant for housing facilities: Removal of intermediate host – pest control

Notification: None required

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Introductions should not be done unless no opportunity for introduction of a suitable intermediate host exists.

Conditions for restoring disease-free status after an outbreak: It is necessary to break the life cycle and remove possible intermediate hosts.

Experts who may be consulted: Most research regarding acanthocephalans revolves around emerging human infections and identification of new species in new hosts. Parasitologists at each veterinary university diagnostic laboratory can be consulted for acanthocephalan identification.

References:

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WOODEN TONGUE (*Actinobacillus lignieresii*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primarily cattle, but also sheep, horses, pigs, dogs and rarely chickens.	Normal oropharyngeal and rumen flora enters tissues through epithelial damage.	Granulomas of tongue and lymph nodes of head and neck.	Variable; life threatening without treatment.	Surgical debridement, systemic sodium iodide and long-term antibiotics	Early recognition and treatment, isolation of affected animals.	No

Fact Sheet compiled by: Amanda Guthrie

Sheet completed on: 16 March 2011; updated 5 April 2013, updated 2018

Fact Sheet Reviewed by: Mark Drew and Nancy Carpenter

Susceptible animal groups: Most commonly cattle are affected, but can infect sheep, horses, pigs, dogs and rarely chickens.

Causative organism: *Actinobacillus lignieresii*; Gram-negative coccobacillus.

Zoonotic potential: No

Distribution: Occurs sporadically worldwide, preferentially in areas with copper deficiency or pasture with abrasive weeds.

Incubation period: Unknown

Clinical signs: Disease mainly affects the tongue and the lymph nodes of the head and neck. Characteristic lesion is pyogranulomatous inflammation of the tongue with purulent discharge. Inability to eat or drink may be noticed, as well as excess salivation, rapid weight loss, and painful and swollen ulcerated tongue. With chronicity, the tongue becomes fibrous, shrunken and immobile. Draining lymph nodes in this area may become enlarged and abscessed with purulent discharge, rarely granulomas can form in and around the jaw, lungs, esophagus, udder, skin or internal organs. Sheep frequently are affected by purulent granulomas of the face, lips, nose, jaw, and neck.

Post mortem, gross, or histologic findings: Poor body condition, pyogranulomatous lesions containing pus in and around the mouth. Oral ulcers and encapsulated abscesses of the local lymph nodes may be noted. In chronic cases, fibrous connective tissue proliferation of the tongue can be observed.

Diagnosis: Reasonable suspicion based on clinical signs and it may be confirmed with microscopic exam of cytological specimens or by direct culture. Purulent discharge contains small brown-white granules which consist of colonies of Gram-negative rod-shaped bacteria.

Material required for laboratory analysis: Smears of pus, fine needle aspirate samples.

Relevant diagnostic laboratories:

Kansas State University Veterinary Diagnostic Lab
 1800 Denison Avenue
 Manhattan, KS 66506
 Phone: 866-512-5650
 Fax: 785-532-4481
 dlaboffice@vet.k-state.edu
<http://www.vet.k-state.edu/depts/dmp/service/>

Treatment: Surgical debridement, systemic sodium iodide – which is not labeled for use in food animals; call FARAD about withdrawal times, and antibiotics. Streptomycin is considered the antibiotic of choice; also tetracyclines, erythromycin and tilmicosin are effective but require extended duration.

WOODEN TONGUE (*Actinobacillus lignieresii*)

Prevention and control: Early recognition and treatment and isolation of affected animals is critical. Animals with weeping lesions should be isolated and areas should be cleaned routinely as these bacteria only survive for a few days in the environment. Low-quality dry stalky feed, grass seeds, coarse hay and scrub can predispose to disease by causing oral abrasions. Tooth eruption may also allow for entry of bacterium into oral epithelium.

Suggested disinfectant for housing facilities: Routine disinfection as organisms only live for a few days outside of an animal host.

Notification: This disease is not reportable.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: None, this organism is normal gastrointestinal flora; it is not considered very contagious.

Conditions for restoring disease-free status after an outbreak: None.

Experts who may be consulted:

Kansas State University Veterinary Diagnostic Laboratory
1800 Denison Avenue
Manhattan, KS 66506
866-512-5650

References:

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ACTINOMYCOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Many mammal species, including humans; birds	Endogenous infection into susceptible tissues or by bite wound	Local abscesses, chronic draining fistulas, bony infections, or infections of body cavities. Clinical signs referable to the involved area	Can be mild if restricted to local infection but can be fatal depending on infection location, spread, and time to diagnosis.	Surgical drainage and debridement. Appropriate antibiotic therapy continued for several weeks after elimination of clinical signs.	Ensure good oral care. Limit the amount of rough forage fed and limit the number of plant awns in environment.	Yes, but most human infections are endogenous.

Fact Sheet compiled by: Rebecca Bloch

Sheet completed on: 24 June 2011; updated 21 December 2012

Fact Sheet Reviewed by: Amy Swinford, John Gilliam

Susceptible animal groups: Horses, cattle, small carnivores, goats, sheep, wild ruminants, monkeys, rabbits, squirrels, hamsters, marsupials, humans, river otter, and birds

Causative organism: *Actinomyces* spp. including *A. bovis*, *A. hodeovulneris*, *A. israelii*, *A. naeslundii*, *A. pyogenes*, *A. suis*, and *A. viscosus*. These organisms are anaerobic to microaerophilic, Gram positive, rod shaped bacteria that may produce branching filaments. The disease process termed lumpy jaw has many bacterial agents that include *Fusobacterium necrophorum*, *Bacteroides*, *Streptococcus*, *Staphylococcus*, *Corynebacterium*, *Nocardia*, and *Actinobacillus* in addition to *Actinomyces* species. *A. bovis* has been stated as being the causative agent of lumpy jaw in cattle but it has also been suggested that *Actinomyces* infection in exotic bovids is secondary to a tooth root infection, rather than a primary infection (J. Oosterhuis personal communication). If the mandible undergoes a traumatic incident that interrupts the blood supply to a tooth causing it to become devitalized, this damage leads to necrosis and then secondary invasion by various bacteria, including *Actinomyces* species.

Zoonotic potential: Generally, the disease is not contagious except via bite wounds. The only suggested documented zoonotic infection in the literature was caused by *Actinomyces pyogenes*, since reclassified as *Arcanobacterium pyogenes*.

Distribution: Normal flora of the oral and nasopharyngeal membranes. This species is secondarily found in the gastrointestinal tract. In humans, these organisms are also found in the female genitourinary tract.

Incubation period: This organism requires 24-48h for growth in media but infections are endogenous and require introduction of the bacteria into susceptible tissue to initiate infection generally through tissue trauma or, less frequently, through bite wound.

Clinical signs: Lesions include localized abscesses, chronic draining fistulas, bone infections, or infections of body cavities. Drainage from the lesions is serosanguinous and often contains small yellow granules. Infection may be associated with fever. Clinical signs are referable to the area of involvement. In cattle, humans, and marsupials, *Actinomyces* sp. associated with osteomyelitis is characterized by dislodgement of teeth, inability to chew, and mandibular fractures. In several hosts, this bacterium can cause soft tissue infections. In horses, it may manifest as supra-atlantal or supraspinous bursitis, or sometimes cervical abscesses. *Actinomyces* endophthalmitis has been documented in a dog. *Actinomyces* spp. has been associated with plant awn foreign

ACTINOMYCOSIS

bodies and associated disease such as discospondylitis, in small carnivores. In humans, in addition to other sites of infection, actinomycosis can be associated with contraceptive intrauterine devices.

A. bovis is associated with osteomyelitis in cattle, typically causing formation of periosteal new bone and fibrosis in the mandible, most commonly on the horizontal ramus. It can occasionally cause granulomatous abscesses in the soft tissues of the head, esophagus, forestomachs, and trachea.

A. actinoides is occasionally found in enzootic pneumonia of calves and seminal vasculitis in bulls.

A. hordoevulneris causes localized abscesses and systemic infections such as pleuritis, peritonitis, visceral abscesses, and septic arthritis in dogs. Infection is associated with migrating plant awns.

A. israelii is associated with chronic granulomatous infections in humans but has rarely been isolated from pyogranulomatous lesions in pigs and cattle.

A. neslundii has been isolated from suppurative infections in several animal species, the most common being aborted porcine fetuses.

A. pyogenes (currently *Arcanobacter pyogenes*) is associated with infections in many organ systems in many species of animals. Infections include suppurative mastitis, suppurative pneumonia, septicemia, vegetative endocarditis, endometritis, intracranial abscesses or suppurative meningoencephalitis, septic arthritis, wound infections, and liver abscesses.

A. suis causes pyogranulomatous porcine mastitis. Chronic, deep seated abscesses may fistulate.

A. viscosus causes chronic pneumonia, pyothorax, and localized subcutaneous abscesses in dogs. Thoracic lesions are pyogranulomatous while cutaneous lesions are granulomatous abscesses, often with fistulous tracts. Lesions generally develop after a traumatic injury such as a bite wound.

A. denticolens has been reported to cause mandibular lymphadenopathy in horses with possible fever, nasal discharge, and depression, making it clinically similar to strangles.

Post mortem, gross, or histologic findings: Aggregates of Gram positive, filamentous, non-acid-fast bacteria with associated inflammation in the areas of infection. While it is possible to detect *Actinomyces* sp in tissue sections stained with hematoxylin and eosin (sulfur granules are round or oval basophilic masses with a radiating arrangement of eosinophilic terminal clubs), special stains such as Gomori methenamine silver, paminosalicylic acid, McCallen-Goodpasture, and Brown-Benn may be needed.

Diagnosis: Grossly, yellowish particles up to several millimeters in diameter in the lesions or tissue may be observed. These particles, called sulfur granules, are suggestive of *Actinomyces* infection but can also be seen with other types of bacteria (*Nocardia* sp.). In the case of *A. viscosus* infection soft, grayish white granules may be seen in the pus or exudate. Clinical presentation, Gram stain, and histopathologic visualization of the bacteria and granules are supportive of the diagnosis. Definitive diagnosis requires culture but is not always possible as this group of organisms is sometimes difficult to grow.

Material required for laboratory analysis: Culture swab or tissue sample from the affected area.

Relevant diagnostic laboratories: Any laboratory capable of running bacterial cultures should be able to culture this organism. Although most strains do not require anaerobic incubation, they do benefit from increased carbon dioxide concentration.

Treatment: Appropriate surgical drainage or debridement in addition to antimicrobial administration. Iodine compounds, penicillin, and isoniazid have been used to treat bovine cases. In small carnivores, a penicillin derivative is the drug of choice but penicillins have difficulty penetrating pyogranulomatous lesions which may necessitate prolonged therapy. Chloramphenicol and clindamycin can also be used. Antibiotic impregnated beads have been used in the treatment of bone infections. A published suggestion for treatment of jaw osteomyelitis includes surgical debridement of the lesion followed by surgical fistulation to allow lavage with sterile water, hydrogen peroxide, 5.25% sodium hypochlorite, and then 2% Betadine. Once infection is eliminated or contained, surgical repair of the tooth and bony defect can occur.

Prevention and control: Reduce feeding of rough or excessively fibrous plant material that might cause

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trauma to the oral cavity. Reduce any environmental stressors such as overcrowding. Good oral care to help prevent food impaction or entry of bacteria in dental caries.

Suggested disinfectant for housing facilities: Since *Actinomyces* sp. are normal flora and generally found in the oral cavity of the animals they effect, environmental decontamination of the environment has less importance. However, the bacteria can reside in the environment in organic material and these organisms can be removed through thorough cleaning of any organic material from the environment followed by disinfection with 10% bleach or any of the commercially available disinfectants mixed to manufacturer's instructions.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Since most infections are endogenous, unless the infected animal is likely to bite another animal, no special measures beyond individual health care need to be taken.

Conditions for restoring disease-free status after an outbreak: Special attention should be paid to husbandry practices and oral care in the animals of concern.

Experts who may be consulted:

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 P.O. Drawer 3040
 College Station, TX 77841
 aswinford@tvmidl.tamu.edu
 979-845-3414

References:

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ADENOVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals Birds Reptiles	Vertical. Direct contact. Fecal-oral. Venereal.	Mammal: respiratory, conjunctivitis, liver disease, gastro-enteritis. Avian: abnormal eggs and production, respiratory disease. Reptile: none to “poor doer” to unexplained death.	Variable: asymptomatic to death. Usually disease is sporadic and limited to the young and immune-compromised	Supportive/ symptomatic	Vaccination (carnivores); however, this approach is not common. One case of vaccine-induced disease.	No as virus is highly host specific.

Fact Sheet compiled by: Natalie D. Mylniczenko

Sheet completed on: 29 January 2011; 6 October 2012; update 19 April 2018

Fact Sheet Reviewed by: Erika Travis-Crook

Susceptible animal groups:Mammals:

Infectious canine hepatitis (canine adenovirus 1): foxes, wolves, ferrets, raccoons, skunks, ursids (black bears); Eurasian otter (n=1), hedgehog (n=1) and other small carnivores

Adenoviral hemorrhagic disease virus: cervids

Acute hepatic necrosis: California sea lion (CSLAdV-1)

Dolphin:

Simian adenoviruses: 1-30 (oncogenic and non-oncogenic): SA8 baboons (infants-pneumonia)

Birds:

Many avian adenoviruses (quail bronchitis virus, hemorrhagic enteritis, etc.)

Egg drop syndrome/duck adenovirus A: chickens, ducks, quail

Pigeon adenovirus

Reptiles:

Agamid adenovirus 1: bearded dragon

Snake and lizard adenoviruses

Amphibian and fish have adenoviruses that are of no known clinical significance at this time.

Causative organism: Adenovirus. Non-enveloped DNA virus.

Zoonotic potential: None

Distribution: Worldwide

Incubation period: Mammal 8-10d, avian 3-4d for respiratory, 10-24 for egg production.

Clinical signs: Mammal: “blue eye”, young animals, nonspecific gastrointestinal signs. Course is typically peracute or acute.

Birds: young birds, respiratory disease; change in egg quality/production

Reptiles: none to chronic ‘poor doer’ to death

Post mortem, gross, or histologic findings: respiratory, gastrointestinal and ocular systems typically affected.

Mammals:

Carnivores: hemorrhage of stomach and serosal surfaces (coagulation impairment), hepatic congestion and hepatomegaly. Focal hepatic necrosis.

Cervids: pulmonary edema and hemorrhagic enteropathy.

Birds: enteritis, splenitis (marble spleen disease), hepatitis, bronchitis, pulmonary congestion

Reptiles: enteritis and hepatitis, rarely encephalitis and esophagitis.

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Diagnosis:

Mammals: Antemortem is difficult since signs are non-specific and rapid. Virus isolation, immunofluorescence, or characteristic intranuclear inclusion bodies in the liver or other lesions. Serology is available.

Birds: serology, agar gel immunodiffusion, fluorescent antibody, virus isolation

Reptiles: polymerase chain reaction or electron microscopy

Material required for laboratory analysis:

Mammals: serum, swabs, tissue from liver and lung

Birds: serum, tissue from lesions

Reptiles: tissues from lesions or cloacal/fecal swabs

Relevant diagnostic laboratories:

Mammals:

Mammalian tests (bovine, canine, equine, porcine, llama)-many veterinary diagnostic labs

Primate: <http://www.vrl.net/>

<http://zoologix.com/primate/index.htm>

Birds:

National Veterinary Services Laboratories

USDA-APHIS-VS-NVSL

Avian Viruses Section

Head: Dr. Mia Kim Torchetti

Phone: (515) 337-7551

E-mail: mia.kim.torchetti@aphis.usda.gov

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/lab-info-services/sa_diagnostic_tests/ct_diagnostic_tests

Charles River: Avian Vaccine Services

<https://www.criver.com/products-services/avian-vaccine-services>

Penn State Animal Diagnostic Laboratory

Wiley Lane

University Park, PA 16802

Phone: 814-863-0837

Fax: 814-865-3907

adlhelp@psu.edu

<http://vbs.psu.edu/facilities/adl/services/tests/avian-virology>

Reptiles:

Zoo Medicine Infectious Disease Lab

University of Florida

2015 SW 16th Ave.

Building 1017 Room V2-238

Gainesville, FL 32610

Contact: April Childress

Phone: 352-294-4420

childressa@ufl.edu

<http://labs.vetmed.ufl.edu/sample-requirements/microbiology-parasitology-serology/zoo-med-infections/>

Treatment: Supportive/symptomatic.

Prevention and control: Vaccination (carnivores)-modified live vaccine for canids.

ADENOVIRUS

Virus shed in urine, nasal and conjunctival secretions, and feces.
 Virus persists in the kidney and may be shed for months after recovery.
 Do not translocate cervids from affected areas to non-endemic areas.

Suggested disinfectant for housing facilities: Adenoviruses are very stable in the environment but are susceptible to 1% sodium hypochlorite, 2% glutaraldehyde and quaternary ammonium compounds.

Notification: Some states require notification with birds and deer.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Vaccination prior to introduction should be considered; however, incidence rate in most species is low. Animals may shed virus in urine and other secretions for up to 6 months.

Conditions for restoring disease-free status after an outbreak: Outbreaks are not typical.

Experts who may be consulted: See Diagnostic Laboratories.

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AEROMONAS INFECTIONS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Fish, Amphibians, Reptiles, Waterfowl, Marine mammals	Horizontal transmission, close contact with infected individual, ingestion of bacterium, direct inoculation through wounds, especially from contaminated water and detritus. Snake mite (<i>Ophionyssus natricis</i>) capable of transmitting bacteria	Acute mortality, dermal hyperemia, skin wounds, pustular dermatitis, stomatitis, fasciitis, muscle cavitations, pneumonia, gastro-intestinal disease	Mild to severe depending on immune status, and route of infection	Antibiotics, appropriate wound management, supportive care	Ubiquitous in environment and may comprise part of the normal intestinal flora. Opportunistic infection. Prevention through good environmental and personal hygiene practices, optimal husbandry, UV irradiation or ozonation of water; vaccination for <i>Aeromonas salmonicida</i>	Yes

Fact Sheet compiled by: Douglas P. Whiteside

Sheet completed on: 31 March 2011; updated 17 July 2013; Updated 27 January 2018

Fact Sheet Reviewed by: Karen Liljebjelke, Stephen Raverty

Susceptible animal groups: Fish (especially salmonids, goldfish, carp), amphibians, reptiles, waterfowl, marine mammals. Occasionally isolated from invertebrates.

Causative organism: *Aeromonas hydrophila*, *A. salmonicida*, *A. shigelloides*, *A. formicans*, *A. sobria*, and *Aeromonas* sp. of which of the at least seven recognized species, four of which are considered more pathogenic. It is a Gram negative, oxidative positive, facultative anaerobic, polar flagellated bacterial rod.

Zoonotic potential: Yes, but also direct environmental exposure. Opportunistic zoonotic pathogen especially in immunocompromised or debilitated individuals.

Distribution: Worldwide distribution. Common in fresh, brackish and salt water environments, particularly with increased detritus or sewage and carried by some invertebrate and vertebrate species.

Incubation period: 24-48 hours

Clinical signs:

Fish: Acute mortality, septicemia, erythema, exophthalmia, hemorrhages in skin, fins, muscle and oral cavity, with skin boils and ulceration. Fecal casts or bloody discharge from vents

Amphibians: Acute mortality, septicemia, anorexia, ventral erythema with cutaneous hemorrhage especially ventral thighs, edema in subcutis, anasarca, hemorrhagic ulcerations of digit tips and jaw. May feature digital amputation due to vasoconstriction, secondary to septicemia.

Reptiles: Acute mortality, septicemia, pneumonia, ulcerative stomatitis particularly in snakes, dermal ecchymoses, epidermal ulceration, anorexia, listless, labored respirations, harsh respiratory sounds, mouth gaping, steady decline in status, rule out predisposing or underlying environmental or host factors.

Waterfowl: Upper respiratory tract infections, salpingitis, enteritis, septicemia, localized abscessation, and arthritis

Marine mammals: Septicemia, pneumonia

Humans: Gastroenteritis, watery diarrhea which can be chronic in nature, septicemia, pustular dermatitis, cellulitis, necrotizing fasciitis, pneumonia, peritonitis, cholecystitis, bacteremia and hepatitis.

Post mortem, gross, or histologic findings

Fish: Cavitating dermal ulceration, furuncles and myositis, exophthalmus, serosanguineous ascites commonly observed. Splenomegaly and swollen kidneys are common. Multifocal areas of necrosis and hemorrhage in

AEROMONAS INFECTIONS

the spleen, liver, kidney and heart with numerous bacilli. Punctate colonies of extracellular bacteria and a lack of associated inflammatory infiltrate are hallmarks of *A salmonicida* infections in salmonids. Carp erythrodermatitis.

Amphibians: Ventral erythema, hepatosplenomegaly, ascites, anasarca, pulmonary congestion, petechiae and ecchymoses in skeletal muscle, coelomic serosa, kidneys, and spleen

Reptiles: Dermal hyperemia, ulceration, stomatitis, hepatomegaly, exudates in trachea and lungs, ascites, splenomegaly, renomegaly, intestinal edema

Waterfowl: Salpingitis, peritonitis, arthritis, or septicemia

Marine mammals: Severe pneumonia, septicemia, ulcerative dermatitis

Humans: Pustular dermatitis, cellulitis, necrotizing fasciitis, osteomyelitis, pyomyositis, pneumonia, bacteremia, peritonitis and meningitis

Diagnosis: Isolation on routine media (heart infusion agar, blood agar, MacConkey, Tryptone soya agar) with subsequent identification, commercial systems, molecular identification (amplified fragment length polymorphism (AFLP) analysis). Results must be taken in context of clinical signs and pathologic findings.

Material required for laboratory analysis: Transport media (Cary-Blair medium is most suitable). Transport at room/environmental temperature yields greatest recovery.

Relevant diagnostic laboratories: Any laboratory that can perform bacteriological isolation, identification, and antimicrobial resistance.

Treatment: Antibiotic selection is dependent on susceptibility testing. In general, these bacteria are susceptible to aminoglycosides, carbapenems, extended spectrum cephalosporins, azithromycin, monobactams, nitrofurans, extended spectrum penicillins (piperacillin, piperacillin-tazobactam), phenicols, fluoroquinolones, and tetracyclines, with variable susceptibility to potentiated antifolates (trimethoprim-sulfas).

Aeromonas spp. produce strong *B*-lactamases, so they resistant to narrow spectrum penicillins (e.g. amoxicillin, ampicillin, ampicillin-sulbactam, ticarcillin, oxacillin, penicillin) and cephalosporins (e.g. cefoxitin), sulfamethoxazole, erythromycin, and clarithromycin.

Prevention and control: In species other than fish, infections are often opportunistic or secondary to debilitation or immunosuppression. Maintain good environmental hygiene, water quality and optimal husbandry conditions; ultraviolet irradiation or ozonation of water sources; proper food storage and follow safe cooking and thawing recommendation; follow all wound care procedures recommended by veterinarian or physician; practice good hygiene; wash hands often.

Suggested disinfectant for housing facilities: UV irradiation or ozonation of water sources is possible. Most disinfectants are effective such as sodium hypochlorite, chlorhexidine, quaternary ammonium products, phenolics, accelerated hydrogen peroxide, Virkon®

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Ubiquitous and opportunistic bacteria. Good quarantine procedures. Tank water to discharge or if recirculating, ensure appropriate treatment and disinfection. Ideally isolate infected animals for treatment.

Conditions for restoring disease-free status after an outbreak: Environmental hygiene, povidone iodine disinfection of fish eggs.

Experts who may be consulted:

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 University of Florida IFAS Extension
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 Gainesville, FL 32608-0136
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***AEROMONAS* INFECTIONS**

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AFRICAN HORSE SICKNESS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Equidae, carnivores, camel	Infectious, non-contagious vector borne disease transmitted by <i>Culicoides</i> midge; mechanical transmission by biting flies is possible; ingestion of virus infected meat	Respiratory form: fever, dyspnea, nasal discharge, conjunctivitis. Cardiac form: fever, swollen head and neck, colic. Mixed form: combination of respiratory and cardiac form signs. Fever form: fever	Mortality depends on serotype and species affected - most severe in horses and mules, typically acute or sub-acute illness with high morbidity and mortality of respiratory, cardiac and mixed forms, fever form is mild with no mortality	No effective treatment but supportive care warranted	Vaccinate, reduce exposure to vector, test and quarantine prior to importation	No

Fact Sheet compiled by: Priscilla H. Joyner

Sheet completed on: 31 March 2011; updated 1 February 2018

Fact Sheet reviewed by: John Sykes

Susceptible animal groups: Equidae, carnivores. Horses are highly susceptible with a mortality rate as high as 95% with per-acute disease, while mules are less susceptible. African donkeys, zebra, elephants and camels are generally resistant to disease. Antibodies to all 9 serotypes have been reported in elephants and zebra. Dogs are susceptible to disease if they ingest virus infected meat. Wild African carnivores are less susceptible.

Causative organism: Orbivirus of the family Reoviridae including 9 serotypes (1-9)

Zoonotic potential: No. However vaccine strains have caused encephalitis and retinitis in humans following trans-nasal transmission

Distribution: Endemic in Africa with outbreaks reported in the Middle East and Europe. Dependent on climatic factors favoring the *Culicoides* vector such as warm, humid weather and high rainfall. Distribution of disease has potential to expand with changes in climate and potential vector distribution. Virus transmission is greatly reduced when biting midge activity is reduced following onset of winter and frost.

Incubation period: 3-14 days depending on form of infection: acute respiratory form 3-5 days, cardiac form 1-2 weeks, fever form 4-14 days.

Clinical signs: The respiratory form can be acute or peracute causing fever (40-42 °C), respiratory distress (RR>50), paroxysmal coughing, nasal discharge, congested conjunctiva, and abnormal stance. Mortality rate may reach 95%. The cardiac form causes fever (39-41 °C), swelling of the supraorbital fossa extending to head and neck causing dyspnea and colic. Mortality rate may be as high as 50%. The most common form is the mixed form. A combination of respiratory signs with head and neck swelling is seen with a mortality rate of 70%. The fever form is characterized by mild pyrexia with occasional congestion of conjunctiva, depression and inappetence but minimal mortality. In endemic areas, this disease can be confused with equine encephalosis or equine viral arteritis. Dogs usually develop the respiratory form of disease. Zebra may develop a mild fever.

Post mortem, gross, or histologic findings:

Gross lesions vary based on the form of disease. In the respiratory form, lesions include pulmonary edema, hydrothorax, frothy fluid in the trachea, bronchi and bronchioles, occasional pleural effusion, edematous lymph nodes, congestion and hyperemia of abdominal viscera, and petechiae of the epicardium and endocardium. In the cardiac form yellow, gelatinous infiltrations of subcutaneous and intramuscular tissues of the head and neck, as well as hydropericardium, myocarditis with petechiae of the endocardium and epicardium, petechiae of the peritoneum and ventral tongue, flaccid or slightly edematous lungs, and hemorrhagic gastritis may be seen. The mixed form of disease produces a combination of lesions characteristic of the respiratory and cardiac forms.

Diagnosis: Virus neutralization is the gold standard test although RT-PCR is used for rapid screening samples

AFRICAN HORSE SICKNESS

from suspected clinical cases. Serology (ELISA, complement fixation, virus neutralization, lateral flow assay, Luminex assay) and virus isolation are also available. For determination of serotype use virus neutralization or RT-PCR. Outbreaks should be diagnosed using more than one test when possible.

Material required for laboratory analysis: Serum, whole blood, (Lithium heparin or EDTA blood tubes), fresh tissue not frozen (spleen, lymph node, lung), formalin fixed tissue (10:1).

Relevant diagnostic laboratories: Call prior to sample submission

Foreign Animal Disease Diagnostic Laboratory for PCR testing of sick animal samples: must be in packages
USDA-APHIS- FADDL

40550 Route 25

579 Edwards Ave, Calverton, NY 11933 (This is a hold location address and must be included on way bill)

Orient Point, NY 11957

For Friday fed ex shipping: must check box 6 on label to ensure Saturday delivery

Director: Dr. Kimberly Dodd

Telephone: (631) 323-3256

Fax: (631) 323-3366

Email: Kimberly.A.Dodd@aphis.usda.gov

National Veterinary Services Laboratory for VI, ELISA, and PCR testing

USDA-APHIS-NVSL

P.O. BOX 844, 1920 DAYTON AVENUE, AMES, IA 50010

Telephone: (515) 337-7514

Treatment: None, but supportive care is warranted.

Prevention and control: Imported equids should be free of clinical signs on day of export and should not have received AHS vaccine within 40 days (infective period) prior to export, and be quarantined in vector protected facilities for 14-40 days prior to export and throughout transportation. Importation of equine semen follows the same guidelines. Review the OIE website for the most up to date recommendations on export/import requirements. In endemic areas, vaccinate susceptible animals using approved vaccines. Reduce vector exposure by stabling equids at peak times of vector activity. Establish vector control methods.

During an outbreak, quarantine the area, stop all equid movement in or out, test suspect cases, vaccinate susceptible equids and conduct epidemiological investigation. Do not feed carcasses from infected individuals to carnivores.

Suggested disinfectant for housing facilities: Commercial chlorine, iodine and quaternary ammonia based compounds

Notification: Reportable to the OIE

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Do not introduce naive animals to infected animals. Animals at risk of exposure should be vaccinated prior to introduction to new groups.

Conditions for restoring disease-free status after an outbreak: Clean areas with appropriate disinfectants.

Experts who may be consulted:

Alan Guthrie, MMedVet, PhD

Director, Equine Research Centre

University of Pretoria

Onderstepoort, South Africa

alan.guthrie@up.ac.za

References:

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AFRICAN HORSE SICKNESS

<http://www.oie.int/animal-health-in-the-world/official-disease-status/african-horse-sickness/>

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AFRICAN SWINE FEVER

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Domestic and wild pigs and peccaries	Direct: oral and nasal routes, skin wounds. Indirect: feeding uncooked infected pork products, fomites or bites of soft ticks (<i>Ornithodoros</i> spp.).	Acute: pyrexia, weak hind legs, cyanotic skin, hemor-rhages. Chronic: emaciation, joint swelling, abortion.	Inapparent disease to acute death.	None	Prevention: no vaccine, control soft ticks, do not feed uncooked pork. Control: test, slaughter, quarantine, disinfect.	No

Fact Sheet compiled by: Cora Singleton
Sheet completed on: 1 March 2011; updated 1 October 2012.
Fact Sheet Reviewed by: Pat Morris, Alex Ramirez
Susceptible animal groups: Swine. Warthogs (<i>Phocochoerus aethiopicus</i>), bushpigs (<i>Potamochoerus porcus</i>), and giant forest hogs (<i>Hylochoerus meinertzhageni</i>) act as reservoir hosts in Africa. Ticks of the genus <i>Ornithodoros</i> are considered the natural arthropod host.
Causative organism: African swine fever virus is a large icosahedral DNA virus, the only member of the genus <i>Asfivirus</i> in the Asfarviridae family.
Zoonotic potential: No
Distribution: Africa, parts of Europe (Spain and Portugal), the Caribbean
Incubation period: 3-19 days; acute form 3-7 days.
Clinical signs: Acute disease – Pyrexia, severe depression, weak hind legs, ocular discharge, erythema, cyanotic skin blotching, extensive hemorrhages, diarrhea, cough, convulsions. High mortality. Chronic disease – Pyrexia, depression, emaciation, joint swelling, pneumonia, necrotic skin patches, abortion. Low mortality, persistent viremia.
Post mortem, gross, or histologic findings: Widespread petechial and ecchymotic hemorrhages (lymph nodes, kidneys, skin, larynx, urinary bladder), dark red to purple areas on skin, occasional button ulcers in cecum, enlarged spleen.
Diagnosis: Agent identification – Culture, hemadsorption test, fluorescent antibody test (FAT), PCR; serology – ELISA, indirect fluorescent antibody test (IFA), immunoblotting test, counter immunoelectrophoresis. OIE prescribed test for international trade – ELISA (alternative IFA)
Material required for laboratory analysis: Contact regulatory agencies prior to collecting and shipping samples which should include tissues (lymph node, kidney, spleen, lung) and blood (serum and EDTA-anticoagulated whole blood).
Relevant diagnostic laboratories: Foreign Animal Disease Diagnostic Laboratory USDA-APHIS-VS-NVSL-FADDL 40550 Route 25 (for packages)

AFRICAN SWINE FEVER

Orient Point, NY 11957
P.O. Box 848 (for letters)
Greenport, NY 11944-0848
Director: Dr. Fernando Torres-Velez
Phone: (631) 323-3256
Fax: (631) 323-3366
Email: Fernando.J.Torres-Velez@aphis.usda.gov

Treatment: No effective treatment.

Prevention and control: Prevention – vaccines are not effective. Prevention includes control of pig movements and implementation of serological surveys to detect carrier pigs; control of natural reservoirs (soft ticks); and avoidance of feeding uncooked pork products. Control measures include depopulation of infected pigs, disinfection of premises, area quarantine, and control of pig movement.

Suggested disinfectant for housing facilities: Sodium hydroxide, hypochlorites, formalin, sodium carbonate, ortho-phenylphenol, iodine compounds.

Notification: Reportable to the USDA/APHIS through the State Veterinarian or the federal Area Veterinarian in Charge. The disease is also reportable to the World Organization for Animal Health (OIE).

Measures required under the Animal Disease Surveillance Plan: Foreign animal disease – reportable.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Infections must be reported to USDA/APHIS for management.

Experts who may be consulted:

USDA State Veterinarians or federal Area Veterinarians in Charge

References:

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ALEUTIAN DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mustelids; most common in farm-raised mink; rarely observed in ferrets over last several years.	Vertical (not reported in ferrets) and horizontal transmission (air, direct contact with urine, feces, or blood; contact with contaminated fomites.	Chronic wasting, weakness, reproductive failure, melena, CNS signs, and renal failure.	The disease can be fatal; some pet ferrets are carriers and may not show clinical signs for years.	No effective therapy although; anti-inflammatory or immune suppression treatment may minimize organ damage and clinical signs in pet ferrets.	Biosecurity in facilities; test and cull positive animals or minimally isolate in pet situations.	No.

Fact Sheet compiled by: Gwen E. Myers

Sheet completed on: 1 February 2011; updated 15 August 2013

Fact Sheet Reviewed by: A. Hossain Farid; Katrina Ramsell

Susceptible animal groups: Mustelids; notably, mink, weasels, and ferret.

Causative organism: Parvovirus.

Zoonotic potential: None has been identified. However, rare reports of a possible relationship between Aleutian mink disease parvovirus (AMDV) and human infection are noted. Two mink farmers with vascular disease and microangiopathy similar to that in mink with Aleutian disease were found to have AMDV-specific antibodies and AMDV DNA. These findings raise the suspicion that AMDV may play a role in human disease. See article at the end of sheet marked.

Distribution: Worldwide; predominantly on mink farm operations; uncommonly reported in pet ferrets recently.

Incubation period: Variable, but long period inapparent carrier state can occur. AMDV can be detected in blood by PCR in most animals within 10 days post-infection. Viral replication reached its peak around 10 days post-infection thus incubation period is considered short.

Clinical signs: Pathogenesis of this disease is an immune system response of producing a large increase in antibodies resulting in a hypergammaglobulinemia. The formed antigen/antibody complexes are unable to neutralize the virus but they are deposited and cause damage within various tissues and organ systems, including kidneys, liver, bile ducts, respiratory system, spinal cord, gastrointestinal tract, urinary bladder, and blood vessels. Subsequently, inflammation occurs with an elevation in plasmacytes and lymphocytes and significant inflammation will result in disease with the organs affected. However, ferrets with mild inflammation may have no clinical signs.

Post mortem, gross, or histologic findings:

Gross: Hepatomegaly, splenomegaly, renal changes (varying from swelling, petechiation to atrophy and pitting), and enlarged mesenteric lymph nodes. Infected ferrets may have few or no gross lesions.

Histologic: Plasma cell infiltration in the kidneys, liver, spleen, lymph nodes, and bone marrow; bile duct proliferation; membranous glomerulonephritis and fibrinoid arteritis; lymphoplasmacytic meningitis.

Diagnosis: Presumptive diagnosis is based on clinical signs and hypergammaglobulinemia. Common testing modalities: counterimmunoelectrophoresis (CEP), ELISA, and PCR. Tissue biopsies usually done post-mortem.

ALEUTIAN DISEASE

Material required for laboratory analysis: Blood; serum for CEP/ELISA; urine, saliva, feces, tissues for PCR.

Relevant diagnostic laboratories:

PCR and virus sequencing:

Weymouth AD Laboratory, Weymouth Nova Scotia (CIEO)

Nova Scotia Department of Agriculture Pathology Laboratory, Truro, Nova Scotia (CIEP)

Dalhousie University Faculty of Agriculture

c/o Hosain Farid, Ph.D.

Department of Plant & Animal Sciences

Agricultural Campus

P.O. Box 550

Truro, Nova Scotia, B2N 5E3, Canada

a.farid@dal.ca

(902) 893-6727

PCR and ELISA testing:

Blue Cross Animal Hospital

Attention: Dr. Blau – CEP tests

401 N. Miller Avenue

Burley, Idaho 83318

Phone: (208) 678-5553

Fax: 208-677-8957

PCR and ELISA:

University of Georgia

Infectious Diseases Laboratory

110 Riverbend Rd.

Riverbend North, Room 150

Athens, GA 30602

(706) 542-8092

PCR:

Wisconsin Veterinary Diagnostic Lab

445 Easterday Lane

Madison, Wisconsin 53706

(608) 262-5432

Treatment: No effective treatment. Selective breeding for mink that can tolerate the virus.

Prevention and control: Test and cull on mink farms; no vaccine option. Strict biosecurity and quarantine in ferret colonies and shelters. Ferrets in a seropositive household should have no exposure to ferrets outside of the household although cagemates are considered already exposed.

Suggested disinfectant for housing facilities: Clean environment with 10% bleach solution. Steam clean pens and spray with 2% sodium hydroxide.

Notification: None required.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Not recommended - infected animals should be isolated or culled.

Conditions for restoring disease-free status after an outbreak: Following removal of infected animals and

ALEUTIAN DISEASE

environmental cleaning, restocking can be considered. Identify the source and route of infection to prevent re-infection.

Experts who may be consulted:

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References:

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ANAPLASMOSIS

Two groups in Anaplasmataceae: <u>Group 1</u> - infects red blood cells; <u>Group 2</u> - infects other blood cells						
Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p><u>Group 1</u> – Ruminants</p> <p><u>Group 2</u> - Wide range of mammals (including humans)</p>	<p><u>Group 1+2</u> - Biological transmission via ticks; mechanical transmission by infected blood cell transfusion; transplacental.</p> <p><u>Group 1</u> mechanical transmission (e.g. biting flies)</p>	<p><u>Group 1</u> Anemia, lethargy, pale mucous membranes</p> <p><u>Group 2</u> Headache, pyrexia, chills, myalgia, anemia. Nausea, vomiting, diarrhea. In latter stages, bleeding problems, respiratory and organ failure, death.</p>	<p><u>Group 1</u> - Severity increases with age</p> <p><u>Group 2</u> - typically mild, more severe in mature or immunosuppressed animals, or with co-infections</p>	<p><u>Group 1</u> – Oxytetracycline, chlortetracycline (in feed)</p> <p><u>Group 2</u> - Doxycycline</p>	<p><u>Group 1</u> - Control ticks and biting flies; prevent entry of carriers; vaccination; prophylactic antibiotics.</p> <p><u>Group 2</u> - control tick exposure, insect repellents</p>	<p><u>Group 1</u> - no</p> <p><u>Group 2</u> - yes</p>

Fact Sheet compiled by: Dorothy Geale and Enrique Yarto

Sheet completed on: 20 Jan 2019

Fact Sheet Reviewed by: Gretchen Cole

Susceptible animal groups:

Group 1 - Cattle, sheep, goats, mule deer and other ruminants.

Group 2 - Mammals: humans, cattle, sheep, goats, camels (dromedary), donkeys, pigs, domestic dogs and cats, small mammals; birds and many wildlife species.

Infected domestic cattle become persistent carriers after recovery and play an important role in the epidemiology of the disease. Due to global warming, vector tick distribution and increase in the horse industry, horses should be considered as a potential reservoir for *A. phagocytophilum* and cross infectivity should be assessed.

Causative organism: Obligate intracellular bacteria: Order Rickettsiales, Family Anaplasmataceae, Genus *Anaplasma*.

Group 1 - infect red blood cells (*A. marginale*, *A. ovis*)

Group 2 – infects white blood cells and platelets (*A. phagocytophilum*, *A. bovis*, *A. platys*)

Biological transmission by ticks occurs in both groups. Iatrogenic mechanical transmission by transfer of infected blood cells occurs in Group 1 by contaminated scalpels, needles or tattooing, dehorning and castration equipment and in Group 2, in humans by blood transfusions or organ transplants. Natural mechanical transmission is effected in Group 1 by biting flies (most commonly horse and stable flies). For Group 1, transplacental transmission is reported especially with acute infection in the 2nd or 3rd trimester. It also occurs in Group 2 but its epidemiological role is not well characterized for either group.

Group 1

- *A. marginale* infects cattle and is the agent of bovine anaplasmosis. Major reservoirs are cattle and ticks. Less pathogenic is *A. marginale* subspecies *centrale* which is never reported in North America and used as a live vaccine strain in South Africa, Israel and some South American countries.

- *A. ovis* infects sheep, goats, deer (mule deer and reindeer), red fox primarily in North America.

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Group 2

● *A. phagocytophilum* (previously *Ehrlichia phagocytophila*, *E. equi*, and the human granulocytic ehrlichiosis agent) infects a wide variety of mammals including ruminants (cattle, white tailed deer), horses, rabbits, pigs and small rodents (e.g., white-footed mice, wood rats, gray squirrels), marsupial (Virginia opossum), skunk, hedgehog, bears, fox, and raccoon. Additional snakes (northern alligator lizard, Pacific gopher snake) have been reported.

Red foxes (*Vulpes vulpes*) have been identified as hosts of *Anaplasma* spp. (*A. phagocytophilum*, *A. ovis*, *A. platys*) and may contribute to the maintenance of *A. phagocytophilum* in Europe.

Raccoons have (*Procyon lotor*) have been reported as hosts for *A. bovis*, *A. phagocytophilum* and other Anaplasmatae playing a role in the maintenance of *A. phagocytophilum* in the USA and Europe.

In camels, age has been identified as a risk factor for the prevalence of *A. phagocytophilum* in a farmed camels (*C. dromedarius*) in Iran where camels less than 5 yr had a prevalence of 44.3% while camels older than 5 yr had a prevalence of 25.4%.

● *Anaplasma bovis* (previously *Ehrlichia bovis*) infects cattle, deer, and raccoon dogs which are reservoirs in Asia and Africa.

● *Anaplasma platys* (previously *Ehrlichia platys*) is reported in dogs and rarely in cats, red fox, impala, and sheep in Asia, Europe and South America and is the only *Anaplasma* to infect platelets.

Zoonotic potential: Group 1 *Anaplasma* sp. are not infective for humans and therefore not zoonotic.

A. phagocytophilum of Group 2, first described in sheep in Europe, has become an emerging pathogen of humans in both Europe and the U.S. It is transmitted by *Ixodes* ticks as biological vector. Blood transfusions or organ transplants also pose a zoonotic risk. Additionally, red deer and wild boars have been found to be infected with human pathogenic variants of this bacteria.

Distribution:

Group 1 – *A. marginale* occurs worldwide and in all states of US, except Hawaii. It is endemic throughout the Gulf Coast states and several of the Midwestern and Western states. Outbreaks are often seasonal and coincide with the emergence of arthropod vectors in warmer months (spring, early summer, and early fall).

Group 2 - *A. phagocytophilum* also occurs worldwide and in US primarily in the west, upper midwest and northeast but future distribution may change with tick vectors. States reporting the highest incidence in 2010 included Minnesota, Wisconsin, New York, New Jersey, Rhode Island, and Connecticut.

Norway, UK, Sweden, Switzerland, and Germany have reported infections in ruminants, dogs, and people.

Infections in camels in Iran is reported with regional variation.

Infections in wild felids have been reported in Brazil and Africa.

Incubation period:

Group 1 - ranges from 7 to 60 days (*A. marginale*, *A. ovis*, *A. marginale* subsp. *centrale*) depending on dose. Rickettsemia doubles every 24 hours with acute clinical disease in 7-10 days in susceptible species.

Group 2 – ranges from 7 to 14 days in humans, sheep and dogs.

Clinical signs:

Group 1 – Clinical signs are highly variable, ranging from subclinical infection in calves under a year to severe peracute disease in adult naïve cattle, characterized by significant production losses (milk or weight gain), bull infertility, severe anemia, icterus, inappetence, dehydration, constipation, dark yellow urine, weight loss, pyrexia, abortion and death. The acute form generally occurs in cattle from 1-3 years old, with similar but more moderate clinical signs. All recovered animals become persistent carriers and reservoirs of infection for life.

Group 2 – Humans (*A. phagocytophilum*): Clinical disease is associated with acute parasitemia of which the duration and severity is variable. Co-infection with other pathogens results in greater severity of symptoms. Genetic variants of *A. phagocytophilum* and be associated with mild or flu-like clinical signs or rash in 10%

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of patients. The aged and immunosuppressed show more severe signs. These signs include headache, pyrexia, chills, myalgia, nausea, ataxia, organ failure, susceptibility to opportunistic infections, neuritis or respiratory complications. The US case fatality rate from 2000-2010 was 1% with a rise in incidence from 1.4 to 6.1 cases per million. In 2016, the Centers for Disease Control reported 4151 human cases of anaplasmosis in the US, a 14% increase between 2015 and 2016.

Dogs (*A. platys*; *A. phagocytophilum*): Most often disease is subclinical or a mild, flu-like presentation that is self-limiting. Animals with an acute infection often have vague signs including pyrexia, malaise, lethargy, anorexia, and general muscle pain resulting in reluctance to move. *A. phagocytophilum* most commonly causes clinical disease in dogs older than 8 years with joint pain and lameness so it must be distinguished from Lyme disease. Gastrointestinal, respiratory and neurological signs may also occur. Infections may be subclinical or in a carrier state. In endemic areas, over 40% of dogs may be seropositive, while morbidity is low. Dogs co-infected with *Borrelia burgdorferi* and *A. phagocytophilum* are nearly twice as likely to develop clinical disease. *A. platys* produces clinical disease related to a cyclic thrombocytopenia (typically $<20,000/\mu\text{l}$ for 1-2 days then repeats in 1-2 weeks). Although usually mild, more severe clinical signs including pyrexia, lethargy, pale mucus membranes, petechial hemorrhages, epistaxis, and lymphadenopathy occur.

Other animals (*A. phagocytophilum*): Sheep may have mild clinical signs of lethargy with abortions in ewes. Similarly in endemic areas, dairy cattle exhibit abortions, drop in milk production and respiratory disease. Complications occur due to secondary bacterial infections (e.g., pastuerellosis, septicemic listeriosis). Other ruminants and cervids may exhibit anorexia, dullness, fever, weight loss, coughing, abortion, and low fertility. Horses may have acute onset with older animals developing fever, lethargy, inappetence, limb edema while young animals typically have mild disease. A report in maned wolves (*Chrysocyon brachyurus*) describes coughing and tachypnea due to severe pulmonary congestion, splenomegaly, ataxia, anorexia, lethargy, dehydration; mild jaundice, petechiation; leukocytosis, anemia, hyperfibrinogenemia.

Post mortem, gross, or histologic findings:

Group 1 - include anemia, jaundice, splenomegaly and the liver may be enlarged with a deep orange color. Hepatic and mediastinal lymph nodes may appear brown and the gall bladder distended with thick brown or green bile. Serous effusions may occur in body cavities, edema, petechial hemorrhages in the epi- and endocardium often accompanied by severe gastrointestinal stasis. Reticuloendothelial phagocytosis of erythrocytes may be evident microscopically in various organs, most notably in the spleen.

Group 2 - *A. phagocytophilum* is only one of four human neutrophil intracellular organisms forming morulae (cytoplasmic vacuole containing multiple coccoid to ellipsoid basophilic rickettsia) approximately 1.5 μm to 2.5 μm in diameter (reported up to 6 μm). During acute rickettsemia, the organism has been demonstrated in the alveolar macrophages, Kupffer cells, and other tissue macrophages. Sites of persistence in-between recurrent rickettsemia remain to be established.

Diagnosis:**Hematology**

Group 1 - Initially based on clinical signs, history of tick exposure and clinical pathology (lymphopenia, mild to severe thrombocytopenia, mild to moderate nonregenerative anemia, elevated ALP, mild to moderate hypoalbuminemia and hyperfibrinogenemia may occur). In the acute phase, the presence of characteristic intracellular inclusions (marginal bodies) on Giemsa/Wright's/Diff-Q-stained blood smears (buffy coat recommended) along the margins of the erythrocyte (*A. marginale*) or more centrally (*A. marginale* subsp. *centrale*).

Group 2 - Neutrophils infected by *A. phagocytophilum* (1-27%) contain distinctive granulocytic morulae which appear in the peripheral blood at 4-14 days and persist up to 8 days. In animals with polyarthritis, synovial fluid may exhibit decreased viscosity and an increased leukocyte count (>3000 cells/ μl ;

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predominantly neutrophils but $\leq 1\%$ may contain morula). *A. platys* morulae may be found in circulating platelets. Hematology is not reliable for pre-symptomatic or carriers.

Antibody serology

Group 1 - Enzyme-linked immunosorbent assay (ELISA) marketed by VMRD will not differentiate between *Anaplasma* species as the test is based on major surface protein 5 (MSP5) which is highly conserved in the genus. The reported *A. marginale* sensitivity 95% and specificity 98% is limited by cross-reactivity, low early sensitivity and low specificity for true negative cattle after oral chlortetracycline treatment. The complement fixation (CF) and card agglutination tests (CAT) are no longer considered to be valid and thus not used for diagnosis of bovine anaplasmosis.

Group 2 - ELISA (IDEXX SNAP 4Dx) for *A. phagocytophilum* (sensitivity 99.4%, specificity ~100%), reportedly detects as early as 8 days post-inoculation (dogs with *Ehrlichia ewingii*) do not likely cross-react, some cross-reactivity with *A. platys*). The indirect immunofluorescence assay (IFA) detects a 4-fold increase in IgG-specific antibody titer to *A. phagocytophilum* antigen in paired serum samples (taken the 1st week of illness and 2-4 weeks later). Note: IgM tests are not always specific, and that the IgM response may be persistent. Seroconversion in dogs may occur as soon as 2-5 days after morulae appear in the peripheral blood. Positive titer $\geq 1:80$, most will have titer $\geq 1:320$.

Antigen detection

Group 1 + 2 - polymerase chain reaction (PCR) is the most specific method (nested PCR theoretically detects 0.0000001% rickettsemia or 30 infected red blood cells per ml); can distinguish between species, but poses problems due to non-specific amplification, requiring confirmation of the amplified fragment (sequencing). Should be repeated if negative in suspect carriers (*A. marginale*).

Other means of diagnosis

Group 1 + 2 - Immunohistochemistry can demonstrate *Anaplasma* antigen in a biopsy/necropsy sample. The organism can be isolated in cell culture (mainly research as it is impractical for clinical cases). The gold standard for *A. marginale* is the demonstration of the organism 4-8 weeks after inoculation of suspect blood into splenectomized calves.

Material required for laboratory analysis: For both Group 1 + 2:

Anticoagulant blood, thin and thick blood films.

At necropsy: thin blood films of liver, kidney, spleen, lungs; and peripheral blood.

PCR: whole blood (EDTA)

Sample blood prior to starting antimicrobials to avoid false negative test results.

Relevant diagnostic laboratories: *Anaplasma* sp. can be diagnosed at most accredited diagnostic laboratories using ELISA (in-house SNAP 4Dx, IDEXX for *A. phagocytophilum*); cELISA for *A. marginale* (VMRD, Antech (FastPanel™ PCR Canine Ehrlichiosis/Anaplasmosis Profile) for *A. platys* and *A. phagocytophilum*, cross-reacts with *Ehrlichia*. Zoologix PCR (*A. platys*) also offers tickborne disease PCR panel, that includes *A. platys*.

Treatment:

Group 1 - In acute outbreaks, parenteral oxytetracycline (cattle) is used as recommended by extension specialists. The survival rate is high in the early stages of the disease (PCV $>15\%$). Blood transfusions, electrolyte solutions, and hematinic drugs may be beneficial in later stages of the disease. Convalescent period of up to 3 months. Cattle remain immune for life but become persistent carriers.

Group 2 - Treatment may be difficult as clinical signs often do not appear until the disease has progressed. Testing for co-infections with other tick-borne organisms is recommended. Tetracycline antibiotics (usually doxycycline in humans and dogs) for 10-14 days (or at least until 3 days after fever subsides). A marked improvement is usually seen in 24-48 hours.

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Prevention and control: Group 1 + 2 organisms need control of tick vectors. Additionally for Group 1, strict sanitation with needles and surgical/dehorning instruments; remove carriers from herd; and chlortetracycline during vector season (medicated salt-mineral blocks or feed blocks).

In South Africa, Australia, Israel, and South America, live *A. centrale* is used as a vaccine. Universal vaccines are not available that are effective for geographically diverse strains for *A. marginale* or *A. phagocytophilum*. A conditional killed vaccine made from a Mississippi strain is available in some southern states. In California, a modified live vaccine (Anavac®) is available for cattle <11 months but lacks efficacy and is rarely used.

Identification of regional vectors are important to control zoonotic anaplasmosis. Use of Environmental Protection Agency (EPA) registered insect repellents is also recommended.

Suggested disinfectant for housing facilities: No disinfectant. Application of acaricides and removal of leaf litter/brush (tick habitat) can be effective.

Notification: *A. marginale* is a reportable disease in ~ 30 states and tracked nationally through National Animal Health Reporting System (USDA) in ~ 48 states. *A. phagocytophilum* is monitored through National Notifiable Disease System (CDC).

Measures required for introducing animals to infected animal: Infected animals become carriers and act as a reservoir of infection for life. In endemic areas, early infection or vaccination in cattle promotes life-long immunity. With no killed vaccines available in the US, separation of carriers and non-infected introduced mature animals is essential.

Conditions for restoring disease-free status after an animal outbreak:

Group 1 - Carriers may relapse when immunosuppressed (e.g. corticosteroids), when infected with other pathogens, or after splenectomy. As a lifelong reservoir of infection, they should be removed. No anti-microbials are approved in the US for eliminating *A. marginale* infections in cattle. Allegedly, the carrier state may be eliminated with a long-acting oxytetracycline treatment, but experimental work at Kansas State University in the mid -2000s found chemosterilization inconsistent. Although, long term oral tetracycline at high extra-label doses had greater success, parenteral administration was less so, and conflicting results suggests differences in susceptibility of *A. marginale* strains. Chemosterilized animals are fully susceptible to re-infection.

Experts who may be consulted:

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ANATRICHOSOMA

Animal group(s) affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals (primates, marsupials, rodents, carnivores)	Unknown	Cutaneous, nasal, oral, gastric, or ocular nodules or ulcers	Clinically insignificant to severe ulceration	Avermectins, benzimidazoles	Hygiene and sanitation; control of wildlife; prophylactic parasite treatments	Yes

Fact Sheet compiled by: Beth Bicknese

Sheet completed on: 30 April 2011; Last updated 12 Dec 2017

Fact Sheet reviewed by: Jennifer D'Agostino

Susceptible animal groups

Mammals. Reported in Old World monkeys, apes, and marmosets under human care, wild-caught tree shrews, wild American and Australian marsupials, wild rodents, domestic cats and dogs, and humans. Severe inflammatory cutaneous lesions in carnivores and some primates (including humans) suggest these are aberrant hosts.

Causative organism

Anatrichosoma cutaneum and *A. cynomolgi* have been found in nasal and cutaneous lesions in wild-caught and captive primates and are the presumed cause of most human and domestic animal cases. *A. buccalis* has been found in the oral mucosa of opossums, and was suspected in one human case. *A. ocularis*, *A. gerbilis*, and *A. haycocki* have been reported in wild or wild-caught tree shrews, gerbilid and murid rodents, and *Antechinus* spp. marsupials, respectively.

Adult females tunnel through the epidermis, laying embryonated ova which reach the surface through normal exfoliation. Ova are then swallowed and passed in feces or released directly into the environment. Adult males reside in the dermis.

Mechanism of infection is unknown. Attempts at experimental direct infection have been unsuccessful, suggesting an indirect life cycle, but no intermediate host has been identified. One report found free immature *A. haycocki* in the intestine of *Antechinus* spp. hosts, suggesting an enteric route of infection in this species. Lesions have recurred after treatment in captive primates, suggesting either re-infection or incomplete response to treatment.

Zoonotic potential

Eight human cases reported (Japan, Vietnam, Malaysia, Italy, USA), including one case in Illinois in 2010 and two in Iowa in 2014, all with recent travel to Mexico. Exposure route unknown.

Distribution

Documented in wild animals from the Americas, the Middle East, Africa, India, and Australia, and in humans and domestic animals in the Americas, Europe, Africa, and Asia.

Incubation period

Unknown; clinical lesions are associated with migration of adult worms.

Clinical symptoms

Cutaneous (*A. cutaneum*, *A. cynomolgi*): Nodules or track-like lesions with ulceration, apparent predilection for glabrous skin. Severe ulcerative pododermatitis in domestic cats.

Nasal (*A. cynomolgi*, *A. cutaneum*): Nodules or tracks in the nasal mucosa of primates, minimal local inflammation.

Oral (*A. buccalis*): Nodules or tracks in the oral mucosa. Minimal local inflammation in opossums, mucosal ulceration in one suspected human case.

Gastric (*A. gerbilis*): Nodules or tracks in the gastric mucosa of gerbilid and murid rodents.

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<p>Ocular (<i>A. ocularis</i>): Adults visible within the corneal or conjunctival epithelium of tree shrews, minimal local inflammation.</p> <p>Glandular (<i>A. haycocki</i>): Adults within the tissue of paraocloacal glands or encapsulated in the lumen of the cloaca of <i>Antechinus</i> spp., minimal local inflammation.</p>
<p>Post mortem, gross, or histologic findings</p> <p>Histopathology shows adults and ova embedded in epithelial tissue.</p>
<p>Diagnosis</p> <p>Mucosal swab, skin scraping, biopsy, fecal flotation for ova (<i>Trichuris</i>-like, bipolar plugged). Ova have also been identified by cytology of an otic flush in a dog.</p>
<p>Material required for laboratory analysis</p> <p>Swab, scrape, biopsy, or flush of lesion; feces.</p>
<p>Relevant diagnostic laboratories</p> <p>None</p>
<p>Treatment</p> <p>Avermectins and benzimidazoles have effectively resolved clinical lesions in reported cases, recurrence is infrequently reported.</p> <p>Primates: Fenbendazole 10-25 mg/kg PO once daily for 3-10 days.</p> <p>Domestic cat: Ivermectin 0.3 mg/kg SC weekly for 4 weeks.</p> <p>Human: Mebendazole 100 mg twice daily for 20 days or albendazole 400 mg once daily for 3 days.</p>
<p>Prevention and control</p> <p>The mechanism of infection is unknown. However, control of feral animals and wildlife in exhibit areas, sanitation and hygiene with regular removal of feces from enclosures, and routine prophylactic deworming are expected to be beneficial.</p>
<p>Suggested disinfectant for housing facilities</p> <p>None specified; expect sensitivity as for <i>Trichuris</i> spp. and <i>Capillaria</i> spp.</p>
<p>Notification</p> <p>None required.</p>
<p>Measures required under the Animal Disease Surveillance Plan</p> <p>None.</p>
<p>Measures required for introducing animals to infected animal</p> <p>None specified; treat infected animals prior to introduction if possible.</p>
<p>Conditions for restoring disease-free status after an outbreak</p> <p>None required; treat exposed individuals if possible, eliminate feces in enclosure.</p>
<p>Experts who may be consulted:</p> <p>Dwight D. Bowman, MS, PhD (ddb3@cornell.edu) Professor of Parasitology C4-119 VMC Dept. Micro & Immunol Cornell University College of Veterinary Medicine Tower Road Ithaca, NY 14853-6401</p> <p>Dr. Heather Stockdale Walden (hdstockdale@ufl.edu) University of Florida College of Veterinary Medicine Department of Comparative, Diagnostic, and Population Medicine 1945 SW 16th Ave., V2-155 Gainesville, FL 32608</p>

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ANGIOSTRONGYLUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p><i>A. cantonensis</i> – non-human primates, marsupials, horses, dogs.</p> <p><i>A. vasorum</i> – canids, red pandas, stoats, badgers.</p>	<p>Ingestion of intermediate (snails and slugs) or paratenic (shellfish, frogs, lizards) hosts.</p>	<p><i>A. cantonensis</i> - variety of neurologic signs, including ataxia, ascending paresis, hypereesthesia, seizure, muscle wasting, coma, +/- gastrointestinal signs.</p> <p><i>A. vasorum</i> – cough, dyspnea, exercise intolerance, hemorrhage, anorexia, weight loss, occasional CNS signs.</p>	<p><i>A. cantonensis</i> causes severe progressive neurologic disease in nonhuman primates, often resulting in death or euthanasia.</p> <p><i>A. vasorum</i> can be asymptomatic to fatal in canids. It appears fatal in red pandas.</p> <p>Recovery for both is independent of severity of presenting signs.</p>	<p>Primary supportive care for both. Treatment with anthelmintics (fenbendazole, milbemycin, topical moxidectin) may shorten clinical course of <i>A. vasorum</i> infections.</p> <p>The use of anthelmintics is controversial in <i>A. cantonensis</i> infections.</p>	<p>Avoidance and removal of intermediate/paratenic hosts. Monthly topical moxidectin has been recommended.</p> <p>Monthly prophylactic anthelmintics have been used in red pandas.</p>	<p><i>A. cantonensis</i> has been reported in humans.</p>

Fact Sheet compiled by: Kristina M. Delaski

Sheet completed on: 14 February 2018

Fact Sheet Reviewed by:

Susceptible animal groups:

- *A. cantonensis* – Rats are the definitive hosts. Aberrant hosts include several species of non-human primates, marsupials (wallaby, bettong, opossum), nine-banded armadillos, Tawny frogmouths, cockatoos, raptors, horses, and dogs.
- *A. vasorum* – Dogs and red foxes are the definitive hosts. Other canids (coyote, wolf, jackal), European otter, ferrets, badgers and red pandas have also been infected, and red pandas have been reported to shed infective larvae.

Causative organism: *Angiostrongylus cantonensis*–neurologic disease; *A. vasorum*–cardiopulmonary disease

Zoonotic potential: *A. cantonensis* has been extensively studied in humans and is considered a zoonotic disease. Transmission is through ingestion of an intermediate or paratenic host, usually raw or undercooked seafood in endemic areas.

Distribution: *A. cantonensis* is endemic in the Pacific Islands and Southeast Asia, but has spread to the Americas, including the US, the Caribbean islands and Brazil. It is now considered endemic in the southwestern US. *A. vasorum* is endemic to Europe, Africa and South America. It has recently been documented in Newfoundland, Canada and in West Virginia.

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Incubation period: *A. cantonensis* larvae can be seen in the CNS hours to days following ingestion, although typical incubation is 2-3 weeks in humans. The prepatent period for *A. vasorum* ranges from 28 to 108 days.

Clinical signs:

- *A. cantonensis* – Due to migration of the L3 larvae in central nervous tissue and subsequent eosinophilic inflammation, infection can result in a variety of neurologic signs. Most common signs include ascending bilateral paresis and muscle wasting, urinary bladder paresis, hyperesthesia, and occasional gastrointestinal signs. Seizures, cranial nerve palsies, and coma have also been reported. In humans, ocular *larva migrans* can occur.
- *A. vasorum* – Signs can vary and may be absent early in infection or with low parasite burdens. Interstitial pneumonia and hemorrhage is most common, leading to fibrosis. This results in tussis, dyspnea, exercise intolerance, anorexia and weight loss. Vascular lesions associated with adult worms can lead to pulmonary hypertension and congestive heart failure. Coagulopathy of unknown etiology has been documented and can be the presenting clinical sign. This results in anemia, melena, subcutaneous hematomas, and other sequelae depending on location of hemorrhage. Central nervous system signs are often related to intracranial hemorrhage but can also be the result of aberrant larval migration. Signs vary depending on location of lesions. Red pandas were reported with cough, dyspnea and exercise intolerance, although apparent asymptomatic infections can occur.

Post mortem, gross, or histologic findings:

- *A. cantonensis* – Cerebral and cerebellar meningitis, with varying degrees of malacia. Nematodes are often found near the cerebral blood vessels or free in the white matter of the central nervous system with mild to moderate inflammation. Similar lesions may occur in the spinal cord. Hemorrhage in the central canal of the spinal cord has been reported.
- *A. vasorum* – Adult worms present in the lumen of the pulmonary artery and right ventricle. They can be differentiated from *Dirofilaria immitis* by the small size of *A. vasorum*. Interstitial pneumonia with hemorrhage, granulomas around eggs/larvae, and fibrosis. Adult worms cause thromboarteritis and intimal proliferation in affected vessels. Cases with coagulopathy may have intracranial, intrathoracic, or intra-abdominal hemorrhage. Due to larval migration, L1 larvae may be found in a large variety of tissues at necropsy. The presence of undifferentiated eggs and larvae is characteristic. Necropsies of infected red pandas have found mineralized fibrous tissue in the lungs, with nodules centered on nematode eggs and coiled larvae. Granulomas around larvae have also been reported in pulmonary lymph nodes.

Diagnosis:

- *A. cantonensis* – Definitive diagnosis is difficult. Fecal analysis is of little value, as the infection is only patent in rats. Hematology shows eosinophilia, and CSF often shows an eosinophilic pleocytosis. Occasionally, larvae may be seen in CSF samples. High field MRI has been able to detect cavitations caused by larval migration in humans, but has yet to be useful in canine cases. ELISA tests on serum were not very sensitive, but those performed on CSF were reported as promising. PCR testing is under development. Neither ELISA nor PCR testing is available commercially.
- *A. vasorum* – Definitive diagnosis in canids is by detection of larvae in feces through Baermann examinations or detection of larvae in bronchoalveolar samples. The larvae have a characteristic tail morphology (kinked tail and dorsal spine). Radiographs may show a broncho-alveolar pattern, but are often non-specific. Possible hematologic changes include anemia, eosinophilia, thrombocytopenia, and hypercalcemia. Decreased serum fructosamine has been reported in infected dogs. PCR testing is available but may not detect chronic infections. Combination of fecal PCR and Baermann tests may improve detection. Antigen and antibody ELISA tests are under development and may be the most sensitive method of testing. Commercial antigen-detection kits for *Dirofilaria immitis* have been reported to cross-react with *A. vasorum*.

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Material required for laboratory analysis:

- *A. cantonensis* – CSF for cytology and PCR. ELISA tests for serum were not very sensitive, but those for CSF were reported as promising.
- *A. vasorum* – Pooled fecal samples, collected over 3 consecutive days, for Baermann examination. PCR testing is available for fresh feces, tracheal lavage fluid, and whole blood. Antigen and antibody ELISAs are in development for use on serum, and a canine patient-side antigen test for serum is available through the UK branch of IDEXX.

Relevant diagnostic laboratories: IDEXX offers a PCR test for *A. vasorum*, which can be performed on fresh feces, tracheal lavage fluid, or whole blood in EDTA. IDEXX has also developed a rapid, patient-side antigen test for serum, available in the UK. It has been reported to cross-react with other species of *Angiostrongylus* in definitive hosts. More information is available here: <http://angiodetect.co.uk/>

Treatment: Supportive care for both. Treatment with anthelmintics (fenbendazole, milbemycin, topical moxidectin) is effective for *A. vasorum* in dogs, though caution is urged, as rapid die-off of adult worms may cause severe secondary reactions (ascites, dyspnea) in the patient. All treated animals are monitored with multiple post-treatment fecal Baermann exams. The use of anthelmintics are controversial in *A. cantonensis* infections, due to risk of increased damage sustained due to inflammatory reactions in the central nervous system. Treatment with prednisolone and albendazole has prevented death in a Geoffroy’s tamarin, but the individual had permanent neurologic deficits. Supportive care for *A. cantonensis* consists of fluid support, analgesics, sedatives, and glucocorticoids.

Prevention and control: Prevention is centered on restricting access to intermediate and paratenic hosts. *A. cantonensis* is carried by rats, so pest control is an important component of prevention. Both nematodes can infect a wide range of gastropod intermediate hosts, which can then in turn infect paratenic hosts when frogs, lizards, or shellfish consume the gastropods. Chickens have also been reported to be possible paratenic hosts. Collection animals should have limited access to these sources of infection. Monthly topical moxidectin has been recommended for prevention of *A. vasorum* in dogs. Monthly doses of milbemycin have been used as prophylaxis in red pandas in endemic areas, but as no trial studies have been conducted, these reports are anecdotal.

Suggested disinfectant for housing facilities: 1% bleach, 1-5% glutaraldehyde and cresol-based products are effective disinfectants.

Notification: None

Measures required under the Animal Disease Surveillance Plan: N/A

Measures required for introducing animals to infected animal: As neither nematode is transmitted directly, no special measures are necessary, as long as the enclosure has been cleared of intermediate hosts.

Conditions for restoring disease-free status after an outbreak: Adult *A. vasorum* worms can live in vasculature for up to five years, and ova shedding can be intermittent. Repeated negative fecal exams and PCR tests would likely indicate lack of infection.

Experts who may be consulted:

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ANTHRAX

Animal Group(s) Affected	Transmission (Animal)	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, including humans; and ratites.	Ingestion of spores that can come from soil, infected carcass, soil-contaminated forage or blow-fly contaminated browse. Usually direct transmission, possibly biting flies.	Sudden death, fever followed by death, excitement followed by stupor, respiratory and cardiac distress, colic, diarrhea and vomiting, edema.	Peracute and acute in ruminants and equids. Commonly fatal. Subacute and chronic forms in suids and carnivores from carcass consumption.	Long acting antibiotics given early. Multiple classes of antibiotics are effective. Vaccination.	Rapid detection followed by quarantine, carcass disposal, treatment and movement of adjoining animals, removal of contaminated feed or items, vaccination, and site decontamination.	Humans affected via contact with diseased carcasses or via animal products (meat, bone meal, leather, wool, bristles)

Fact Sheet compiled by: Thomas W. deMaar; updated by Vikki Milne

Sheet completed on: 21 January 2011; updated 4 August 2013

Fact Sheet reviewed by: Martin Hugh-Jones, Mark Drew

Susceptible animal groups: Domestic and wild ruminants are most commonly affected. However humans, equids, and other mammals - such as elephants are susceptible. Suids and carnivores may develop subacute to chronic gastrointestinal type disease after eating infected carcasses. It has been reported in ostriches and rheas. Scavenging birds and mammals, primarily carrion feeders, are known to pass spores through their digestive system without becoming infected as vegetative cells are killed in their acidic stomachs.

Causative organism: *Bacillus anthracis* (spore forming, non-motile, Gram positive rod)

Zoonotic potential: Humans affected via contact with diseased carcasses or via animal products (meat, bone meal, leather, wool, bristles, drum skins) from contaminated carcasses. Cutaneous, gastrointestinal, and inhalation forms of disease occur. It is considered a potential bioterrorism agent.

Distribution: World-wide, especially in areas with neutral or alkaline calcareous soils. Outbreaks can occur after soil disturbance following drought or flood conditions. In US, it occurs sporadically with limited distribution and is more common in west and midwest US, and is enzootic in west Texas, North and South Dakota, and northwest Minnesota.

Incubation period: Typically 3-7 days (range 1-14 days) (OIE standards: up to 20 days). Spores maybe inactive in lungs for several weeks before causing disease.

Clinical signs:

Peracute (ruminants): sudden death.

Acute (ruminants and horses): Abrupt fever and excitement followed by depression, respiratory/cardiac distress, staggering, convulsions, severe colic, and anterior edema; cutaneous signs can be seen in cattle and horses with biting fly infections. Process can lead to death.

Chronic (pigs and carnivores): Oropharyngeal and gastrointestinal signs of disease, usually followed by recovery but death occurs if systemic. Death is not uncommon in free-ranging African lions.

ANTHRAX

<p>Post mortem, gross, or histologic findings: Carcass presented with absence of rigor mortis and rapid decomposition. Dark blood may ooze from mouth, nostrils, eyes, ears, vulva and anus. Edema may be apparent. Carcass will show lack of blood clotting and hemorrhages of serosal surfaces. Organs, particularly the spleen will be congested and enlarged. Oropharyngitis, pharyngeal edema, diphtheritic membranes or ulcers of tonsils are seen in suids and carnivores. Gastrointestinal inflammation and mesenteric lymphadenitis may be seen in suids and carnivores. Hemorrhagic lymphadenitis is histopathologic observation.</p>
<p>Diagnosis: Documentation of <i>Bacillus</i> spores in dried blood sample. PCR, culture, IFA, ELISA and Western Blot tests are available.</p>
<p>Material required for laboratory analysis: Whole blood for culture can be taken post mortem from vein due to lack of clotting. Dried blood smears from similar source can be obtained or blood dried on a cotton swab. Prior to submission, laboratory must be notified for suspicion of anthrax.</p>
<p>Relevant diagnostic laboratories: Diagnostic laboratory with microbiological capacity. Confirmation is accomplished thru NVSL.</p>
<p>Treatment: Immediate antibiotic therapy. Numerous classes of antibiotics are effective: oxytetracycline, penicillins, aminoglycosides, fluoroquinolones, macrolides, and sulfonamides.</p>
<p>Prevention and control: Rapid detection and prevention of disease spread via quarantine and removal of affected animals. Vaccination of susceptible animals in enzootic areas. Move animals from potential contamination prior to periods of increased exposure. Do not use meat or animal products from uninspected or unknown sources, cases of sudden death, or emergency slaughters. Do not open carcasses in suspected cases. Do not contaminate soil during necropsy. Use protective clothing during necropsy. Post exposure antibiotics are recommended after exposure to aerosolized spores.</p>
<p>Suggested disinfectant for housing facilities: Cremation or deep burial of carcasses and contaminated materials. Disinfect using formaldehyde, oxidizing agents such as peroxides, 5% lye, quicklime (anhydrous calcium oxide), and bleach; however prolonged contact is required. A commercial product Mold Control 500® has been approved. Several protocols for large scale premise decontamination have been utilized. Formaldehyde (5%) can be used on soil if contamination is minimal otherwise soil removal is advised.</p>
<p>Notification: Reportable to USDA National Animal Health Reporting System (B051)</p>
<p>Measures required under the Animal Disease Surveillance Plan: None</p>
<p>Measures required for introducing animals to infected animal: Wait at least 21 days after outbreak is completed and quarantine varies although the recommended time is 21 days before movement is allowed.</p>
<p>Conditions for restoring disease-free status after an outbreak: Where the disease is known to be endemic, disease-free status will only be granted after an extended period, ~10 years, without cases and with surveillance. For normal sporadic cases, there should be at least 5 years without cases but with vaccination.</p>
<p>Experts who may be consulted: Ginger Harvey, DVM or Kristina Lantz, DVM National Veterinary Services Laboratories P.O. Box 844 Ames, IA 50010, USA 515-337-7070/515-337-7083 Fax: 515-663-7073 ginger.r.harvey@aphis.usda.gov / kristina.lantz@aphis.usda.gov</p>
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ASPERGILLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Vertebrates, invertebrates	Environment-acquired via spores. It is not considered contagious.	Primarily respiratory but can become systemic. Occasionally a cutaneous disease.	May cause severe disease in immune-compromised hosts.	Antifungal drugs - polyenes, azoles, allylamines, pyrimidines	Minimize environmental accumulation of fungus; prevent immune-suppression of host; prophylactic treatment	Only if spore-forming conidiospores are present

Fact Sheet compiled by: Joseph A. Smith

Sheet completed on: 1 February 2011; updated 15 July 2013

Fact Sheet Reviewed by: Mark Mitchell, Mark Papich, Patrick Redig, James Wellehan

Susceptible animal groups: Vertebrates and invertebrates can be affected. However, it affects primarily immunocompromised individuals (e.g. those young, geriatric, stressed, affected by concurrent disease, or undergoing management changes). Higher incidence of disease is associated with penguins, waterfowl, raptors, sea birds, and galliforms. Birds from polar or pelagic environments tend to be more susceptible. High environmental load of fungal spores is a predisposing factor for the development of disease, but exposure to ambient levels can also result in disease. Prolonged corticosteroid and antibiotic use have both been associated with increased risk of disease.

Causative organism: Primarily *Aspergillus fumigatus*, a saprophytic mold; occasionally *A. flavus*, *A. nidulans*, *A. niger*, and *A. terreus*. *A. flavus* traditionally is more associated with mycotoxin production (aflatoxicosis). Fungi use a nomenclature inconsistent with the rest of biology as separate names for asexual anamorph stages and sexual teleomorph stages of the same organism exist, resulting in multiple species names and paraphyletic taxa. The anamorph genus *Aspergillus* is the same fungus as several teleomorph genera, including *Neosartorya*, *Eurotium*, and *Emericella*.

Zoonotic potential: Aspergillosis is reported in people, but the infections usually are acquired from environmental exposure. Immunocompromised humans are more susceptible. Theoretically, any lesions where spore-forming conidia are present (e.g., some air sac granulomas in birds) may release spores into the environment which could be inhaled, and thus pose some zoonotic potential. In immunocompetent humans, the most common clinical presentation is fungal sinusitis.

Distribution: Worldwide distribution; ubiquitous in the environment. The fungus proliferates in soil, decaying vegetation, and moist environments with poor ventilation. Pre-formed spores can also be easily aerosolized in dry, dusty environments. Contaminated ventilation systems have been a risk factor for disease.

Incubation period: Highly variable. May cause acute disease or prolonged chronic infections. Clinical progression depends primarily on immune response and degree of environmental exposure.

Clinical signs: Primarily affects the respiratory system and may cause dyspnea, stridor, cyanosis, coughing, vocalization changes, and sneezing. The most common site of infection in mammals is the upper respiratory tract. The organism frequently infects cavities, such as sinuses, air sacs, guttural pouches, and similar locations. Signs of disseminated disease depend on the organs affected. Fungal plaques on large blood vessels may cause rupture and fatal hemorrhage. Nonspecific signs of disease such as lethargy, weakness, and weight loss are common. In birds, aspergillosis can result in marked leukocytosis.

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Post mortem, gross, or histologic findings:

Gross-Rhinitis, sinusitis, tracheitis, air sacculitis, pneumonia, disseminated granulomatous lesions in any organ. Lesions are yellow to pale pyogranulomatous or granulomatous nodules or plaques. In some air sac lesions, white or green, cottony spore-forming conidiospores can be observed.

Histologic-Granulomatous inflammation with intralesional fungal hyphae measuring 3-6 µm with parallel walls, evenly distributed septa, and progressive dichotomous branching at acute angles. Angioinvasion with thrombosis.

Diagnosis: Fungal culture combined with cytology or histopathology of affected tissues is the gold standard for a definitive diagnosis. The fungus can be cultured from normal tissues without pathologic lesions, so it is important to combine culture with microscopic evaluation. Fungi can be enhanced with special stains (e.g. periodic acid-Schiff [PAS], Grocott's methenamine silver [GMS]) or immunohistochemical labels to aid in microscopic evaluation. Other supportive diagnostics include PCR, serology, antigen blood tests (e.g. galactomannan), endoscopy, radiology, protein electrophoresis, and complete blood counts. Because of the ubiquity of this genus, serological results correlate poorly with disease.

Material required for laboratory analysis: Swabs or biopsies of affected tissues for culture, cytology, histopathology, and PCR. Serum or plasma for serology, antigen blood tests, and protein electrophoresis.

Relevant diagnostic laboratories: Almost any commercial diagnostic lab can perform fungal cultures, cytology, or histopathology. *Aspergillus* is readily cultured on a Sabaroud's dextrose plate incubated at 37° C for 48 hours. An *Aspergillus* diagnostic panel consisting of ELISA serology, galactomannan antigen testing, and protein electrophoresis is offered by the University of Miami Avian and Wildlife Laboratory.

The Fungus Testing Laboratory
 Department of Pathology, Room 329E. Mail Code 7750
 The University of Texas Health Science Center at San Antonio
 San Antonio, Texas 78229-3900
 210-567-4131
 210-567-4076

Treatment: Antifungal drug classes that have been used to treat aspergillosis include polyenes (amphotericin B), azoles (voriconazole, itraconazole, ketoconazole), allylamines (terbinafine), and pyrimidines (flucytosine). Newer echinocandins (caspofungin) and azoles (posaconazole, ravuconazole) are being used in human medicine. The effectiveness of azoles varies widely. Most isolates tested are susceptible to voriconazole. Terbinafine is synergistic with voriconazole, and a terbinafine/voriconazole combination is the current treatment of choice. Supportive care, treatment of concurrent disease, and removing any sources of stress or immunosuppression are also important components of treatment.

Prevention and control: Because clinical disease caused by *Aspergillus* spp. is typically caused by either high environmental exposure with or without immunosuppression, methods at prevention and control should be aimed at controlling these predisposing factors. Environmental sanitation, adequate ventilation, and air filtration can all help to reduce environmental fungal spore loads. Ensuring that substrates that support fungal growth, such as dead plant materials, are not present in the enclosure will reduce exposure. Enilconazole can be considered when environmental treatment is indicated. Commercial formulations of enilconazole (e.g., Clinafarm EC) have been developed to disinfect poultry facilities. Minimizing stress and concurrent disease can help reduce disease caused by immunosuppression. Prophylactic treatment using antifungal drugs (e.g., itraconazole) has been used during periods of stress or prolonged antibiotic use for highly susceptible species. Animals with aspergillosis should be investigated for other causes of immunosuppression.

Suggested disinfectant for housing facilities: Bleach is the most effective disinfectant. Efficacy of other classes of disinfectants is variable and may be species and strain dependent.

ASPERGILLOSIS

Notification: Not a reportable disease.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Aspergillosis is not considered a contagious disease. However, case clusters that mimic "outbreaks" can be caused by common environmental predisposing factors such as high environmental spore loads or environmental stressors. These environmental factors should be considered when introducing animals to the environment of an infected animal.

Conditions for restoring disease-free status after an outbreak: Not applicable.

Experts who may be consulted

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Animal Group(s) Affected	Transmission	Clinical Signs and Lesions	Severity	Treatment	Prevention and Control	Zoonotic
<p>Captive psittacine birds</p> <p>Canaries, finches.</p> <p>Wild free-ranging goose, swan, duck and gull species.</p> <p>Rare proven examples of broad species jumps, consistent histopathologic findings have been reported sporadically in a variety of avian species.</p>	<p>Primarily direct transmission, initial assumption of urofecal-oral route has been challenged by experimental work suggesting that tissue inoculation may be required.</p> <p>Viral shedding in urine, feces, choanal secretions and possibly feathers.</p> <p>Some evidence for vertical transmission to egg, not proven to live hatchlings.</p>	<p>Cause of psittacine “proventricular dilatation disease” (PDD). From asymptomatic to severe gastrointestinal signs and/or neurological signs leading to death.</p> <p>Gross PM: classic lesions are emaciation, dilation of crop, proventriculus or ventriculus, ventricular muscle atrophy, duodenal distension. No lesions may be present with primarily neurological forms.</p> <p>Histology: non-suppurative inflammation in peripheral, central and/or autonomic nervous systems.</p>	<p>Birds infected with ABV may or may not show clinical disease.</p> <p>Once clinical signs develop, avian bornaviral infection is generally considered a progressive disease which ultimately becomes fatal.</p> <p>Acute outbreaks with high mortality have been described in psittacine aviaries.</p>	<p>No specific treatment.</p> <p>Suppressing T lymphocyte function may improve clinical signs (e.g., cyclosporin)</p> <p>Inconsistent results with the use of various COX II NSAIDs.</p> <p>Antiviral drugs inadequately investigated.</p> <p>Supportive and symptomatic treatment and good husbandry can prolong life.</p> <p>Possibility of complete cure is not certain.</p>	<p>No vaccine.</p> <p>Avoid introducing infected birds into new flocks.</p> <p>Excellent husbandry practices, strict quarantine protocols including determining the disease and ABV status of all newly introduced birds.</p> <p>Isolate infected or exposed birds.</p> <p>Standard disinfection protocols should be effective (enveloped virus).</p>	No.

Fact Sheet compiled by: Pauline Delnatte, Dale A. Smith

Sheet completed on: September 1, 2018

Fact Sheet Reviewed by: Michael Lierz, Ian Tizard

Susceptible animal groups (affected by different viral genera):

Psittacine birds: Infection by *Psittaciform 1* and *2 orthobornaviruses* has been reported in more than 80 species of captive psittacine birds in at least 33 different genera. Certain species, such as African grey parrots, blue and gold macaws, cockatoos, and Amazon parrots, seem most frequently affected.

Passerine birds: *Passeriform 1* and *2 orthobornaviruses* have been described from captive canaries (*Serinus canaria*), a Bengalese/Society finch (*Lonchura striata domestica*) and 3 estrildid finches.

Anseriformes, Laridae: *Waterbird 1 orthobornavirus* infection was initially recognized in North America in free-ranging mute (*Cygnus olor*) and trumpeter swans (*Cygnus buccinator*), and in Canada geese (*Branta canadensis*). This and related viruses have since been identified in a range of wild goose and duck species, as well as in several species of gulls. Investigations in Europe have identified *Waterbird 1 orthobornavirus* infections in three additional species of geese in Denmark and a Eurasian oystercatcher (*Haematopus ostralegus*) in Germany.

Single reports of what are interpreted as broad “breaks” in fidelity of a given virus for a given host group have

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been reported, thus molecular sequencing of bornaviruses from avian host groups or species not listed above is highly recommended.

Causative organism: The first avian-associated bornavirus (ABV) was identified in 2008 as the cause of PDD in psittacine birds. Bornaviruses are enveloped, single-stranded, RNA viruses in the family *Bornaviridae*. There are currently five species of bornaviruses affecting birds: *Passeriform 1 orthobornavirus* (canary bornaviruses 1-3), *Passeriform 2 orthobornavirus* (estrildid finch bornavirus), *Psittaciform 1 orthobornavirus* (parrot bornaviruses 1-4, 7), *Psittaciform 2 orthobornavirus* (parrot bornavirus 5), and *Waterbird 1 orthobornavirus* (aquatic bird bornaviruses 1-2). Parrot bornaviruses 2 and 4 are the most commonly identified viruses in psittacine birds. Additional viruses await formal classification.

Zoonotic potential: None reported

Distribution: *Parrot bornaviruses:* PDD, the originally-recognized clinical syndrome of ABV infection in psittacine birds, was first identified in the late 1970's in the United States. ABV infection and associated disease have been described in captive psittacines around the world. Worldwide dissemination is assumed to have resulted from the trade in captive birds. While there is some evidence for the presence of exposure of wild parrots to an ABV in South America, a reservoir in wild psittacine birds has not been confirmed. *Canary bornaviruses:* Reports suggest these viruses are prevalent in captive European canaries. *Aquatic bird bornaviruses:* Infection, initially recognized as widespread in North America, has also been reported from free-ranging wild birds in Denmark and Germany. A lack of reports from other parts of the world likely reflects a lack of dedicated investigation.

Incubation period: Poorly investigated but appears extremely variable. Reports suggest a minimum of 11 days under experimental conditions up to months or years under natural conditions. There is a suggestion of an acute form (birds die within days or weeks after acute onset of symptoms) and of a persistent form (birds are able to live for years without clinical impairment), likely this simply reflects a continuum.

Clinical signs: Descriptions of PDD in psittacine birds predate the discovery of ABV. Birds infected with ABV may or may not develop clinical disease. Clinical signs result from pathology in the autonomic, central, and/or peripheral nervous systems, and vary in nature, severity and duration. Non-specific signs include depression, lethargy, weight loss, muscle atrophy, abdominal enlargement, polyuria and polydipsia, as well as sudden death. Classic gastrointestinal signs associated with myenteric plexus dysfunction include dysphagia, crop stasis, regurgitation, impaction, maldigestion (passage of undigested seeds), and progressive loss of body condition. Central and peripheral nervous system signs include changes in awareness and demeanor, tremors, seizures, erratic head movements, torticollis, head-pressing, opisthotonos, abnormal gait and posture, inability to perch, proprioceptive and motor deficits, ataxia, paralysis, *status epilepticus*, and ophthalmologic abnormalities (dilated pupils, anisocoria, chorioretinitis, retinal degeneration, and blindness). Change in behaviour has also been noted. The factors that govern the development of clinical disease in ABV positive birds are not known, but are likely related to features of the host immune status as well as of the infecting virus. Affected birds can develop secondary opportunistic infections that increase mortality.

Post mortem, gross, or histologic findings: Consistency among clinical signs and gross and histologic findings is not always present. Gross lesions include mild to severe emaciation, atrophy of the pectoral, proventricular and ventricular muscles, proventricular and ventricular dilatation and duodenal distension. Proventricular rupture and resulting peritonitis have been rarely reported. One report described accumulation of fluid in the subarachnoid space. Occasionally, no gross lesions are observed. Microscopic lesions consist of non-suppurative inflammation in peripheral, central and autonomic nervous tissues. Similar infiltrates may also be present in adrenal glands and myocardium. Cerebellar Purkinje cell necrosis, neuronophagia, myelin degeneration, gliosis and axonal swelling can accompany the inflammatory lesions.

Diagnosis: PDD as a clinical entity and infection with ABV are not synonymous. Histological lesions in biopsy or post-mortem samples remain the gold standard for diagnosis of PDD. Detection of virus/viral RNA/viral antigen or antibodies against ABV provides evidence of infection or exposure, but does not differentiate among patients with clinical PDD, asymptomatic shedders and previously exposed birds. There is no standardized

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screening protocol for PDD and/or ABV infection in psittacine flocks, but screening programs involving separation of birds based on a combination of repeated RT-PCR assessment of choanal and/or cloacal swabs (feather sampling has also been recommended) and serology have been used to derive flocks clear of ABV infection.

Clinical signs and pathologic lesions: ABV infection should be considered as a differential diagnosis for clinical signs referable to the digestive and/or nervous systems. Prior to the identification of the avian bornaviruses, techniques for the antemortem diagnosis of PDD included plain and contrast radiography, contrast fluoroscopy, and crop biopsy. Crop biopsy has a variable and often low sensitivity.

Detection of virus, RNA or antigen:

Reverse Transcriptase Polymerase Chain Reaction (RT-PCR): Both gel-based and real time RT-PCR have been developed using primers for various segments of the ABV genome, particularly the N and M genes. Not all primers will detect all bornaviruses that affect avian species.

Immunohistochemistry (IHC): IHC can be used to identify ABV antigen in formalin fixed biopsy or necropsy specimens. Moderately intense, diffuse, intracytoplasmic staining accompanied by intranuclear staining in neurons and certain epithelial cells is considered positive for viral antigen presence.

Virus culture: Various genotypes of ABV have been successfully grown in a range of avian cell lines.

Cytopathic effects do not occur, thus virus must be demonstrated by Western blot, immunohistochemistry, indirect immunofluorescence or RT-PCR.

Sequence analysis: The recognition of an increasing number of bornaviral species and genotypes makes genome sequencing a critical component in the diagnosis of ABV infection and of PDD.

Detection of antibodies: Indirect immunofluorescence (using infected cell cultures which present multiple antigens), Western blot, and indirect ELISA assays using various sources of monoclonal primary antigen have been used in psittacine birds and waterfowl. Several private laboratories offer serologic testing, which is also used as a research tool. Test specificity and sensitivity are difficult to determine and compare due to the absence of a gold standard for diagnosis, and the relatively poor correlation between the presence of antibodies, fecal shedding of ABV, and the presence of pathologic lesions and/or clinical disease. Considerable research is required before we will understand which viral proteins are the most immunogenic, and the role of antibodies in resistance to infection and to the development of clinical signs.

Material required for laboratory analysis:

Ante-mortem: Crop tissue (histology +/- IHC +/- RT-PCR); choanal and cloacal swabs, feces (less sensitive than cloacal swabs), and possibly calami of plucked chest contour feathers (RT-PCR); plasma or serum (serology).

* Pooling multiple cloacal swabs or droppings from a single bird over several days or samples from multiple birds in an aviary increases test sensitivity as shedding of virus is frequently intermittent.

Post-mortem: Brain, proventriculus, ventriculus, adrenals and vitreous of the eye (most consistently infected tissues) (RT-PCR +/- sequencing); brain, proventriculus, ventriculus (histology +/- IHC). As lesions vary in location and severity, submission of a full suite of tissues is highly recommended.

*RT-PCR can also be performed on formalin-fixed paraffin embedded (FFPE) tissues in some laboratories.

Relevant diagnostic laboratories:

Histopathologic assessment: can be performed by any veterinary pathology diagnostic service.

RT-PCR testing: can be carried out by any molecular laboratory with appropriate primers. Information on test validation and primer selection should be requested.

- Animal Health Laboratory, University of Guelph, Guelph, Ontario, Canada. (**AAVLD accredited**)
<http://www.guelphlabservices.com/ahl/>
- Diagnostic Services, Faculté de médecine vétérinaire, University of Montréal, St. Hyacinthe, Quebec, Canada. (**AAVLD accredited**)
<http://servicedediagnostic.com>

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- Infectious Disease Laboratory, University of Georgia, Athens, Georgia, USA.
<http://www.vet.uga.edu/idl/index>
- Veterinary Molecular Diagnostics Inc, Milford, Ohio, USA
www.vmdlabs.com/

Commercially available RT-PCR and ELISA testing, offered as a panel:

- Animal Genetics Inc. (USA), Tallahassee, Florida, USA; (Europe), Cornwall, England.
<http://animalgenetics.com>

RT-PCR, Real-time RT-PCR, Serology, Sanitation of flocks, located in Europe:

Clinic for Birds. Reptiles, Amphibians and Fish, Justus-Liebig University Giessen, Germany,
https://www.uni-giessen.de/fbz/fb10/institute_klinikum/klinikum/kvraf
email: kvraf@vetmed.uni-giessen.de

Treatment: No specific treatment exists. Supportive and symptomatic therapy may prolong life for months to years.

Non-steroidal anti-inflammatory drugs: Celecoxib, tepoxalin and meloxicam have been recommended but evidence of effectiveness has been inconsistent. A study in cockatiels suggested that the use of meloxicam would actually be detrimental to birds affected with PDD.

Immunosuppressive protocols: May be of therapeutic benefit, especially selective T-cell elimination. Cyclosporine appears to hold the most promise, based on limited research.

Antiviral drugs: Have been described as beneficial by some authors (e.g., amantadine), but have been reported as having no apparent effect on fecal shedding of virus by others. Ribavirin reduces viral replication in tissue culture but does not appear to have a measurable effect on viral shedding *in vivo*. Most recent research suggests favipiravir may be able to eliminate Borna disease virus 1 and parrot bornavirus 4 from infected cultured cells. *In vivo* studies have not been carried out.

Prevention and control: Preventive measures are intended to avoid introduction of an ABV into new flocks and include excellent husbandry and sanitation practices and strict quarantine protocols. Screening of birds in quarantine should include a combination of PCR and serologic testing (see Diagnosis above). The interpretation of test results can be challenging as diagnostic test protocols vary among laboratories, and an understanding of the biology of the disease is necessary for interpretation (e.g., intermittent shedding, asymptomatic carriers, testing of non-psittacine species, etc.). The possibility of vertical transmission of ABV complicates the management of infected aviaries. Pairing ABV positive birds, incubating their eggs artificially, and hand-raising the chicks separately until their ABV status is determined may be a viable option for critically endangered species. There is currently no vaccine against ABV infection. Recent studies have suggested that serum antibodies are not protective, and that persistent infection is a result of ABV escaping recognition by the innate immune system. Early research on vaccine development has been published, with reduction of infection (and hence clinical disease) in one study, and prevention of clinical disease (but not infection) in another.

Suggested disinfectant for housing facilities: Although there are no data on environmental survival of the ABVs or sensitivity to disinfectants, they are assumed to have the same stability as other enveloped RNA viruses of similar size and structure. Disinfection with phenols, formaldehyde or hypochlorites is thus recommended.

Notification: None legally required; information regarding the infection and exposure status of birds being transferred between institutions is recommended.

Measures required under the Animal Disease Surveillance Plan: Not applicable.

Measures required for introducing animals to infected animal: The mixing of infected and non-infected birds is not recommended. There is no known strategy to prevent viral transmission between in-contact birds. Excellent hygiene appears to prevent spread within a facility containing infected and uninfected birds.

Conditions for restoring disease-free status after an outbreak:

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There is no system of “ABV-free” certification. Clearing infection from a flock requires a rigorous program of repeated cycles of testing using both PCR and serology, separation of birds positive for virus or antibodies, and retesting. Reliance on PCR testing when serologic testing is not available makes recognition of infected birds more difficult. Some populations of captive birds appear to have a very high prevalence of infection.

Experts who may be consulted:

- Dr. Dale A. Smith, Professor, Department of Pathobiology, Ontario Veterinary College, University of Guelph, Canada (dalesmit@uoguelph.ca)
- Schubot Exotic Bird Health Centre, Department of Veterinary Pathobiology, Texas A&M University, USA. Contact research group through Dr. Ian Tizard, Professor. (ITIZARD@cvm.tamu.edu)
- Dr. Monika Rinder, Clinic for Birds, University of Munich, Oberschleissheim, Germany.
- Dr. Michael Lierz, Clinic for Birds, Reptiles, Amphibians and Fish, Faculty of Veterinary Medicine, Justus Liebig University Giessen, Giessen, Germany.

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Birds, predominantly carried by waterfowl and shorebirds, various mammals	Fecal-oral and fecal-cloacal (i.e., contaminated water), airborne and direct contact through mucous membranes, ingestion of infected tissues, fomites and mechanical vectors	LPAI- typically asymptomatic HPAI- Respiratory, digestive or nervous system signs, sudden death	Asymptomatic to fatal in all animals affected	Anti-viral drugs in humans	Preparedness protocol including guidelines for facility during an outbreak, surveillance techniques and biosecurity protocols. Minimize contact between captive birds and wild birds. Quarantine new birds for at least 30 days.	Yes

Fact Sheet compiled by: Rae Gandolf

Sheet completed on: 1 January 2011; updated 1 November 2012

Fact Sheet Reviewed by: Carol Cardona, Walter Boyce

Susceptible animal groups: Avian influenza viruses can infect a wide variety of species. Whereas aquatic birds typically exhibit few signs of infection, once the virus spreads to poultry it can become more virulent and can potentially cause severe disease in mammalian species that may come in contact with them.

Aquatic birds- migratory waterfowl (Anseriformes) and shore birds (Charadriiformes) act as the major natural reservoir species; infection is typically asymptomatic in ducks infected with the low pathogenicity viruses (LPAI); wild birds have only rarely been infected with high pathogenicity viruses (HPAI).

Poultry, other gallinaceous birds- typically mild clinical signs or subclinical with LPAI; some viruses (of the H5 or H7 subtypes) may mutate to HPAI while circulating in a flock, potentially resulting in very high mortality.

Mammals and other avian species- HPAI H5N1 exhibits a wide and growing host range including humans; LPAI viruses may infect mammals and other avian species but infections are typically undetected because they are asymptomatic. Examples of species with confirmed HPAI include primates, suids, felids (domestic house cats and several nondomestic species), pinnipeds, canids (raccoon dogs, rarely in domestic dogs), viverrids (palm civets), mustelids (ferrets, stone martens, mink), lagomorphs (pikas, rabbits), rodents, whales and a broad range of avian species including psittacines.

Causative organism: Influenza A, an enveloped RNA virus in the family Orthomyxoviridae. Influenza A viruses are classified according to subtypes, based on two surface proteins (hemagglutinin (H) and neuraminidase (N)). Avian influenza viruses are further classified according to their virulence in chickens

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(HPAI or LPAI). Although they are classified as LPAI in chickens, some isolates can still cause disease in other species.

Disease significance:

- Appearance and spread of HPAI H5N1 in poultry has increased the risk of spillover into human and non-human hosts.
- Poultry farms can sustain very high mortality and morbidity, leading to high costs and trade restrictions on poultry products
- Zoonotic infections of humans may lead to the development of viruses with pandemic potential, especially HPAI H5N1
- LPAI and HPAI viruses may emerge and cause disease in captive and free-ranging wildlife species

Zoonotic potential: Yes

Distribution: LPAI viruses occur worldwide in migrating birds and poultry. Infections have been confirmed in Africa, Asia, Australia, Europe, North America and South America. New HPAI viruses emerge periodically in poultry and HPAI H5N1 has become established in several Asian and African countries

Incubation period: Highly variable; Humans: typically 2-7 days (up to 17 days), poultry: 1-7 days, wild birds: typically 1-7 days. However, the actual incubation period of a given virus in any species (i.e., 9000 species of birds) will vary based on host and virus.

Clinical signs: This virus can infect the respiratory, digestive, or nervous systems, alone or in combination, depending on the host. Signs correlate with the location of the infection, and vary depending on viral subtype, environmental factors, age, health status and species.

LPAI (birds) - asymptomatic to conjunctivitis and mild respiratory symptoms (free-ranging and domestic species), decreased egg production (documented in domestic poultry, may apply to other species)

HPAI (birds)- sudden death of large numbers of birds, especially in poultry; may also see any of the following: marked depression, sinusitis, lacrimation, cyanosis of the head, edema of the head, green to white diarrhea, coughing, sneezing, blood-tinged oral and nasal discharges, podothelial ecchymoses, neurologic disease, decreased egg production, loss of egg pigmentation and deformed or shell-less eggs.

HPAI (mammals)- pyrexia and difficulty breathing or rapid breathing are typically the initial symptoms and may be followed by conjunctivitis, coughing, mucosal bleeding, diarrhea, vomiting, abdominal pain, neurologic signs, multi-organ failure, DIC and death. Morbidity and mortality are variable. Among zoo animals, fatal cases were reported among captive tigers and leopards in Thailand, but captive leopards, tigers, Asiatic golden cats and lions at a wildlife rescue center in Cambodia all recovered after an illness lasting 5-7 days.

Post mortem, gross, or histologic findings: Highly variable in birds, ranging from no lesions in peracute deaths to subcutaneous edema on the head and neck; edema and subcutaneous hemorrhages on the feet; fluid in the nares and oral cavity; conjunctivitis; hemorrhagic tracheitis; lung hemorrhage and congestion; petechiae throughout the abdominal fat, over serosal surfaces and peritoneum; congested kidneys sometimes plugged with urate deposits; hemorrhagic or degenerated ovaries with areas of necrosis; yolk from ruptured ova within the peritoneal cavity; yolk peritonitis and air sacculitis. It is important to note that the occurrence of peritonitis, tracheitis, edema of the wattles or neck, or petechial hemorrhages in the proventriculus may be particularly suggestive of an HPAI infection. Findings in mammals infected with HPAI are also broad-ranging and may include pulmonary congestion and edema, conjunctivitis, multi-organ congestion, widespread internal hemorrhages, encephalitis and myocarditis.

Diagnosis:

- Virus isolation and/or RT-PCR assays can identify avian influenza viruses in clinical samples. These tests can also distinguish some viral subtypes.

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- Viral antigens and antibodies can also be detected with ELISAs including rapid tests. As of 2008, the World Organization for Animal Health (OIE) recommended that antigen detection tests be used to identify avian influenza only in flocks and not in individual birds.
- Serologic tests including agar gel immunodiffusion, hemagglutination inhibition and ELISAs are useful as supplemental tests. Blocking or competitive ELISAs are species independent and can be very useful for detecting prior exposure to AI virus in wild birds. AGID may be insensitive in some avian species and HI requires the proper viral antigen to be useful. Serology is not useful in the diagnosis of HPAI in susceptible species because they will die before they seroconvert.

Material required for laboratory analysis: Oropharyngeal, tracheal or cloacal swabs or, in small birds or for surveillance, feces may be used in live birds; additionally, organ samples (trachea, lungs, air sacs, intestine, spleen, kidney, brain, liver and heart) can be tested in dead birds. Links to sample collection protocols for the National Wildlife Health Center lab may be found on the USGS avian influenza page:

http://www.nwhc.usgs.gov/disease_information/avian_influenza/

Relevant diagnostic laboratories: Diagnostic testing for avian influenza is generally performed by specialized county, state, regional, or national laboratories, such as the USDA-approved laboratories in the National Animal Health Laboratory Network (NAHLN) (NAHLN@aphis.usda.gov; 515-663-7731). Authorities should be consulted regarding regulations for sending samples to authorized diagnostic laboratories.

Treatment: Four antiviral drugs - amantadine, rimantadine, zanamivir, and oseltamivir - are active against selected human influenza viruses. Studies suggest that these drugs may also be helpful in avian influenza infections in humans although many currently circulating strains are resistant to amantadine and rimantadine. In poultry, HPAI is managed primarily by flock eradication but LPAI may be managed with vaccination, eradication or quarantine.

Prevention and control: Each institution should have general preparedness protocol including advance communication with regulatory officials regarding potential courses of action, guidelines concerning the operation of the facility during an outbreak, surveillance techniques for captive animals and wildlife on the premise, preventative measures to protect public health, vaccination planning and biosecurity protocols (hand washing, disinfecting, quarantine, etc.). Additionally:

- Staff should be provided with information regarding human health precautions and trained for proper use of personal protective equipment.
- Case definition criteria for avian influenza should be established for captive species in order to identify the disease early and institute the biosecurity protocol.
- A testing plan should be established and a laboratory where the testing will be done should be identified.
- The entire collection should be catalogued as influenza susceptible or resistant based on the likelihood of infection in the event of exposure. The expected clinical appearance of infection of the susceptible birds and mammals in the collection should be recorded to prepare for a possible outbreak.
- Minimize contact between captive birds and wild birds
- Quarantine new birds for at least 30 days
- In the face of an outbreak, captive birds could potentially be vaccinated. > 25,000 captive birds were vaccinated with a H5N2 inactivated vaccine in European zoological facilities since 2005. Most birds seroconverted following the second booster vaccination, and semi-annual to annual vaccination is recommended. A negative correlation exists between antibody response and increasing mean body weight. Some species (pelicans and owls), may fail to respond to vaccination. Different species may have differing responses to vaccination, including duration of immunity, which may require regular serologic monitoring and additional booster vaccinations. Approval for zoological institutions to administer vaccinations to birds

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in the U.S. will be conditional and overseen by a federally accredited veterinarian. Restrictions on transporting vaccinated birds or releasing them back into the wild may be imposed.

Suggested disinfectant for housing facilities: Avian influenza viruses are relatively unstable in the environment and are inactivated by extremes in pH, heat, and dryness. The virus may persist for a long time in cool aquatic environments. The virus may survive over 100 days in cool fresh water and indefinitely when frozen. In the presence of organic matter, AI virus can be inactivated by aldehydes. After removal of organic matter, several classes of disinfectants are effective at destroying avian influenza virus: phenolics (One Stroke Environ), quaternary ammonium compounds (Roccal), oxidizing agents (Virkon), dilute acids (eperacetic acid), and bleach.

Notification: Any suspect cases should be reported to the state veterinarian or USDA Veterinarian (USDA Veterinary Services:1-866-536-7593)

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Not recommended

Conditions for restoring disease-free status after an outbreak: No official disease-free status offered

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AVIAN POXVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Avian	Mechanical spread by invertebrate vectors. Direct contact between birds or indirect contact with contaminated surfaces.	Cutaneous or “dry” form: skin nodules. Diphtheritic or “wet” form: internal lesions in upper alimentary or respiratory tracts. Systemic infection.	Small, focal skin lesions to widespread severe lesions; respiratory difficulties, to peracute death in certain species.	Treat secondary bacterial infections. May need to provide supportive fluids and food.	Vector control and good hygiene.	No

Fact Sheet compiled by: Sharon L. Deem

Sheet completed on: updated 31 July 2018

Fact Sheet Reviewed by: Kristi Delaski

Susceptible animal groups: Avians. Over 275 species of birds in 23 orders are known to be susceptible.

Causative organism: Avipoxvirus in the family Poxviridae. Large (up to 400 nm) double-stranded, enveloped DNA. 17 types of *Avipoxvirus* spp. have been identified to date.

Zoonotic potential: No

Distribution: Worldwide with exception of no published reports from the Arctic or Antarctic.

Incubation period: Variable with approximate range of 4 days up to several months.

Clinical signs: The signs vary with virulence of the virus, susceptibility of the host, distribution and type of lesions in an infected bird, and other complicating factors. Manifestations are cutaneous (“dry”), diphtheritic (“wet”), systemic, or some combination of the three. Cutaneous lesions are characterized by the appearance of nodular lesions on feather-free regions of the body; in editor’s experience, often in non-gallinaceous species, these lesions occur as single nodules which may resemble a proliferative neoplasm. Diphtheritic lesions are moist, necrotic lesions on the mucous membranes of the mouth and upper respiratory tract. Septicemic form is associated with acute depression, anorexia, dyspnea and death and has been most frequently reported in certain passerine species (e.g., canary). Infected birds can have peracute infections (death) or may become latent carriers. Also note that when stressed (e.g., during transfer intra- and inter-zoo, other illness), it has been suggested that birds may recrudescence and develop new lesions which may first appear as red-swollen areas. Any bird with these developing lesions should be immediately separated from other birds and caged individually while avian pox is or is not confirmed.

Post mortem, gross, or histologic findings: Gross lesions are proliferations of epithelial cells. Diphtheritic form may appear as white, opaque, slightly elevated nodules to coalescing yellowish, caseous, necrotic material with the appearance of a pseudomembrane.

Diagnosis: Gross lesions in cutaneous infections are often highly suggestive of pox infection but are not definitively avian pox. Diphtheritic infections are often harder to diagnosis on gross observations due to differential diagnoses (e.g., trichomonosis). Histologic evaluation for Bollinger bodies (eosinophilic intracytoplasmic epidermal inclusions) on light microscopy is acceptable for diagnosis. Virus isolation on the chorioallantoic membrane of embryonated chicken eggs or in cell cultures of avian origin may be used. PCR techniques are also available for detection of avian pox DNA from DNA-extracted direct from lesion or extracted virus culture. PCR targeting the 4b core protein has been widely used for construction of avian poxvirus phylogenies and virus strain differentiation.

Material required for laboratory analysis: Biopsy of cutaneous nodules and diphtheritic mucous membranes for detection of the pathognomonic Bollinger bodies.

AVIAN POXVIRUS

Relevant diagnostic laboratories:

State or university veterinary diagnostic laboratories in most states can perform diagnostic testing.

National Wildlife Health Center
6006 Schroeder Road
Madison, WI 53711-6223.
Phone: (608) 270-2400
Fax: (608) 270-2415

National Animal Disease Center
P.O. BOX 70
1920 Dayton Avenue
Ames, IA 50010

Treatment: No direct treatment for virus infection itself exists. However, secondary bacterial infections should be treated. Supportive care may be needed to provide supplemental food and water for those birds that cannot see or eat properly.

Prevention and control: Mechanically transmitted virus, therefore control of vectors (e.g., mosquitoes, flies) and fomites is very important. Perch design and cage structure important to minimize cross infections and to decrease abrasions that allow entry of the virus.

Suggested disinfectant for housing facilities: Any strong disinfectant, including bleach.

Notification: None required.

Measures required under the Animal Disease Surveillance Plan: None required.

Measures required for introducing animals to infected animal: Keep birds with pox lesions in quarantine until no clinical signs are present.

Conditions for restoring disease-free status after an outbreak: Clean with common disinfectants (e.g., bleach) and keep mosquitoes and other mechanical vectors to a minimum.

Experts who may be consulted:

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BABESIOSIS

Animal group (s) affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ubiquitous in wildlife wherever there are tick infestations. Variety of mammal species, birds, humans	Numerous species of Ixodid ticks; some evidence for direct (blood-blood contact) transmission of <i>Entopoloypoides</i> in primate colonies.	Severe hemolytic anemia, hemoglobinemia, hemoglobinuria, fever, possible neurologic signs, anorexia, slight jaundice, or subclinical. Majority of infections in wildlife are subclinical.	May be severe, with acute clinical presentation and death. Clinical disease often less severe in free-ranging animals than domestic animals.	Imidicarb, tick control	Tick control is primary means of preventing Babesia infection; host immunity through exposure.	<i>Babesia microti</i> , carried by wild rodents, has caused human infection

Fact Sheet compiled by: Tiffany M. Wolf

Sheet completed on: December 16, 2012, updated February 27, 2013, updated January 9, 2018

Fact Sheet Reviewed by: Arno Wünschmann, Ulrike Munderloh, Sam Telford

Susceptible animal groups: Most mammal orders, not in marine mammals (Cetacea, Pinniped), several avian species, humans.

Causative organism:

Babesia bovis, *bigemina*, and *odocoilei* (ungulates); *B. caballi* and *B. equi* (horses, renamed *Theileria equi*); *B. canis*, *gibsoni*, *annae* (canids); *B. lotori* (raccoons); *B. mephitis* (striped skunk); *B. microti* (rodents); currently there are 14 distinct avian *Babesia* species. *Babesia sp.* found outside of North America may be encountered in the zoological setting in animals that are directly imported from other countries. Additionally, *Entopoloypoides macaci*, which is closely related to *B. microti*, is often identified in colonies of research primates, such as rhesus macaques, African monkeys, and baboons, and will infect other species of primates.

Zoonotic potential:

Illness caused by *B. microti* in humans is typically mild or inapparent, but *B. microti* does infect and can cause significant illness in immune-intact persons, though illness tends to be more severe in immune-compromised persons. Other species such as *B. divergens*-like MO-1 and *B. duncani* are known to cause disease mainly in immunocompromised people. More severe, and often fatal, babesiosis occurs in splenectomized people.

Distribution:

Typically follows that of the tick vector: *B. bovis* and *B. bigemina* are transmitted by *Rhipicephalus microplus* and *R. annulatus* respectively and found in Mexico and occasionally southern Texas and California. *B. odocoilei* is transmitted by *Ixodes scapularis* and *I. pacificus* which are found in eastern half of U.S. and Canada (*I. scapularis*) and Pacific coast of U.S. and Canada (*I. pacificus*). *B. caballi* and *B. equi* was eradicated from the U.S. and is absent in Canada. *B. canis* and *B. gibsoni* are transmitted by *R. sanguineus* and found throughout most of the U.S. and southeastern Canada. *B. lotori*'s tick vector is unknown, but found in eastern U.S., Texas and California. *B. annae* has been reported from raccoons and foxes in Massachusetts. *B. mephitis* has been reported in skunks in Maryland. *B. microti* is transmitted by *I. scapularis* and found in northeastern and upper Midwest U.S. The geographic distribution of avian *Babesia* species is not fully understood.

BABESIOSIS**Incubation period:**

B. bovis and *B. bigemina* – incubation is generally 2-3 weeks post-tick infestation, and from 5 days to 3 weeks post-blood inoculation, depending on dose of inoculum. Ticks must feed for 2-3 days for successful transmission of *B. canis*. Incubation period for humans is reported as 1-6 week from beginning of tick feeding. Chronic infections may recrudescence if an animal is stressed or becomes immunocompromised for any reason.

Clinical symptoms:

Nonspecific clinical signs include fever, anorexia, depression and lethargy, lymphadenopathy. Erythrocyte destruction by the parasite and host immune response results in mild to severe hemolytic anemia, icterus, hemoglobinemia, hemoglobinuria, splenomegaly. In rare cases where *Babesia*-infected erythrocytes obstruct brain capillaries, neurologic signs may be noted.

Post mortem, gross, or histologic findings:

Pathologic findings may include icterus, generalized lymph node enlargement, hepatomegaly and splenomegaly (due to red pulp hyperplasia), abomasal mucosal ulcerations, hemorrhage into the intestinal tract, and dark red kidneys (hemoglobinuric nephrosis). Edema and hemorrhage of tissues such as the cardiac muscle, intestinal serosa, and lymph nodes may be observed, as well as fluid in the body cavities and pericardial sac. The bladder is frequently distended with dark red urine. For fulminating ruminant infections, Giemsa-stained brain crush smears are helpful to detect parasitized erythrocytes in brain capillaries. Also, the spleen often contains large numbers of parasitized cells, which may be appreciated on impression smears taken from cross sections of the spleen.

Diagnosis:

Microscopic visualization of piroplasms within erythrocytes in Giemsa, Wright's or Diff-Quick®-stained thin or thick whole blood smears. Piroplasms become more difficult to find on blood smears after the acute phase of infection passes. Serologic tests (cELISA, IFA, CFT), nucleic acid probes and PCR are also available. Gross splenomegaly is a common finding, particularly in naïve or unnatural hosts. Impression smears of spleen may be made for the identification of parasitized cells.

Material required for laboratory analysis:

Whole blood (EDTA) for smears and PCR, serum for serological testing.

Relevant diagnostic laboratories:

Several veterinary diagnostic laboratories offer serologic and PCR testing for *B. bovis*, *B. bigemina*, *B. caballi*, *B. equi*, *B. canis*, and *B. gibsoni*. Research labs with *Babesia* expertise are good options to work up samples.

Treatment:

Treatment is most successful in the early phase of the disease. Chemotherapy may not completely eliminate infection and may be unsuccessful in the later stages of the disease. Imidocarb dipropionate (1mg/kg IM), diminazene aceturate (3mg/kg IM), phenamidine diisethionate (8-13 mg/kg), and amicarbalide diisethionate (10 mg/kg IM), have been used to treat babesiosis in artiodactylids. Similarly, imidocarb, diminazene, and phenamidine have also been utilized to treat *B. canis* and *B. gibsoni*. Primaquine phosphate is preferred treatment in felids and birds. Quinine and clindamycin, or atovaquone and azithromycin are used to treat zoonotic babesiosis and might be tried for nonhuman primate infections. In addition to specific therapy, supportive care with fluids, blood transfusions, iron, and antibiotics may be important as well. Supportive therapy may be contraindicated in severely anemic animals that are easily stressed with handling.

Prevention and control:

Free-ranging animals sharing zoo habitats are often already infected with *Babesia* as well as vector ticks. The primary means of controlling outbreaks is through control of the tick vector. Elimination or reduction of tick

BABESIOSIS

infestation may be accomplished via application of acaricides, prophylactic use of chemotherapeutics, range burning, prolonged pasture rest, and repellents. Additionally, care should be taken to prevent accidental transmission through the transfer of infected blood between animals via routine surgical or vaccination procedures. Vaccines of noninfectious material have been developed, and although they do not prevent infection, they do ameliorate the severity of disease. Additionally, differences in strain antigenicity limit cross-protection by the vaccine.

Suggested disinfectant for housing facilities:

Disinfectants are generally not effective in preventing the spread of babesiosis. However, standard measures should be taken to prevent the transfer of infected blood between animals. *R. sanguineus*, the vector for canine babesiosis, is typically found indoors in kennels and other housing situations; such facilities should be treated with appropriate acaricides.

Notification:

Babesiosis caused by *B. bovis*, *B. bigemina*, *B. equi* and *B. caballi* are reportable diseases and state and federal authorities must be notified immediately of infection. Public health officials may need to be notified if zoonotic infection has occurred or is suspected.

Measures required under the Animal Disease Surveillance Plan:

Equine piroplasmiasis is considered a foreign animal disease in the U.S., therefore any equids imported must be serologically screened by the National Veterinary Services Laboratory using the competitive enzyme-linked immunosorbent assay (cELISA) prior to importation. Current information regarding the USDA's requirements for disease surveillance can be found at <https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/monitoring-and-surveillance>.

Measures required for introducing animals to infected animal:

Animals that have been treated for and survive infection should be considered chronic carriers. The most important means of preventing transmission is through vector control. A premunition approach may be an alternative strategy for introducing naïve animals into endemic areas for conservation purposes.

Conditions for restoring disease-free status after an outbreak:

Disease-free status may not be realistic, particularly where wildlife is involved in the maintenance of endemnicity.

Experts who may be consulted:

Sam Telford III, Department of Infectious Diseases and Global Health, Tufts Cummings School of Veterinary Medicine

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Document No. A0304.0600.

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BALANTIDIUM COLI

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Suids Primates, especially captive great apes, and humans	Fecal-oral Contact with infected suids or primate, or water contaminated by same; environmental transmission	Asymptomatic Mild cases present recurrent watery and mucoid diarrhea, abdominal pain. Acute severe typhlitis/ulcerative colitis with anorexia, dysentery, lethargy, vomiting, diarrhea.	Ranges from asymptomatic carriers to severe diarrhea in susceptible species. Invasive form of the disease is considered fatal, and occasionally can be extra-intestinal.	Paromomycin, metronidazole, tetracyclines, iodoquinol; supportive care	Avoid contact with infected suids or carriers, or water/food contaminated with their feces. Environmental and personal hygiene measures.	Yes

Fact Sheet compiled by: Kathryn C. Gamble

Sheet completed on: 26 September 2013; updated 16 February 2018

Fact Sheet Reviewed by: Kay Backues; Tony Goldberg

Susceptible animal groups: The parasite most commonly infects suids and great apes, including humans. In great apes, free-ranging populations appeared to present exceptionally low to no prevalence infection, or were unaffected by these limited *B. coli* burdens while greater than 50% of captive groups and those in closer proximity to humans were infected. *B. coli* has been reported in other non-human primates (e.g., macaques and lemurs); in cercopithecine monkeys, the parasite is observed in both captive and free-ranging populations. Additionally, the parasite is reported in artiodactylids (cows, camels), and, rarely, in laboratory rodents (e.g., guinea pig and rat) and dogs. Since the last update, it also has been reported in free-ranging South American sea lions (*Otaria flavescens*) and fin whales (*Balaenoptera physalus*) but its significance is not yet clear in these newly identified host taxa.

Causative organism: *Balantidium coli*; a cosmopolitan, holotrichous ciliated protozoan and largest ciliate to infect humans. Other *Balantidium* species can be observed as commensals in tortoises.

Zoonotic potential: Yes; both trophozoites and cysts can initiate infection. Occupational exposure to suids, poor hygiene, or foreign travel have been associated with human infections, although disease as a result of infect in humans is not considered common.

Distribution: Worldwide but higher prevalence in tropical and subtropical regions.

Incubation period: 4-5 days

Clinical signs: Clinical signs are consistent with gastrointestinal irritation and typically are watery mucoid diarrhea. Following ingestion of the cysts, excystation occurs in the small intestine, then the trophozoites - excysted or ingested - colonize the large intestine and cecum. If invasion occurs through the mucosa, extraintestinal disease has been reported, including to the liver, lung, and bone.

Suids are mostly asymptomatic unless the parasite invades the mucosa, and this only occurs when prior mucosal damage enables its entry. Similarly, cercopithecine monkeys are rarely symptomatic. In great apes,

BALANTIDIUM COLI

chimpanzees are typically asymptomatic or present mild clinical signs of diarrhea, which may be simply unformed feces. Asymptomatic chimpanzees may recrudescence with diarrhea when stressed by other illnesses. *B. coli* may compete with commensal protozoa, such that perturbation of the normal gastrointestinal microflora may facilitate infection or recrudescence. In gorillas, ulcerative colitis; severe diarrhea; and potential abdominal abscessation or colonic fistulation have been noted.

Postmortem, gross, or histologic findings: Ulcerative colitis with large numbers of characteristic *B. coli* organisms invading the colonic mucosa.

Diagnosis: Direct examination of feces by saline smear allows diagnosis. While flotation can be utilized, cysts are the only form detected routinely by this method, and sedimentation may assist in concentration of the organism. Iodine staining may assist in identification of the organism. Detection of trophozoites (30–150 x 25–120 µm) in diarrhea is most common; this stage is a distinctive ovoid with an elongated end, peripheral short cilia, and containing a large cytostome. This stage also has a distinctive spiraling motility with uniform ciliate beating. The infective form (cyst 45–65 µm) is usually found in formed feces, is round, and contains the ciliated organism within a transparent double wall. Both forms have a large, kidney-shaped nucleus. At necropsy, scrapings of the colonic and cecal mucosa can be performed for evaluation and staining with H&E.

Material required for laboratory analysis: Feces.

Relevant diagnostic laboratories: Any diagnostic laboratory with routine parasitologic capabilities should be able to diagnose this infection. The required diagnostic methods, such as fecal flotation, are readily available in-house in any laboratory performing routine fecal exams.

Treatment: Antiprotozoals (e.g., tetracyclines, iodoquinol, metronidazole, and paromomycin) are recommended in clinical cases. Symptomatic treatment of clinical disease will be necessary in severe cases. Starch-rich diets support of the organism's replication and may account for the increased susceptibility of captive as opposed to free-ranging great apes.

Prevention and control: Carrier animals should be managed separately from susceptible animals. For example, co-housing of cercopithecine monkeys with great apes should be considered cautiously. Excellent sanitation should be practiced between carrier animal enclosures and susceptible animals as this parasite has a direct life cycle and requires no intermediate host. That said, this approach should include pest control as some roach species have been demonstrated to carry the protozoan within their gastrointestinal tract. Treatment of carrier animals to reduce environmental contamination should be considered.

Suggested disinfectant for housing facilities: The *B. coli* trophozoite cannot survive for in a dry environment, and generally is expected not to survive beyond 48 hours. Regular removal of feces assists with reduction of environmental contamination. Cysts can survive outside body for two weeks or more at ambient temperatures and are highly resistant to disinfection. Bleach (sodium hypochlorite) at routine disinfection concentrations is not sufficient to destroy the organism.

Notification: None.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Treatment of carrier animals to reduce their burden prior to introductions to naïve animals should be considered. Introduction stressors might produce clinical disease in carrier animals. Testing of relevant species for this organism during quarantine periods is advisable.

Conditions for restoring disease-free status after an outbreak: As a carrier state exists, disease-free state is difficult to attain. Effective management of carriers and treatment of ill individuals is more realistic.

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Experts who may be consulted:

Veterinary Advisors for Great Ape Species Survival Plans

Chimpanzee:

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Avian, mammals, including humans	Ingestion of embryonated eggs or infected carrier hosts	Depression, lethargy, agitation, tremors, head or body tilt, circling, ataxia, lateral recumbency, coma	Asymptomatic to fatal	Early aggressive treatment with albendazole and high dose corticosteroids have shown to be effective, ocular larva migrans can be killed using laser treatment	Personal/ environmental hygiene, wear gloves and additional PPE when working with potentially infected animals/ equipment	Yes

Fact Sheet compiled by: Emily L. Blizzard

Sheet completed on: 27 May 2011; updated 1 October 2012; updated 01 January 2018

Fact Sheet Reviewed by: Laurie A. Baeten, Kerri Pedersen, Michael J. Yabsley

Susceptible animal groups: avian; mammal, including human

Causative organism: Recognized species of *Baylisascaris*

Parasite	Primary Definitive Host(s)
<i>B. ailuri</i>	Red Panda
<i>B. columnaris</i>	Skunks
<i>B. devosi</i>	Martens, fishers, and wolverines
<i>B. laevis</i>	Groundhogs, marmots, ground squirrels, rodents
<i>B. melis</i>	Badgers
<i>B. potosis</i>	Kinkajous, possibly other procyonids (e.g., olingo)
<i>B. procyonis</i>	Raccoons and other procyonids (e.g., kinkajou)
<i>B. schroederi</i>	Giant pandas
<i>B. transfuga</i>	Bears
<i>B. tasmaniensis</i>	Tasmanian devils, quolls, native “cats”
<i>B. venezuelensis</i>	South American spectacled bear

Zoonotic potential: Yes, for *B. procyonis*. Other species unknown.

Distribution: *Baylisascaris procyonis*, the most studied species, is a common ascarid parasite of raccoons (*Procyon lotor*) and has a widespread distribution throughout the United States, Canada, and Costa Rica and has been introduced to Japan, China, and several countries in Europe. In the US, the highest prevalence rates occur in the Midwestern, Northeastern, and Western states. In the Southeastern US, infections are most common in mountainous regions (Tennessee, Kentucky, and North Carolina) although isolated areas of high prevalence have been detected in regions of Texas, Georgia, Florida and North Carolina. In Canada, *B. procyonis* is found in British Columbia, Nova Scotia, Ontario, Prince Edward Island, and Quebec. Accurate distribution maps are unavailable for the majority of *Baylisascaris* species since they are relatively rarely studied. Although humans are considered accidental hosts for *B. procyonis*, over 50 cases of baylisascariasis have been documented. Documented cases resulted in severe permanent neurologic and/or ocular deficits or death. Moreover, recent studies utilizing improved serological detection assays for *B. procyonis* suggest that subclinical infections are common in areas of parasite endemicity. Within the US and Canada, *B. columnaris*, *B. melis*, and *B. transfuga* may pose a zoonotic risk to humans and are probably found throughout the range of their natural hosts.

Incubation period: Once *Baylisascaris spp.* eggs are shed by a definitive host into the environment, eggs develop into an infective-stage larva within 10-14 days depending upon environmental conditions. Following ingestion by a susceptible host, larvae hatch from the egg and can migrate through numerous tissues

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(depending on the *Baylisascaris spp.* involved) including the central nervous system (CNS), as early as 3 days post infection. In susceptible species, clinical disease or death can be observed 9-10 days post-infection. In more resistant host species (or if low numbers of larvae are ingested), CNS symptoms may not appear until 2-4 + weeks post-infection.

Clinical signs: Clinical signs in paratenic hosts, including humans, vary based on number of larvae ingested, the tissues through which larvae migrate, and host. Pathogenicity varies among *Baylisascaris* species. *Baylisascaris procyonis* and *B. melis* are the most pathogenic, followed by *B. columaris*, but little is known about other *Baylisascaris* species. Clinical signs and symptoms associated with baylisascariasis are often nonspecific but may include, although not limited to, depression, lethargy, tremors, partial paralysis, head or body tilts, ataxia, circling, cognitive deficits, easy agitation/ irritability, and death.

Post mortem, gross, or histologic findings: Many infected animals will have no gross lesions. However, inflammation and traumatic damage may be observed through the liver, lungs, and other organs of animals infected with large numbers of larvae. In these hosts, granulomas may be grossly visible in many tissues such as the liver, lungs, heart, diaphragm, pancreas, spleen, kidneys, mesentery, mesenteric lymph nodes, intestinal wall, skeletal muscles, brain, and eyes. Histologically, extensive inflammatory tracts and larvae may be observed.

Diagnosis: Humans: Suspect *Baylisascaris* infections may be diagnosed using serologic methods such as ELISA (recombinant antigen-based) and Western blotting. Ocular examinations may identify the presence of non-species specific larval nematode ocular migrans. Neural larva migrans may be identified using neuroimaging and encephalography although additional testing will be needed to identify the species involved.

Animals: Postmortem necropsies of suspected animals are the most conclusive way to diagnose *Baylisascaris* infections. In suspected intermediate hosts, clinical signs, history of exposure, serology, post mortem necropsies, PCR, and recovery and/or identification of larvae can be used to diagnose *Baylisascaris*. To determine the species of *Baylisascaris* present, PCR and sequence analysis should be performed. Fecal floats or necropsy and examination of small intestine can be used to diagnose infection in definitive hosts.

Material required for laboratory analysis: Adult nematode specimens may be examined microscopically and identified morphologically although adult males are needed to determine species. Genetic identification may be needed for larva migrans found in intermediate hosts and/or immature nematodes in definitive hosts.

Relevant diagnostic laboratories: Veterinary clinics can run routine fecal exams to diagnose infection in definitive hosts. In intermediate hosts, veterinary diagnostic laboratories capable of PCR analysis and/or histology should be able to perform diagnostic testing on suspected animal cases. Human cases should be referred to the Health Department or the CDC for testing.

Treatment: Aggressive treatment with albendazole (25-50 mg/kg per day orally for 10-20 days) combined with high doses of corticosteroids is recommended in humans. Treatment appears to be successful when administered quickly following exposure. If albendazole is not available, mebendazole or ivermectin may be used. Ocular larva migrans can be killed using lasers followed by a regime of anti-inflammatory drugs and steroids to aid in the possible recovery of any remaining visual acuity. Intestinal infections in various definitive hosts such as raccoons, other procyonids, skunks, domestic dogs, and bears can be successfully treated with common antihelmintics such as pyrantel pamoate (20 mg/kg), ivermectin (1 mg/kg), moxidectin (1 mg/kg), albendazole (50 mg/kg x 3 days), fenbendazole (50 mg/kg x 3 days), and flubendazole (22mg/kg x 3 days). Animals should be monitored regularly after treatment to ensure complete clearance of worms.

Prevention and control: Continued education of the public, human health, wildlife, and veterinary professionals should be made a priority. Recent research using anthelmintic baits combined with the removal of latrine sites has shown to decrease prevalence rates among intermediate hosts. Further research is needed to determine the exact distribution, potential for spread, transmission dynamics, and impacts on wildlife.

Suggested disinfectant for housing facilities: Areas should be cleaned immediately to avoid accidental ingestion of eggs by children or pets. Personnel should wear appropriate personal protective equipment and avoid contaminating hands and clothes with potentially contaminated materials. Eggs are not immediately

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infectious and must develop in the environment for a period of time (10-14 days up to several months, depending on environmental conditions) before becoming infective. Frequent sanitation will limit the buildup of eggs on these surfaces. However, eggs will continue to accumulate in the surrounding environment and once the eggs embryonate, they can remain viable for several years. Currently few methods are available for decontaminating areas infested with *B. procyonis* eggs. Highly concentrated caustic chemicals such as a 50/50 mixture of xylene and absolute alcohol, boiling lye, or boiling Lysol may be used to decontaminate potentially infected areas. The most effective way of decontaminating an area is flaming. Although burning is the most effective way to kill eggs, it is not feasible for flammable areas such as roofs, decks, etc. In the laboratory, exposing infectious eggs to water heated to 62°C for 1 minute has been shown to inactivate larvae.

Notification: *Baylisascariasis* in humans is reportable in some states; check your local requirements. Infection in animals is not reportable, except in Washington State where infections in animals, other than raccoons, is reportable.

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Animals displaying neurologic symptoms are not infective to other intermediate hosts. However, impaired intermediate hosts are likely to become prey for various carnivore or omnivore species. If ingested by an appropriate definitive host, the parasite cycle within a system could be perpetuated.

Definitive hosts known to harbor infections should be quarantined, placed on an anthelmintic regime, and monitored regularly for infection. Before placing susceptible animals in cages that had contact with infected animals, the cages should be decontaminated.

Conditions for restoring disease-free status after an outbreak: None

Experts who may be consulted:

Centers for Disease Control and Prevention – Division of Parasitic Diseases

Local Health Departments

USDA APHIS Wildlife Services - State Agencies

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BERTIELLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Nonhuman primates, Dermopterans, Rodents, Australian marsupials, Humans	Via accidental ingestion of intermediate host (oribatid mites) containing cysticercoids.	Typically none. In humans, it has caused intermittent diarrhea, abdominal pain, anorexia, and weight loss.	Low. Natural infection does not pose a serious health hazard for wild animals.	Praziquantel	Screen and treat captive animals, avoid ingestion of soil in endemic areas.	Yes
Fact Sheet compiled by: Sara Childs-Sanford						
Sheet completed on: 15 November 2012						
Fact Sheet Reviewed by: Lily Parkinson						
Susceptible animal groups: Old World primates (including <i>Papio ursinus</i> , <i>Papio cynocephalus</i> , <i>Cercopithecus mona mona</i> , <i>Cercopithecus ascanius</i> , <i>Cercopithecus pygerythrus</i> , <i>Cercopithecus schmidti</i> , <i>Ptilocolobus tephrosceles</i> , <i>Colobus guereza</i> , <i>Colobus angolensis</i> , <i>Pan troglodytes verus</i> , <i>Pan troglodytes schweinfurthii</i> , <i>Macaca fascicularis</i> , <i>Pongo pygmaeus</i> , <i>Pongo abelii</i>), New World primates (including <i>Callicebus personatus nigrifrons</i> , <i>Callicebus oenanthe</i> , <i>Cebus paella fatuellus</i> , <i>Cebus capucinus</i> , <i>Callithrix saguinus</i> , <i>Alouatta caraya</i> , <i>Alouatta guariba clamitans</i>), Dermopterans (including <i>Cynocephalus volans</i> , <i>Cynocephalus variegates</i>), rodents (including <i>Rattus</i> spp., <i>Uromys</i> spp.), Australian marsupials (including <i>Trichosurus vulpecula</i>), humans						
Causative organism: <i>Bertiella</i> spp. (cestode, family Anoplocephalidae). In nonhuman primates: Old World primates (<i>Bertiella studeri</i> , <i>B. satyri</i>) New World primates (<i>B. mucronata</i>) Lemurs (<i>B. lemuriformis</i>) Dermopterans (<i>B. elongata</i> , <i>B. plastica</i> , <i>B. rauschi</i> , <i>B. musasabi</i>) Rodents (<i>B. anapolitica</i>) Australian marsupials (<i>B. trichosuri</i>) Humans (<i>B. studeri</i> , <i>B. mucronata</i>)						
Zoonotic potential: Yes. Human infection (via accidental ingestion of the intermediate host), can occur in those having close contact with the environment of definitive hosts (especially primates) and is most common in children.						
Distribution: Africa, Asia, South America, Australia/New Zealand						
Incubation period: Unknown.						
Clinical signs: Typically none. Humans have been reported to experience intermittent diarrhea, abdominal pain, anorexia, and weight loss. Increased leaf swallowing behavior has been reported in wild chimpanzees.						
Post mortem, gross, or histologic findings: The adult tapeworm may be found in the small intestine.						
Diagnosis: Identification of eggs or gravid proglottids in feces, identification of adult worm at necropsy. Free eggs from gravid segments or feces are 40-46µm long and 36-40µm wide in <i>B. mucronata</i> and 48-60µm long and 40-60µm wide in <i>B. studeri</i> . Should be considered as a diagnosis when fecals continue to be tapeworm positive after treatment with fenbendazole or albendazole.						
Material required for laboratory analysis: Feces						
Relevant diagnostic laboratories: Any veterinary diagnostic laboratory with a parasitologist on staff.						
Treatment: Praziquantel						

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Prevention and control:

Wild: Avoid contact with soil in proximity of definitive hosts. Proper hygiene practices and food safety in endemic areas.

Captivity: quarantine, screen all animals for infection and treat accordingly.

Suggested disinfectant for housing facilities: Commonly used disinfectants are generally not effective in killing tapeworm eggs or larvae. Recent research has demonstrated that sodium hypochlorite is effective in killing *Taenia* spp. eggs, but its effectiveness against *Bertiella* spp. eggs is currently unknown.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Eliminate infection in positive animals prior to introduction to other animals.

Conditions for restoring disease-free status after an outbreak: Repeated negative fecals.

Experts who may be consulted: While no specific researchers are currently reporting expertise in this parasite, parasitology staffs at veterinary colleges would be a good option; it would be prudent to consider Australian veterinary colleges due to parasite location.

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BLASTOMYCOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Canids, primates, marine mammals, felids, horses, bats, ursids, mustelids	Inhalation of airborne conidia from disturbed endemic soil.	Fever, anorexia, coughing, dyspnea, ocular and nasal discharge, loss of body condition, draining, cutaneous lesions, lameness, ocular disease characterized by anterior uveitis or retinal disease.	Disease severity may range from asymptomatic to fatal fulminant respiratory failure. Clinical signs may persist for weeks to months before disease progresses in severity.	Itraconazole is currently treatment of choice in dogs. Amphotericin B is rarely used due to nephrotoxicity. Fluconazole is a lower cost alternative to itraconazole. Treatment duration in dogs usually 3-6 months.	There are no standards for prevention or control due to origin of the organism in the soil.	Normally not zoonotic. Human infection is a result of exposure to a shared environmental source.

Fact Sheet compiled by: Tiffany Wolf

Sheet completed on: August 4, 2010; updated December 23, 2012; updated January 18, 2018

Fact Sheet Reviewed by: Arno Wünschmann, Joni Scheftel, Robert W. Bradsher, Gene M. Scalarone, Alfred M. Legendre, Janelle Renschler

Susceptible animal groups: Blastomycosis has been reported in canids, primates, felids, equids, marine mammals, ursids, mustelids, and bats.

Causative organism: *Blastomyces dermatitidis*

Zoonotic potential: Human infection is generally a result of exposure to a shared environmental source, rather than transmission from another mammalian host. Although very rare, there are reports of zoonotic transmission associated with dog bites, cat scratches, animal necropsies, and a kinkajou bite. Care should be taken to avoid accidental inoculation with contaminated objects such as needles, knives, etc. One report of a localized *Blastomyces* infection in a veterinarian from an inadvertent needlestick following aspiration of a draining lesion.

Distribution: Endemic in Mississippi-Ohio river basin and central Atlantic states of the U.S. and northern Ontario and Manitoba, Canada. It is believed to be a soil saprophyte, associated with acidic, sandy soil, often in close proximity to a water source.

Incubation period: Usually 2-6 weeks, but clinical signs may appear as long as several months to years after infection.

Clinical signs: Blastomycosis is typically a primary pulmonary disease as infection often occurs via the inhalation of aerosolized fungal spores from a soil source. Primary cutaneous disease does occur, although rarely. Infections are often disseminated, and clinical signs are associated with distribution of lesions. Fever, anorexia, coughing, dyspnea, loss of body condition and draining, cutaneous lesions are common clinical signs. Bone or joint involvement can result in lameness. Ocular involvement is also relatively common dogs and may be exhibited by anterior uveitis and subretinal effusion which can lead to retinal detachment and blindness.

Post mortem, gross, or histologic findings: With pulmonary involvement, lesions are often distributed throughout the lungs and consist of multifocal to coalescing white-grey granulomas, sometimes with central abscessation. Regional lymph nodes are typically involved and characterized by granulomas, abscesses, or caseous lesions. Similar granulomatous lesions will be seen with disseminated disease in any involved tissues.

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The distribution of disease throughout the body often includes, but is not limited to, the lungs, skin, eyes, bones, joints, lymph nodes, and central nervous system.

Microscopically, organisms appear as spherical thick-walled yeasts of 8-20µm in diameter with broad-based budding. Organisms can usually be identified cytologically from lymph node aspirates or impression smears from draining skin lesions. Pyogranulomatous inflammation is frequently observed on cytology or histopathology specimens. Organisms may be infrequent in more chronic infections. Histologically, hematoxylin and eosin (H&E) stains may result in poor visualization of fungal elements; therefore, special stains such as Periodic Acid-Schiff (PAS) stain or Gomori's methenamine silver (GMS) stain should be used in addition to H&E.

Diagnosis:

Tracheal wash, bronchoalveolar lavage, impression smears or aspirates of enlarged lymph nodes, skin lesions, or draining exudates are appropriate for cytological examination. Biopsy of granulomatous lesions can be submitted for histopathology. Urine and/or serum can be submitted for antigen concentrations. Serum can be sampled for antibody testing with AGID but this is less sensitive than the urine antigen early in the course of the disease.

Material required for laboratory analysis: Many commercial and state veterinary laboratories can provide cytologic, histopathologic, and serologic diagnostic services. Serum samples for itraconazole concentrations can be sent to The Fungus Testing Lab (University of Texas Health Science Center, San Antonio, Texas) or MiraVista Diagnostics (Indianapolis, Indiana). Mira Vista Diagnostics can also perform the antigen test for Blastomycosis.

Relevant diagnostic laboratories: Many commercial and state veterinary laboratories can provide cytologic, histopathologic, and serologic diagnostic services. Serum samples for itraconazole concentrations can be sent to The Fungus Testing Lab (University of Texas Health Science Center, San Antonio, Texas) or MiraVista Diagnostics (Indianapolis, Indiana). Mira Vista Diagnostics can also perform the antigen test for Blastomycosis.

Treatment: Itraconazole is currently the treatment of choice for blastomycosis, given its ease of administration (per os) and lower toxicity. Itraconazole blood levels should be measured 14-21 days after beginning therapy, and dosage should be increased if below 3.0 µg/mL. Given the high cost of itraconazole, fluconazole is a lower-cost alternative but generally requires a longer treatment duration. Duration is typically 4 months with itraconazole and 6 months with fluconazole. Antigen level may be used to monitor therapy. Treatment should be continued until antigen levels are below 1 µg/mL. Amphotericin B is nephrotoxic and requires intravenous administration but may be an excellent option for animals presenting with serious disease, or not responding to itraconazole therapy. Absorption of compounded itraconazole is inconsistent and may account for treatment failures. Treatment relapse is not uncommon within 1 year of treatment.

Prevention and control: *Blastomyces dermatitides* originates from the soil, and will grow in shaded, sandy, acidic soil with close proximity to water. Although sterilization of soil is not realistic, restriction of access by animals to areas where other cases are thought to have originated may reduce risk. Alteration of the environment to eliminate the growth conditions of the organism may be beneficial.

Suggested disinfectant for housing facilities: Replacing soil or gravel based outdoor housing facilities with concrete floors will reduce the presence of the organism in the housing area. Disinfectants with antifungal spectrum of action may be used on impervious environmental surfaces according to the manufacturer's directions, however, there is no proven method of disinfecting the environment to eliminate *Blastomyces* organisms.

Notification: Human and animal cases may be reportable in certain states. Ask local public health and animal health officials for direction in your area. A national surveillance program does not currently exist in the

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United States.

Measures required under the Animal Disease Surveillance Plan: None currently.

Measures required for introducing animals to infected animal: Blastomycosis is generally not considered a contagious disease that is directly transmitted.

Conditions for restoring disease-free status after an outbreak: Disease-free status can only be achieved after a minimum of 60-90 days to 180 days of therapy accompanied by complete resolution of clinical signs and lesions. Given the relatively common occurrence of relapse, patients should be monitored for return of clinical signs or lesions in the following 12-15 months. Increasing urine or serum antigen levels may indicate a possible relapse.

Experts who may be consulted:

Gene M. Scalarone, Ph.D., Department of Biological Sciences, Idaho State University

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BLUETONGUE VIRUS (BTV)

EPIZOOTIC HEMORRHAGIC DISEASE VIRUS (EHDV)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p><u>BTV</u>: All ruminants are susceptible; camelids; other mammals positive on serological tests without disease; recent evidence of possible BTV disease in carnivores</p> <p><u>EHDV</u>: primarily white-tailed deer but also elk, pronghorn, mule deer, cattle; rarely camelids but likely all ruminants can be infected</p>	<p>Insect vector primarily (biting midges of genus <i>Culicoides</i>; <i>C. sonorensis</i> principally in US); iatrogenic; <i>in utero</i>; possibly oral in carnivores</p>	<p>Pyrexia, oral and nasal ecchymoses and ulcerations, facial edema, conjunctivitis, rhinorrhea, ptyalism.</p> <p><u>BTV</u>: Hoof slough and wool loss in sheep</p> <p><u>EHDV</u>: Hoof slough in deer</p>	<p><u>BTV</u>: variable, dependant on species, isolate, geographic location; sheep, white-tailed deer and pronghorn most likely to be severely affected.</p> <p><u>EHDV</u>: variable; white-tailed deer and pronghorn most likely affected</p> <p>In cattle EHDV/ BTV un-common, generally mild; however, more severe disease associated with specific subtypes or outbreaks reported</p>	<p>Symptomatic</p>	<p>Insect control which is realistically difficult; potential to vaccinate for some strains of BTV; no vaccines available for EHDV</p>	<p>No</p>

Fact Sheet compiled by: Allison Wack

Sheet completed on: 25 January 2011; updated 19 March 2013

Fact Sheet Reviewed by: David Stallknecht, Holly Haefele

BLUETONGUE VIRUS (BTV)
EPIZOOTIC HEMORRHAGIC DISEASE VIRUS (EHDV)

Susceptible animal groups:

BTV: Ruminants: sheep, goats, cattle, bison, deer, antelope, bighorn sheep, North American elk, camelids, greater kudu, muntjac, topi; perinatal infection in Grant's gazelle, gemsbok, sable, buffalo, ibex, hartebeest, addax. Many other ungulates serologically positive without evidence of disease. Clinical signs common in sheep, occasional in goats, and rare in cattle. White-tailed deer, pronghorn and desert bighorn sheep may have severe disease. Abortions caused by BTV contaminated vaccine in dogs. Seropositivity in a variety of large African carnivores. Report of infection and death in 2 Eurasian lynx fed ruminant fetuses and stillborns.

EHDV: Ruminants: white-tailed deer most severely affected, less frequently in mule deer and pronghorn; Black-tailed deer, red deer, wapiti, roe deer, fallow deer, bison, black and white rhinoceros, black bear have been found seropositive. Rare outbreaks in cattle; sheep experimentally infected but rarely develop clinical signs.

Causative organisms: Family Reoviridae, Genus *Orbivirus*

BTV: 26 serotypes worldwide, 15 identified in US (2, 10, 11, 13, 17 considered endemic; 1,3, 5, 6, 9,12, 14, 19, 22, 24 sporadically) in domestic or wild ruminants

EHDV: 7 serotypes, 3 endemic to US (1, 2, and 6), EHDV-6 was first identified in 2006

Zoonotic potential: None; one anecdotal unconfirmed report of BTV infection in a laboratory worker

Distribution:

BTV: World-wide where vectors are present (generally between latitudes of 40°N and 35°S, although may be moving north). Mostly southern and western, also southeastern, US.

EHDV: Disease in North America, Australia, Asia, Africa; seropositive animals in South America

Incubation period:

BTV: 5-10 days in domestic sheep; typically infectious to insect vector for several weeks

EHDV: 5-10 day incubation in deer. May remain viremic for up to 2 months

Clinical signs:

BTV: Variable and species dependent.

Sheep: pyrexia, ptialism, depression, dyspnea, panting, hyperemia and edema of muzzle, lips, tongue, ears, ulcerations and erosions in mouth, sloughing of hooves, abortion, loss of wool 3-4 weeks post infection. Recrudescence possible, severity partially dependant on serotype.

Cattle: pyrexia, rarely hyperemia, vesicles or ulcers in mouth, hyperemia of coronary band, dermatitis, hydranencephaly or cerebral cysts in calves.

Pronghorn and whitetail deer: hemorrhage and sudden death.

EHDV: Three distinct syndromes in deer:

Peracute: fever, anorexia, weakness, swelling of head and neck, respiratory distress; death within 8-36 hours

Acute/classical: multi-organ hemorrhage, ptialism, rhinorrhea, oral and GI ulcerations; mortality may be high

Chronic: ill for several weeks with gradual recovery; may have hoof damage/slough or enough scarring from rumen ulcerations to cause emaciation

Typically subclinical in cattle, but clinical signs include fever, oral ulcers, salivation, lameness associated with coronitis, and weight loss. Fetal resorption and hydranencephaly possible; death uncommon in North America, although lameness and unthriftiness may be prolonged.

Post mortem, gross, or histologic findings:

BTV and EHDV: Clinical signs similar in affected animals, but both highly variable. Sheep: edema of face and ears, crusty exudates on nostrils, hyperemia of coronary bands, ulcers and erosions of oral cavity +/- necrosis and cyanosis; hyperemia, hemorrhage and edema throughout internal organs possible. Hemorrhage at base of pulmonary artery

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BLUETONGUE VIRUS (BTV)
EPIZOOTIC HEMORRHAGIC DISEASE VIRUS (EHDV)

<p>EHDV: In deer, gross findings differ with form of disease, consistent with clinical presentation. Histologic findings include widespread vasculitis with thrombosis, endothelial swelling, hemorrhages, degenerative changes, and necrosis in many organs.</p>
<p>Diagnosis: Serologic tests are only diagnostic with paired serum. BTV: AGID, ELISA, CF, PCR, VI, VN EHDV: AGID, PCR, VI, VN</p>
<p>Material required for laboratory analysis: Serum for AGID, ELISA, CF, VN Whole blood or spleen for PCR Whole blood, spleen, or lung for VI ELISA (if pre and post serum available), PCR, VI may be most useful clinically; positive serology does not correlate well with viremia.</p>
<p>Relevant diagnostic laboratories: NVSL http://www.aphis.usda.gov/animal_health/lab_info_services/downloads/AmesDiagnosticTestingCatalog.pdf TVMDL http://tvmdl.tamu.edu/schedule2.php</p>
<p>Treatment: Symptomatic; analgesics and anti-inflammatories may help address clinical signs</p>
<p>Prevention and control: BTV: Limiting vector exposure, number and habitat. Pyrethroids or organophosphates effective against <i>Culicoides</i>. Vaccination for sheep available in some areas, typically serotype specific MLV (Serotype 10 available throughout US; combo of serotypes 10, 11, 17 in CA; 17 available in Texas). Vaccination recommended in spring prior to vector season in endemic areas; contraindicated in pregnant ewes and during outbreaks. Quarantine of imported animals, serologic screening, and vector control during transport are important for preventing introduction into bluetongue-free areas. EHDV: Limiting vector exposure, as above. No vaccines available.</p>
<p>Suggested disinfectant for housing facilities: Primarily vector borne, unlikely to contaminate environment. However, sodium hypochlorite or 3% sodium hydroxide are effective if disinfection is warranted.</p>
<p>Notification: Required in certain states; check with AVIC or state veterinarian</p>
<p>Measures required under the Animal Disease Surveillance Plan: None</p>
<p>Measures required for introducing animals to infected animal: Seronegative animals (two negative test results >28 days apart with no vector exposure between), vaccinated animals or naturally immune animals (positive serologic test for all applicable serotypes >60 days prior) pose minimal risk for introduction. Introduction of an actively infected animal to a naïve population/area should be avoided. A viremic animal should become negative on PCR or VI prior to being introduced, which should take no longer than 60 days. During that time, the viremic animal should be kept without vector access and treated with insecticides (both animal and environment).</p>
<p>Conditions for restoring disease-free status after an outbreak: Seasonal in endemic areas, unlikely to be eradicated once established in vector population. OIE has firm guidelines for being classified as a BTV free country. Infection by one serotype of either virus usually offers lasting immunity for that serotype, though may not be protective against others.</p>
<p>Experts who may be consulted: NVSL, OIE, state veterinarian</p>
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BLUETONGUE VIRUS (BTV)
EPIZOOTIC HEMORRHAGIC DISEASE VIRUS (EHDV)

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BORDETELLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Dogs	Aerosol; oronasal	Paroxysmal coughing	Mild to severe	Antibiotics, supportive care	Quarantine affected animals	Only in immune-compromised people
Cats	Aerosol; oronasal	Sneezing, pyrexia, nasal discharge, occasional cough				
Birds (turkeys)	Contaminated water; direct contact	Sinusitis with clear nasal discharge, foamy eyes, characteristic snick or cough				
Swine (domestic)	Direct contact, aerosol	Nonprogressive atrophic rhinitis (as sole pathogen), pneumonia				
Rodents	Direct contact, Aerosol	Nasal discharge, sneezing, snuffling, rales, dyspnea				
Rabbits	Direct contact, aerosol	Snuffling, pneumonia				
Horses	Direct contact, aerosol	Nasal discharge, pneumonia				
Seals	Direct contact?	Tracheitis, pneumonia				
Humans (rarely non-human primates)	Direct contact, aerosol	Paroxysmal cough, runny nose, sneezing, pyrexia				

Fact Sheet compiled by: Kortney A. O'Neill; updated by David A. Bemis

BORDETELLOSIS

Sheet completed on: 30 April 2011; updated 5 August 2013
Fact Sheet Reviewed by: Claude Lacasse; Karen Register
Susceptible animal groups: Reported in canids, felids, ursids, suids, lagomorphs, rodentia, aves, primates (human and non-human), insectivores, mustelids, ovids, pinnipeds, equids, and koalas
Causative organism: <i>Bordetella bronchiseptica</i> (most animal cases, rare human cases), <i>Bordetella pertussis</i> (humans, non-human primates), <i>Bordetella parapertussis</i> (humans, ovids); <i>Bordetella avium</i> (birds) and <i>B. hinzii</i> (birds, rodents, rabbits and rare human cases)
Zoonotic potential: <i>B. bronchiseptica</i> , <i>B. hinzii</i> -- usually reported in immuno-compromised people
Distribution: Worldwide
Incubation period: 3-14 days
Clinical signs: Disease may be present in asymptomatic carriers. Paroxysmal cough is most notable sign in dogs and humans and sneezing, oculonasal discharge, rhinitis, pyrexia, or pneumonia may be developed. Sudden death may occur.
Post mortem, gross, or histologic findings: Bronchopneumoina, suppurative bronchitis, tracheitis, mucopurulent rhinitis. The disease rarely causes mortality in animals unless concurrent infection with virus or other bacterial component.
Diagnosis: Bacterial culture, PCR
Material required for laboratory analysis: Oropharyngeal or nasopharyngeal culture swab
Relevant diagnostic laboratories: Any diagnostic lab with capability to perform bacterial culture
Treatment: If the sole infectious agent, the disease may be self-limiting. However, antibiotics decrease course of shedding. Supportive care (antitussives, humidification, expectorants, etc.) can be applied.
Prevention and control: Isolation of any suspected upper respiratory infection animals during active disease. Adequate ventilation and air exchanges (12-20/hr) within holding areas. Vaccination of susceptible species can be utilized.
Suggested disinfectant for housing facilities: Thorough cleaning and disinfection -- most cleansers are effective against <i>Bordetella</i> spp., sodium hypochlorite, chlorhexidine or benzalkonium solution
Notification: Pertussis is reportable in some states
Measures required under the Animal Disease Surveillance Plan: None currently
Measures required for introducing animals to infected animal: Infected animals should be quarantined for 2-6 weeks until clinical signs resolve
Conditions for restoring disease-free status after an outbreak: Clean and decontaminate environment
<p>Experts who may be consulted David A. Bemis, PhD, DACVM (Honorary) Department of Comparative Medicine University of Tennessee Knoxville, TN (865) 974-5576 bemis@utk.edu</p>
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BORDETELLOSIS

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American Association of Zoo Veterinarians Infectious Disease Manual
LYME DISEASE (caused by BORELLIA BURGDORGERI)

Animal group(s) affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals (especially dogs, occasionally horses), some birds (birds are usually asymptomatic and/or are reservoirs)	Tick vector (<i>Ixodes</i> sp); White tailed deer and rodent reservoir hosts, including white-footed mouse (<i>Peromyscus leucopus</i>) in N. America & <i>Apodemus</i> sp in Eurasia)	General: Shifting leg lameness, arthritis, fever, myocarditis CNS signs; Humans: erythema migrans (“bull’s eye” rash); Dogs: renal syndrome, Horses: uveitis, neuroborreliosis	Varies, can be mild lameness or chronic illness	Doxycycline x 30d, azithromycin, minocycline, ceftriaxone, amoxicillin; recrudescence is possible	Prevent tick attachment, Remove ticks as soon as possible; vaccine available for dogs	Yes, tick bite only

Fact Sheet compiled by: Elizabeth E. Hammond

Sheet completed on: 6 Sept 18

Fact Sheet Reviewed by: Anne Burgdorf, Tara Harrison, Kristen J Tobin

Susceptible animal groups: all mammals (dogs, horses but cattle appear less susceptible), some birds (birds are usually asymptomatic, and may be a reservoir host)

Causative organism: *Borrelia burgdorferi* sensu lato (s.l.) & sensu stricto (s.s.) (gram-negative spirochete); *B. burgdorferi* s.s. is the cause of Lyme disease in the US, but *B. mayonii* has recently been identified as the cause of Lyme disease in the Midwest US; different strains may explain varied clinical signs depending on region; tick vector: *Ixodes* sp. (*I. scapularis*, *I. ricinus*, *I. pacificus*) (nymph life stage responsible for transmission)

Zoonotic potential: Yes, tick bite only; tick must attached for at least 24hr

Distribution: temperate areas worldwide

Incubation period: 60-90d

Clinical symptoms: shifting leg lameness, joint swelling, arthritis (knee/elbow most common), lymphadenopathy, anorexia, fever, myocarditis, CNS signs, renal syndrome (dogs) uveitis and neuroborreliosis (horses); in humans there is often a rash (erythema migrans, aka “bull’s eye” rash) at the site of tick attachment

Post mortem, gross, or histologic findings: perivascular lymphoplasmacytic infiltrates in kidneys that can lead to glomerulonephritis (dogs), liver, cerebrum, meninges, and lungs; synovitis with inflammatory cells and fibrin deposits

Diagnosis: serology (ELISA, IFA, EIA, modified Western blot), Western immunoblot can differentiate between vaccine titer and natural infection based on band pattern (dogs); PCR; or culture of organism from urinary bladder (difficult), kidney, spleen, skin, and other organs with evidence of clinical signs of disease, history of exposure and response to treatment; also, xenodiagnosis (feeding uninfected tick larvae on a host and evaluating for signs of infection)

Material required for laboratory analysis: serum, whole blood, tissue

LYME DISEASE (caused by **BORELLIA BURGDOGERI**)

Relevant diagnostic laboratories: Standard diagnostic laboratories can test for serologic evidence of Lyme disease; patient-side ELISA SNAP® test (4Dx Plus, IDEXX, Westbrook, ME 04092, USA) available for dogs; culture of organism requires special growth media

Treatment: Doxycycline x 30d (contraindicated in young animals) or minocycline x28d (minocycline has better nervous system penetration-common in horses), azithromycin, ceftriaxone (especially for neurologic disease), amoxicillin; recrudescence is possible; better chance of resolution if treatment initiated early

Prevention and control: Tick prevention, remove ticks within 48hr (minimum time needed to transmit the organism); Lyme disease killed and recombinant subunit vaccines are available for dogs; human vaccine is no longer available.

Suggested disinfectant for housing facilities: standard disinfectants, such as 1% sodium hypochlorite, 70% ethanol, heat, and UV light, are appropriate (*Borrelia* sp cannot survive free-living in the environment)

Notification: reportable in humans (US), but not in animals

Measures required under the Animal Disease Surveillance Plan: none

Measures required for introducing animals to infected animal: tick control

Conditions for restoring disease-free status after an outbreak: none

Experts who may be consulted: Adam Birkenheuer, DVM, PhD, DACVIM, NCSU; ajbirken@ncsu.edu

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BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Naturally affected: Cattle - <i>Bos taurus</i> and <i>B. indicus</i> ; captive exotic ungulates of Bovidae; felines both domestic and captive exotic; domestic goats.	Ingestion of BSE contaminated feed (i.e., meat and bone meal) or infected carcasses.	Apprehension, nervousness and/or aggression; tremoring, incoordination, especially hindlimb ataxia and difficulty in rising; hyperesthesia.	Average incubation period is 2-8 years. The clinical duration is usually several weeks to 6 months. The disease is invariably progressive and fatal.	None.	Prohibit the feeding of most ruminant or mammalian proteins to ruminants.	Yes.

Fact Sheet compiled by: Linda A. Detwiler

Sheet completed on: 27 September 2013

Fact Sheet Reviewed by: Noelia Silva-del-Rio; Meredith M. Clancy

Susceptible animal groups: Ruminants such as cattle (*Bos taurus* and *B. indicus*), sheep and goats, captive exotic ungulates (eland, gemsbok, Arabian and scimitar-horned oryx, nyala and greater kudu) and American bison (*Bison bison*). Felines both domestic cats and captive exotic cats (cheetah, lion, Asian leopard cat, ocelot, puma and tiger) have been reported as “Feline Spongiform Encephalopathy (FSE)”. Experimentally, nonhuman primates also have been infected via the oral and intracranial routes.

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

Causative organism: The etiological agent has not been fully characterized. Understanding of the causative agent remains imperfect, but a wealth of accumulating evidence has led to the conclusions that: (i) a misfolded form of the protein (PrP^{TSE}), known as a prion, acts as a template to induce normal protein molecules to cascade into the same misfolded configuration; (ii) infectivity is associated with an aggregate (or polymer) of 14-28 misfolded protein molecules; (iii) an as yet unidentified host molecule (?chaperone, ganglioside, non-coding RNA) is probably necessary as a cofactor in replication; (iv) the degree of similarity in the primary structure of the protein in different species influences the ease with which the protein can induce inter-species disease; and (v) in some species, the entire process appears to occur spontaneously in the sporadic form of disease, but can be initiated (i.e., 'transmitted') by the introduction of tissue from a diseased to a healthy host, as would have happened when humans consumed BSE-contaminated meat products. Until 2004, it appeared that a single "strain" caused all cases of BSE. It is now known that there are at least two additional strains called L-Type and H-Type atypical BSE.

Little is known about atypical BSE. The origin and natural routes of transmission, if any, have yet to be determined. Almost all cases have been in older cattle (usually > 8 years of age) that have shown little resemblance to the clinic-pathological picture seen in classical disease. It has been suggested that the disease may be sporadic or be caused by a genetic mutation, but no convincing evidence has been found to support either of these ideas. The correct answer will probably only come by study of the future annual incidence curves of both types of disease. Regardless of the origin of atypical BSE, the possibility of recycling the disease in cattle and other ruminants, as well as the potential for transmission to humans, mandate a

continuation of feed and specified-risk materials (SRM) bans, together with diagnostic testing programs for some time to come.

Zoonotic potential: BSE is the cause of the fatal human disease, variant Creutzfeldt-Jakob Disease (vCJD). Epidemiological evidence indicates that transmission is through the consumption of meat products contaminated with BSE agent, which is found primarily in CNS tissue and distal ileum. During the incubation period, it appears that humans may transmit vCJD to other humans via blood transfusions.

Distribution: The first cases of BSE were recognized in the United Kingdom in 1986 and because of recycling of offals into animal feed, the disease rapidly became epidemic in the UK and spread to most other European countries via the trade of contaminated meat and bone meal and infected animals that entered slaughter channels. Worldwide, the number of cases at the end of 2012 was approximately 190,000, all but 6,000 of which were within the UK. In addition to the officially reported and confirmed cases, it is estimated that as many as 3.5 million animals were infected and may have entered the food and feed chains in the UK without being detected. BSE has also been detected in Brazil (atypical), Canada, Falkland Islands (import), Israel, Japan, Oman (import) and the US (import and atypical). Implementation of feed controls has all but eliminated classical BSE, as there were only 21 total cases reported worldwide in 2012. This number includes both classical and atypical BSE. The UK found only 3 cases in 2012. Statistics regarding the occurrence of BSE may be found at <http://www.oie.int/en/animal-health-in-the-world/bse-specific-data/>. It should also be noted that the absence of reported cases over an extended time in a country might not indicate so much the absence of disease as a lack of adequate surveillance.

Naturally occurring cases of BSE in species other than cattle have been very limited and have been linked to exposure to contaminated feed or infected carcasses. The majority of cases originated in the UK and like BSE in cattle, have declined with the implementation of feed controls. None of the exotic animals were infected in the wild.

Incubation period: The incubation period for BSE is measured in years and in cattle can range from 2-8 years.

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

Clinical signs: Affected animals develop a progressive degeneration of the nervous system. They may display changes in temperament, abnormalities of posture and movement, and changes in sensation, including signs of apprehension, nervousness or aggression, incoordination, especially hind-limb ataxia, tremor and difficulty in rising, and hyperesthesia to sound and touch. In addition, many animals have decreased milk production and loss of body condition despite continued appetite. Only a small proportion of affected cattle exhibit what would be considered typical "mad cow" signs. BSE can be mistaken for other conditions or go unnoticed due to subtlety of the signs. The TSE cases in exotic ruminants had a younger onset age and a shorter clinical duration compared to that in cattle with BSE. Clinical signs in the exotic ungulates are similar to those seen in cattle and include ataxia and wasting.

FSE is characterized by progressive nervous signs, including ataxia, hyper-reactivity and behavioral changes and is fatal.

Clinical pathological, gross, and histopathological findings: No gross pathological lesions are found in animals affected with BSE and histological changes appear to be confined to the CNS. The primary lesions found are non-inflammatory vacuolation of neuronal perikarya and grey-matter^[SEP] neuropil and are usually bilaterally^[SEP] symmetric. Astrocytosis may also be observed. Infected animals may not manifest these lesions until end stage disease. Histological changes that are seen in cattle are similar to those seen in the other affected animal species.

Diagnosis: No live animal test for BSE is available. Historically, the diagnosis of BSE relied on the occurrence of clinical signs of the disease confirmed by postmortem histopathological examination of brain tissue. The current diagnostic tests target the detection of PrP^{TSE} (the misfolded form of the prion protein)

deposits in the CNS. Immunohistochemistry and/or Western blots are usually used as confirmatory tests. In addition, a number of rapid immunoassays have been developed and approved by governments for use as screening tests. These include enzyme-linked immunosorbent assays (ELISAs), automated immunoblotting (Western blotting) and lateral flow devices (LFD).

Material required for laboratory analysis: Clinically suspect cases should be subjected to a standard neuropathological approach in which the whole brain is sampled, and a range of representative areas examined. BSE sampling is dependent upon the test methods approved and used by the national veterinary services. For example, in the US brain stem, including the obex, should be submitted as fresh tissue. Countries using immunohistochemistry as the primary diagnostic test may require samples submitted in formalin.

Relevant diagnostic laboratories:

USDA-APHIS

National Veterinary Services Laboratory

1920 Dayton Ave. (for packages)

P.O. Box 844 (for letters)

Ames, IA 50010

(515) 337-7266

Fax: (515) 337-7397

Treatment: None

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

Prevention and control: Given that the primary, if not sole, route of BSE transmission is through the feeding of contaminated meat-and bone-meat (MBM) to cattle, countries with any risk factors need to implement feed controls. The level of restriction is usually dependent upon the amount of contamination thought to be in the system. Three main factors can increase the stability of a national feed production system:

- (i) Feed bans – these regulations can range from the basic prohibition of feeding ruminant MBM back to ruminants; to prohibiting most animal proteins from being fed to all animals used for food production, including fish.
- (ii) Specified Risk Materials (SRMs) ban – this ban requires that high infectivity tissues such as bovine brain and spinal cord be removed from both the food and feed chain and be destroyed. The intent of this control is to remove the primary source of infectivity from the entire system to prevent the possibility of cross-contamination.
- (iii) Regulation of rendering – although no rendering process can completely remove all detectable infectivity, some are more effective than others. The best procedure identified to date requires 133°C at 3 bars of pressure for 20 minutes.

Experience in countries that have spent considerable effort to eliminate BSE has underlined the need for an extremely high level of compliance with feed controls in order to remove the agent from the system and prevent new infections in cattle. No complacency can be tolerated.

Bovine products and byproducts are widely used for both food and pharmaceuticals, and hence require the highest level of safety. Because of the hardy nature of the BSE agent and its high potential for crosscontamination, the most effective approach to protect bovine products and bovine-derived materials for human use from contamination by BSE is to ensure that infected animals or carcasses never enter processing plants. Because there are presently no diagnostic tools sensitive enough for detection of the disease during its long preclinical incubation, governments must rely on measures to prevent exposure through feed (see above) or prohibit high risk tissues (SRMs) from being used for food or pharmaceuticals.

Suggested disinfectants: BSE is not known to spread laterally between cattle or other animals; hence it is not

necessary to disinfect a premise where infected cattle have been. Regarding BSE, the need for disinfection may arise in diagnostic laboratories, food processing and pharmaceutical manufacturing plants. The agent of BSE shares with other TSE agents the property of unusual resistance to destruction. None of the standard disinfection methods is effective, including irradiation or exposure to various chemical disinfectants. Even harsher conditions that are capable of inactivating all other known pathogens (including bacterial spores), such as heating under pressure at 121°C, exposure to dry heat at 600°C, or immersion in 0.1 N NaOH or 0.5% bleach cannot assure complete inactivation. Currently, the only procedures known to completely eliminate detectable infectivity are exposure to dry heat at 1000°C, immersion in either 1 N NaOH or fresh undiluted bleach, and steam heating under pressure at 132°C. The preferred method is a sequential exposure to both NaOH and steam autoclaving inactivation treatments.

Notification: BSE is a reportable disease in the US.

Measures required for introducing animals to infected animal: This approach is not applicable.

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

Conditions for restoring disease-free status after an outbreak:

As BSE is not known to be laterally transmitted, no remediation for farms or zones within a country is needed. After a case of BSE has been detected within a country, certain measures must be taken to regain negligible risk status. As per the World Organization for Animal Health (OIE), countries detecting BSE must perform a risk assessment to identify historical and existing risk factors. The country must demonstrate that appropriate specific measures have been taken for the relevant period of time defined below to manage each identified risk.

EITHER:

- a) If there has been a case, every case of BSE has been demonstrated to have been imported and has been completely destroyed, and it has been demonstrated through an appropriate level of control and audit, including that of cross contamination, that for at least eight years neither meat-and-bone meal nor greaves derived from ruminants has been fed to ruminants;^{[1][1][1]}_{[SEP][SEP]} OR:
- b) If there has been an indigenous case, every indigenous case was born more than 11 years ago; and the below points have been complied with for 7 years.
 - An education program is in place for those involved in the livestock industry to report all suspected cases of BSE.
 - BSE is reportable and all suspect cases are investigated.
 - Diagnostics are carried out in accordance with the OIE laboratory manual. AND:
 - i) it has been demonstrated through an appropriate level of control and audit, including that of cross contamination, that for at least eight years neither meat-and-bone meal nor greaves derived from ruminants has been fed to ruminants. ii) All BSE cases, as well as:
 - all cattle which, during their first year of life, were reared with the BSE cases during their first year of life, and which investigation showed consumed the same potentially contaminated feed during that period, or
 - if the results of the investigation are inconclusive, all cattle born in the same herd as, and within 12 months of the birth of, the BSE cases if alive in the country, zone or compartment, are permanently identified, and their movements controlled, and, when slaughtered or at death, are completely destroyed.

Experts who may be consulted:

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BOVINE VIRAL DIARRHEA VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Artiodactyla	<p><u>Horizontal:</u> Primarily from a persistently infected animal (“PI”) but also from transiently infected animals. In “PI”, virus is shed heavily and <u>continuously</u> in <u>all</u> bodily secretions. Virus also transmitted by fomites.</p> <p><u>Vertical:</u> Infection of dam during first trimester can produce “PI”.</p>	<p><u>Horizontal:</u> Sub-clinical, respiratory disease, diarrhea, mucosal ulcers, fever, hemorrhagic syndrome, secondary infections, peracute death, and reproductive failure</p> <p><u>Vertical:</u> Infertility, abortion, stillbirths, weak calves and “PI.”</p>	Species and viral strain dependent. Infections can be sub-clinical or cause severe disease and death	Supportive care for transiently infected animals.	Testing, identification and elimination of “PI.” Vaccination with MLV is common in cattle and has been reported to prevent infection in alpacas without ill effects.	No

Fact Sheet compiled by: Peregrine Wolff

Sheet completed on: 2018

Fact Sheet Reviewed by: Beth Bicknese

Susceptible animal groups: Ungulates belonging to the order Artiodactyla (including *Bovidae*, *Suidae*, *Caprinae*, *Camelidae*, *Antilocapridae*, *Tragulidae* and *Cervidae*).

Causative organism: Single stranded RNA viruses belonging to the genus *Pestivirus* and Family *Flaviviridae*. Two species, BVDV-1 (11 sub-genotypes) and BVDV-2 (2 sub-genotypes), have different profiles. In the US, the three commonly isolated sub-genotypes from cattle are BVDV1a, 1b and 2a. Within the genotypes or strains, two 2 biotypes of BVDV classification are based on their effects on cell culture (cytopathic [CP] or non-cytopathic [NCP]). Infections with NCP strains are the most common and it is the NCP strains that result in “PI” animals. Because BVDV is an RNA virus it readily mutates resulting in genetic, antigenic and pathogenic variation.

Zoonotic potential: This disease is not considered to have zoonotic potential at this time. However, the virus can infect human cell lines.

Distribution: Worldwide distribution. The principal reservoirs of BVDV are persistently infected (“PI”) domestic cattle. Numerous wildlife species, both captive and free-ranging have been shown to be serologically positive for BVDV. Persistently infected individuals have been identified in captive and free-ranging wildlife, primarily cervid species.

Incubation period: Experimental infection in mule deer, white-tail deer and cattle, indicates that virus may be isolated from white blood cells, serum, plasma or nasal secretions as early as two days post infection.

Clinical signs: Infections can be transient with no apparent clinical signs or severe with pronounced morbidity and mortality. Both genotypes BVD-1 and BVDV-2 can cause the full spectrum of clinical presentations. BVDV is lymphotropic and immunosuppressive so diseased animals have an increased susceptibility to infectious diseases. Hematology may show mild to severe lymphopenia and neutropenia depending on the virulence of the strain to the host.

“Acute”, “Transient” or “Primary” disease syndromes are described from horizontal transmission

BOVINE VIRAL DIARRHEA VIRUS

which include:

Respiratory: Oculonasal discharge. Due to BVDV immunosuppressive effects clinical signs may be indicative of disease caused by other respiratory pathogens.

Gastrointestinal: Diarrhea and clinical signs resulting from lesions which are primarily ulcerous or erosive and which may involve any region of the GIT. Mixed infections with other common gastrointestinal organisms are not uncommon.

Hemorrhagic/thrombocytopenic: Thrombocytopenia, bloody diarrhea, prolonged bleeding times, petechial and ecchymotic hemorrhages, epistaxis, death.

Mucosal disease (MD): Seen only in “PI” animals that become “super infected” with a CP strain of BVDV. Clinical signs are secondary to severe ulcerative and erosive lesions throughout the gastrointestinal tract and potentially including lameness secondary to lesions associated with inter- digital ulcerations.

From vertical transmission, disease syndromes include:

Reproductive and fetal: Early fetal losses, mummified fetuses, abortions, stillbirths and congenital defects, poor doer during neonatal period.

Persistently infected animals: Persistently infected animals result if the dam becomes infected during the first trimester of gestation. In cattle, infection must occur between 45-125 days with a non-cytopathic strain of BVDV. In white-tail deer, infection occurring between days 45-52 of gestation resulted in a “PI” fawn. The fetus becomes infected and is immunotolerant to the infecting strain of BVDV and will shed high amounts of virus from all bodily fluids throughout its life. The “PI” animal may mount an immune response to heterologous strains of BVDV. Persistently infected individuals may exhibit, runtiness, immunosuppression and secondary infections, but “clinically normal” animals have been documented.

Post mortem, gross, or histologic findings: There are no pathognomonic lesions for BVDV. Pathological diagnosis may be made based on virus isolation or demonstration of the virus within tissues. Transiently infected animals will have gross and histopathological lesions consistent with their clinical syndrome. Persistently infected, but healthy animals may have few postmortem and histopathological lesions. Lymphoid depletion has been reported in both “PI” cattle and experimentally infected fawns.

Diagnosis: Primary goal is to identify the “PI” animal. Virus isolation is the “gold standard”. However, antigen capture ELISA (ACE), immunohistochemistry and RT- PCR are commonly utilized tests as they are rapid, sensitive, affordable and repeatable amongst diagnostic laboratories. Many tests do not differentiate between BVDV1 and 2 and other pestiviruses such as classical swine fever virus, border disease virus (endemic worldwide), pronghorn virus, HoBi-like [isolated in South America and Southeast Asia, Bungowannah (isolated in Australia) and giraffe (isolated in Africa)].

Most tests cannot differentiate between an acute and a persistent infection. The standard for diagnosis of PI infection is two positive tests on samples collected at least two weeks apart.

Material required for laboratory analysis:

Antemortem: Haired skin sample (ear notch or caudal tail fold), or whole blood (buffy coat) collected in EDTA are preferred samples.

Postmortem: Haired skin and lymphoid tissue (mesenteric lymph nodes, thymus, tonsils) spleen, and brain. These tissues should be collected for culture or immunohistochemistry (fresh and formalin fixed). Archived formalin fixed tissue blocks can be tested for BVD via PCR, however detection rates drop after 3 months – 1 year.

Relevant diagnostic laboratories: Check with your local veterinary diagnostic lab to see what tests they perform and the limitations of these tests for the species you are testing.

Treatment: Supportive care of transiently infected animals. Persistently infected individuals should be eliminated from the herd

Prevention and control: Identification and removal of “PI” individuals. All incoming artiodactyls (particularly domestic cattle, sheep and goats) should be quarantined and tested for the presence of BVDV virus.

BOVINE VIRAL DIARRHEA VIRUS

- 1) Animals that can only be handled once and with risk of exposure to BVDV: Combination of Antigen-capture ELISA (ACE) on haired skin combined with PCR on whole blood (buffy coat) collected in EDTA and antibody detection via serum neutralization will have the greatest likelihood of identifying “PI” that may be transiently infected. An animal that is positive on both ELISA (ACE) on haired skin, as well as RT-PCR on whole blood but is negative on serology is considered highly suspicious of being “PI” and should undergo follow up testing in 4-6 weeks. Animals that are positive on RT-PCR and have serum titers are most likely transiently infected individuals.
- 2) Pregnant females that have a serum antibody titer to BVDV: These animals may have been exposed to the virus within the first trimester of pregnancy and be carrying a “PI” fetus. They should be quarantined until the offspring is born. The offspring should then be tested for persistent infection via whole blood (buffy coat) RT-PCR in combination with ELISA (ACE) or immunohistochemistry on a haired skin sample. This will differentiate presence of virus in the face of maternal antibodies if the offspring is sampled post nursing.
- 3) Animals with viremia: should not be introduced to other artiodactyls that may be in the first trimester of gestation.
- 4) Individuals utilizing reproductive manipulation techniques should be alerted that BVDV has been isolated from commercial fetal calf serum.
- 5) Vaccination has not been well studied in wildlife. Alpacas are reported to be protected with no negative effects when vaccinated with a MLV. Vaccination in cattle is primarily focused on the prevention of fetal infections.

Suggested disinfectant for housing facilities: BVDV is an enveloped virus and susceptible to the following classes of disinfectants when used per recommended protocols - hypochlorites, chlorhexadine, alcohol, iodine and iodophores, quaternary ammonium compounds, phenolic disinfectants and aldehydes

Notification: None Required

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Pregnant animals should not be exposed to animals that are viremic. “PI” animals should be identified and removed from the herd.

Conditions for restoring disease-free status after an outbreak: identify and remove “PI” individuals. Any off-spring born to dams that were pregnant during the outbreak should be tested to insure that they are not “PI” and all new additions that may be at risk for infection with BVDV should undergo testing and quarantine prior to introduction to any artiodactyl species.

Experts who may be consulted:

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BOVINE BRUCELLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ungulates (cattle, bison, buffalo, elk, goats, sheep, camel, etc.), swine, carnivores, rodents, voles, pinnipeds, cetaceans, humans.	<p>Usually ingestion of materials contaminated by birthing or abortion products (fluids, placenta, fetus); direct contact with aforementioned materials or semen, ingestion of unpasteurized milk or dairy products.</p> <p>Fomites and mechanical vectors can transmit the organism. The organism can pass abraded skin or intact mucous membranes and persists in the environment for up to 60 days, especially at low temperatures or high organic material. Venereal transmission also can occur with <i>B. suis</i>, <i>B. ovis</i>, and <i>B. canis</i></p>	<p>Abortion (mid to late term), stillborn or weak calves, neonatal death, placentitis, endometritis, epididymitis, seminal vesiculitis, orchitis, testicular abscess, spondylitis or arthritis. Many times, no other outward clinical signs.</p> <p>Chronically infected animals may have decreased milk production or possible hygromas.</p>	<p>High morbidity in naïve herds. Generally, a mild disease in animals (except abortion) with chronically infected herds stabilizing at 30-50% seroprevalence.</p> <p>Produces undulant fever in humans that is chronic and debilitating, but not generally life threatening; however, <i>B. melitensis</i> and <i>B. suis</i> tend to induce myocarditis which can be associated with fatality.</p>	<p>Antibiotics in humans; none in animals</p>	<p>Avoidance, quarantine incoming animals. Test and slaughter of serologic reactors. Long term antibiotics for humans. But antibiotics are of questionable value in animals.</p> <p>Use of personal protective equipment (adequate gloves, protective clothing, respiratory and mucosal membrane protection) for vocational exposure in humans.</p>	Yes

Fact Sheet compiled by: S.W. Jack; updated by Frank Austin

Sheet completed on: 3 May 2011; updated 30 July 2013, April 2018

Fact Sheet Reviewed by: Steve Olsen; Julie Ter Beest; Jim Watson

Susceptible animal groups: Ungulates (cattle, bison, buffalo, elk, goats, sheep, reindeer, camel, etc.), swine, carnivores, rodents, pinnipeds, cetaceans, horses, and humans.

Causative organism: *Brucella* species generally have a preferred natural host but will frequently infect other hosts. *Brucella abortus* (cattle and humans) is the primary causative agent. However, there are other *Brucellae* that include: *B. melitensis* (small ruminants and cattle) although it is not present in US, this organism has been seen in humans in the US from imported non-pasteurized dairy products. *B. ovis* (small ruminants), *B. suis* (swine, reindeer – biovarian 4, cattle), *B. canis* (dogs), *B. neotomae* (rodents), *B. microti* (voles and foxes), *B. ceti* (cetaceans), *B. pinnipedialis* (pinnipeds) and *B. inopinata* (humans). *Brucellae* are gram negative non-enteric facultative intracellular coccobacilli.

Zoonotic potential: Relatively high.

Distribution: World-wide, although regionally it is limited in North America (Greater Yellowstone Area – *B. abortus* in bison and elk). *Brucella suis* transmitted from feral swine is increasingly occurring in cattle housed in the southeast US. Increasingly *B. melitensis* is occurring in cattle in central Asia and the Middle East.

Incubation period: Quite variable, 2 weeks to 1 year or longer.

BOVINE BRUCELLOSIS

Clinical signs:

Abortion, weak calves, neonatal death, placentitis, endometritis, epididymitis, seminal vesiculitis, orchitis, testicular abscess, and spondylitis may occur. Often, no other outward clinical signs. Chronically infected animals may be “poor doers”. In horses, infection may produce “fistulous withers”.

In humans, severe “flu-like” signs, fatigue, headache, fever, chills, night sweats, joint pain, backache, weight and appetite decreases, spontaneous improvement but recrudesces (“undulant fever”). In cattle, *B. suis* does not appear to cause abortions in cattle but does colonize the mammary gland with subsequent high CFU within milk.

Post mortem, gross, or histologic findings:

Aborted fetus: autolysis (common intra-uterine death), subcutaneous edema, serosanguineous fluid in body cavities (peritoneum, pericardium and pleura), possible spleno- and hepatomegaly, pneumonia. Placentitis (inflamed or necrotic cotyledons), “leathery” intercotyledonary zones.

Adults: granulomatous to purulent inflammation of reproductive tract, hygromas, draining tracts.

Diagnosis:

Serology (agglutination – screening; FPA and CF – confirmatory; ELISA -available), Milk ring Test (BRT), perhaps bacterial isolation, newer PCR tests are available. Confirmatory tests: Culture of the organism is the “Gold Standard” for diagnosis. Confirmatory tests include standard tube test, Rivanol test, complement fixation test (CF), fluorescence polarization assay (FPA), particle concentration fluorescence immunoassay (PCFIA), semen plasma test, and standard plate test. 9 CFR Ch. I Part 78

Material required for laboratory analysis:

Microscopic examination of abortion products, Stamp’s modification of Ziehl-Neelsen method for presumptive diagnosis (low yield procedure). Culture of fetal membranes, fetal stomach contents, many fetal organs, vaginal secretions, milk, semen, arthritis or hygroma fluids (not often successful). Serology (ante-mortem or post-mortem) AGID, ELISA (most common procedures).

Relevant diagnostic laboratories:

Samples are to be tested for brucellosis only in cooperative State–Federal brucellosis laboratories or by persons who are authorized by Program officials to conduct the tests. See “Brucellosis Eradication: Uniform Methods and Rules” by the USDA APHIS.

Treatment: None in cattle.

Prevention and control:

All states are free of disease in commercial herds, except for the Greater Yellowstone Park Area. Test and slaughter in domestic cattle. Bulk Milk test (*Brucella* Ring Test) for herd; individual tests include blood agglutination and/or the CARD test. Animals that are positive are quarantined and only able to move to slaughter; samples are collected for culture. Market Cattle Identification (back tags) on sale barn animals to allow trace back in the event of a seropositive reactor. Cattle vaccines are available (e.g. strain 19 [old] and RB51). Personal protective equipment (PPE) for humans is good preventive measure, especially with exposure to birthing fluids.

Regarding vaccination in zoo and wildlife species:

- Abortions have been associated with *Brucella abortus* strain 19 live vaccination in bison; this vaccine offers limited protection against infection and abortion in bison and elk.
- *Brucella abortus* strain RB51 is a live vaccine for use in cattle that protects at least as well strain 19, does not cause abortion, and induces antibodies that can be distinguished from antibodies induced by natural infection.
- Calfhood vaccination of bison with strain RB51 vaccination may reduce transmission of brucellosis; the vaccine is not, however, recommended in elk or reindeer.

BOVINE BRUCELLOSIS

Suggested disinfectant for housing facilities: Most disinfectants are effective, e.g. 2.5% hypochlorite, 70% ethanol, formalin, glutaraldehyde, xylenes, iodophors, phenolics, 20% slaked lime, 2-3% caustic soda, 10 minutes boiling.

Notification: Contact state veterinarian and/or AVIC.

Measures required under the Animal Disease Surveillance Plan:

Brucellosis Eradication Program (see Uniform Methods and Rules)

- Bulk Milk testing (*Brucella* Ring Test)
- Serum testing at sale barns (blood agglutination test)
- Fluorescence polarization assay
- CARD test (cow side rapid diagnostics)
- Market Cattle Identification (back tags)

Currently in the US, sampling surveillance is performed at large slaughter plants or in states with high risk for exposure.

Measures required for introducing animals to infected animal: Infected animals are quarantined and should not be introduced to other animals.

Conditions for restoring disease-free status after an outbreak: Brucellosis Eradication Program (see Uniform Methods and Rules) 12 consecutive months without seropositive evidence of disease is required.

Experts who may be consulted: Federal and state veterinary authorities (AVIC and state veterinarian, respectively); international (OIE).

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MARINE MAMMAL *BRUCELLA*

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p>Marine mammals, humans.</p> <p>Experimental disease has been demonstrated in cattle and guinea pigs, suggesting other terrestrial animals are likely susceptible.</p>	<p>Unknown, but likely similar to terrestrial species, including <i>in utero</i> (vertical transmission), ingestion of milk or contaminated fish, mucous membrane exposure, sexual contact, or contact with infected placenta or birthing fluids.</p> <p>Lungworms appear to serve as vectors of marine brucellae.</p>	<p>Variable, depending on affected organ system and strain of bacteria.</p> <p>Nonapparent disease to stranding, abortion, placentitis, infertility (including orchitis and epididymitis), neurologic signs, cutaneous lesions, osteomyelitis and arthritis, cardiovascular disease, and respiratory distress/disease have been reported in cetaceans.</p>	<p>Variable; serologic evidence of exposure without clinical disease is common.</p> <p>Cetaceans may exhibit inapparent, acute or chronic disease states.</p> <p>Except for a single placenta sample demonstrating placentitis in a Northern fur seal, pathology in pinnipeds has not yet been reported.</p>	<p>A single case of successful treatment of a pulmonary abscess, including intralesional amikacin and oral doxycycline and rifampin, has been reported.</p> <p>WHO reported that human disease may respond to similar antibiotic treatment including rifampin and doxycycline.</p>	<p>Not well defined; serological tests can be used for screening.</p> <p>PCR or culture may identify animals actively shedding bacteria.</p> <p>General biosecurity and quarantine protocols.</p>	Yes.

Fact Sheet compiled by: Claire Erlacher-Reid

Sheet completed on: updated on 30 May 2018

Fact Sheet Reviewed by: Sarah Churgin, Kirsten Gilardi

Susceptible animal groups: Cetaceans, pinnipeds, sea otters, and polar bears; also humans. There are no known reports of isolation or seropositivity in manatees or dugongs.

Causative organism: Small (< 1.5 µm by 0.7 µm), facultative intracellular, gram-negative coccobacilli. *Brucella* species typically have different host preferences, virulence, and zoonotic potential despite 97-99% similarity at genome level. Marine mammal *Brucella* spp. are currently identified as *B. ceti* (cetacean-origin strains) and *B. pinnipedialis* (pinniped-origin strains). Molecular characterization suggests two major *Brucella ceti* clades, one group primarily associated with porpoise isolates and another primarily associated with dolphin isolates. Distinctive genetic variations in *B. ceti* isolates appear to correlate with oceanic distribution and preferred host.

Zoonotic potential: Yes. Outside of laboratory-associated infection, the route of exposure is not known, but food-borne exposure (ie. raw seafood) is suspected. Typing of human isolates suggests increased zoonotic potential associated with a single genotype (ST27). Clinical signs reported in humans include fever, headache, lethargy, myalgia, sinusitis, arthritis, fatigue, and neurological disease in rare cases.

Distribution: Globally distributed in wild species of cetaceans (including Phocoenidae, Delphinidae, Monodontidae, Balaenidae, and Balaenopteridae) and pinnipeds (including Phocidae, Otariidae, and Odobenidae). Seroprevalance fluctuates in wild populations over time.

Incubation period: Not defined.

Clinical signs: Variable, depending on affected organ system and strain of bacteria. Although bacterial strains are host-associated, cross-species infections occur frequently, and may affect expression of disease.

MARINE MAMMAL *BRUCELLA*

Seropositivity and bacterial isolation are reported in pinnipeds without overt disease, suggesting host-adapted or low-pathogenic strains. Stranding, inanition, infertility, abortion, neurologic signs, cutaneous and pulmonary abscessation and musculoskeletal disorders have been reported in cetaceans. Chronic, severe osteoarthritis has been reported in a sea otter.

Post mortem, gross, or histologic findings: A single Northern fur seal placenta sample demonstrated severe placentitis in association with positive PCR testing and immunostaining for *Brucella*. Otherwise, pathology has not been previously reported in pinnipeds. In cetaceans, lesions are primarily seen in reproductive tract (orchitis/epididymitis, necrotizing placentitis/endometritis), reticuloendothelial/hemolymphatic systems (lymphadenitis, splenic necrosis), central nervous system (meningoencephalitis), musculoskeletal system (discospondylitis, osteomyelitis, and arthritis), and lung (interstitial pneumonia, pulmonary abscesses and granulomas, and lungworm associated pneumonia). Additional findings have included blubber abscesses, visceral necrosis, steatitis, mastitis, hepatomegaly, and vegetative endocarditis. Chronic granulomatous osteoarthritis and myelitis were noted in a sea otter with marine *Brucella*.

Diagnosis: Diagnosis can be divided into direct identification and indirect screening methods of detection. Bacterial isolation in culture from infected materials (CSF, brain, lymph node, and lung are most commonly used) remains the gold standard; however this method is difficult at best. Farrell's media or *Brucella*-agar with 5% horse blood may be used and incubated with 5-10% CO₂ for up to 14 days. Molecular characterization by polymerase chain reaction (PCR) methods include outer membrane protein polymorphisms, infrequent restriction site-derivative PCR, insertion sequence IS711 profiling, multilocus sequence typing (MLST) and multiple loci variable number tandem repeat analysis (MLVA). Detection of *Brucella* from a blowhole swab utilizing real-time PCR appears to correlate well with detection of *Brucella* in internal lung tissue. Real-time PCR may be used to screen for the presence of *Brucella* DNA in live cetaceans via blowhole swabs, blood, and/or fecal samples. Immunohistochemical staining can identify the presence of bacteria in tissues, but has not proved to be as sensitive as other methods for surveillance. A number of serologic methods are available for screening (*i.e.* Rose Bengal test and buffered plate agglutination test, the complement fixation test, enzyme-linked immunosorbent assays (ELISA) or the fluorescence polarization assay (FPA), but sensitivity and specificity are variable and seropositivity does not correlate with active disease or bacterial shedding.

Material required for laboratory analysis: Fresh or frozen tissue, especially aborted fetuses (stomach contents, spleen and lung), fetal membranes, vaginal secretions (swabs), reticuloendothelial system (lymph nodes and spleen), brain/spinal cord/CSF, liver and kidney, or other gross lesions. In live animals, bacteria have been recovered from feces, blood, blow hole swabs, fine needle aspirates, and lungworms.

Relevant diagnostic laboratories: Clinicians with susceptible populations of marine animals should inquire about routine bacteriologic testing through their local or regional veterinary or medical diagnostic laboratories.

For culture and bacterial typing:

USDA/APHIS National Veterinary Services Laboratories
Mycobacteria and *Brucella* Section– National Reference Laboratory
1920 Dayton Ave.
Ames, Iowa 50010
(515) 337-7388

Routine culture:

Marine Mammal Diagnostic Laboratory
UC Davis One Health Institute
School of Veterinary Medicine
1089 Vet Med Drive, VM3B
Davis, CA 95616
530-752-4167

MARINE MAMMAL *BRUCELLA*

PCR for *Brucella* spp:

Athens Veterinary Diagnostic Laboratory
College of Veterinary Medicine, University of Georgia
501 D.W. Brooks D.
Athens, GA 30602-7383
(706) 542-5568
www.vet.uga.edu/dlab

Marine mammal cELISA and qPCR:

Mystic Aquarium & Institute for Exploration
Dept. of Research and Veterinary Services
Tracy Romano 55
Coogan Blvd.
Mystic, CT 06355-1997
(860) 572-5955

Treatment: A single successful treatment of pulmonary abscess was reported in a captive dolphin. The treatment included intra-lesional amikacin followed by six to eight weeks of oral doxycycline and rifampin.

Prevention and control: Surveillance using serological tests can be used for screening population exposure. Blowhole and/or fecal PCR may identify animals actively shedding bacteria. General biosecurity and quarantine protocols are recommended for marine mammal rehabilitation and aquarium facilities.

Suggested disinfectant for housing facilities: General measures for cleaning and disinfection should reduce environmental bacterial contamination, as *Brucella* bacteria are readily killed by common disinfectants and do not appear to live long outside the host cells.

Notification: Marine strains of *Brucella* are not currently reportable to State, Provincial or Federal bodies.

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: These measures are not yet defined in marine species. Paired serology may be recommended for animals planned to be introduced, including use of appropriate quarantine protocols.

Conditions for restoring disease-free status after an outbreak: Not defined in marine species.

Experts who may be consulted:

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MARINE MAMMAL *BRUCELLA*

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PORCINE BRUCELLOSIS (*Brucella suis*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Domestic and wild swine, European hare, reindeer and caribou, wild rodents.	Direct contact with infected animals or body discharges, ingestion of infected materials, venereal transmission, fomites.	Can be asymptomatic. Reproductive disease, lameness, posterior paralysis, pyrexia.	Mild to severe; death is rare.	None	No vaccine. Test and quarantine new animals, limit contact with wild swine, isolate or eliminate infected animals, decontaminate premises.	Yes

Fact Sheet compiled by: Cora Singleton

Sheet completed on: updated 8 August 2018.

Fact Sheet Reviewed by: Suzanne Kennedy-Stoskopf

Susceptible animal groups: Biovars 1 and 3 – domestic and wild swine. Biovar 2 – wild swine, European hare. Biovar 4 – reindeer and caribou maintain the infection; moose, cattle, Arctic fox, and wolves can also be infected. Biovar 5 – wild rodents in former USSR.

Causative organism: *Brucella suis*, a small Gram-negative coccobacillus, with five biovars.

Zoonotic potential: Yes. Human brucellosis (biovars 1, 3, and 4) is mainly an occupational risk, seen in farmers, veterinarians, and abattoir workers. Biovar 2 is zoonotic but rarely reported in humans.

Distribution: Biovar 1 and 3 – worldwide. Biovar 2 – Europe. Biovar 4 – Arctic regions (including Alaska and Canada) and Russia. Biovar 5 – former USSR.

Incubation period: Bacteremia usually develops within 1 to 7 weeks (mean 2 weeks) after exposure. Bacteremia can last up to 90 days.

Clinical signs: Infection can be asymptomatic. Clinical problems include reproductive disease (infertility, abortion, weak or stillborn piglets, orchitis, epididymitis, metritis), and pyrexia with less common signs of lameness with swollen joints and tendon sheaths (due to bursitis, synovitis, arthritis), posterior paralysis, spondylitis, and abscess formation in organs. Death is rare.

Post mortem, gross, or histologic findings: Lesions are variable and may include abscess formation in affected organs including the liver, spleen, kidneys, reproductive tract, placenta, lymph nodes, joint capsules, tendon sheaths, and bones.

Diagnosis: Agent identification – bacterial culture, PCR
 Serology – Buffered *Brucella* antigen tests such as buffered plate agglutination test and Rose Bengal test; ELISA; fluorescent polarization assay; complement fixation test. Serologic tests are more useful for identifying infected herds than infected individuals. Buffered antigen tests often preferred.
 USDA presumptive tests – Buffered acidified plate antigen test, standard card test. USDA confirmatory tests – Standard tube test, particle concentration fluorescence immunoassay.

Material required for laboratory analysis: Bacterial culture – lymph node, reproductive organs, vaginal swab, products of abortion, synovial fluid, semen, blood. Serologic tests – Serum.

Relevant diagnostic laboratories: State and federal laboratories that have been specifically approved for conducting swine brucellosis serology.

Treatment: No treatments have been proven effective and economically feasible.

Prevention and control: The United States maintains a federal program for eradication of brucellosis from domestic livestock. Porcine brucellosis is controlled through serologic testing and carcass inspection at slaughter, with trace-back investigations of any suspicious cases. Pigs are not vaccinated against brucellosis. Disease can be prevented by testing and quarantining new animals, eliminating contact with feral swine, and practicing good sanitation.

PORCINE BRUCELLOSIS (*Brucella suis*)

Suggested disinfectant for housing facilities: Hypochlorite solutions, 70% ethanol, isopropanol, iodophores, phenolic disinfectants, formaldehyde, glutaraldehyde, xylene.

Notification: *B. suis* is considered eradicated from domestic swine in the United States. Infections are reportable to the USDA/APHIS through the State Veterinarian or the federal Area Veterinarian in Charge. The disease is also reportable to the World Organization for Animal Health (OIE).

Measures required under the Animal Disease Surveillance Plan: Reportable to USDA/APHIS.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Perform the necessary tests to determine presence or absence of brucellosis in individuals/herd and report results. Quarantine or depopulate infected or exposed animals. Clean and disinfect premises, vehicles, and equipment.

Experts who may be consulted:

Iowa State University College of Veterinary Medicine

Department of Veterinary Diagnostic and Production Animal Medicine

Phone: 515-294-1950

Fax: 515-295-3564

<http://vetmed.iastate.edu/vdpam/>

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CAMPYLOBACTERIOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, including humans; birds; reptiles; fish; and shellfish	Food- or water-borne; fecal-oral spread; direct contact with contaminated surfaces or contact with infected animals	Host-specific: none to severe; diarrheal disease - watery or bloody; possibly with fever, abdominal cramps, nausea, and vomiting; other illnesses, such as abortion and infertility, and periodontal disease	Mild to life threatening; gastroenteritis, with possible sepsis and disseminated infections; children, immune-compromised individuals and the elderly may be at greater risk. Long-term consequences (such as arthritis or Guillain-Barré) occur rarely in people	Extra fluids to remain hydrated as long as diarrhea persists. Recovery often occurs without antibiotics, although they may be used to shorten the duration of clinical signs if administered early in course of disease.	Practice sanitary food preparation; use good personal and environmental hygiene; wear gloves when working with infected animals or surfaces in contact with their feces; wash hands with soap and water. To reduce venereal transmission, use strict hygiene, artificial insemination and vaccination; tetracycline may prevent abortion in ewes	Yes

Fact Sheet compiled by: Teresa J. Sylvina (previously published as Taranjit Kaur)

Sheet completed on: 13 April 2011; updated 22 July 2013

Fact Sheet Reviewed by: Jatinder Singh, Michael R. Cranfield

Susceptible animal groups: Mammals, including humans; birds; reptiles, fish, and mollusks

Causative organism: *Campylobacter* spp.(various)

Zoonotic potential: Yes

Distribution: Surfaces (wet cutting boards or utensils) where raw or partially cooked meat (particularly poultry) is prepared; surface waters and mountain streams exposed to feces from cattle and wild birds; surfaces in contact with feces from infected agricultural animals, pets, wild, zoo and lab animals.

Incubation period: 2-5 days, and may be up to one week

Clinical signs: Clinical signs are host-specific; cross-infection is possible and range from none to severe. Diarrhea tends to be watery or may be bloody; fever, abdominal cramps, nausea, and vomiting may also be present; other illnesses, such as abortion, stillbirths or infertility may occur in cattle and sheep.

Post mortem, gross, or histologic findings

CAMPYLOBACTERIOSIS

Enteric campylobacteriosis: Biopsy specimens from people have shown acute colitis with inflammatory infiltrates of the lamina propria and crypt abscesses. Organisms can stably colonize the small and large intestine, although most animals show cecal and colonic lesions with typhlocolitis; marked inflammation of lamina propria, dominated by neutrophilic polymorphonuclear and mononuclear cells that sometimes extend into submucosa; crypt abscesses and damage to the crypt epithelium is common; a compromised epithelial surface also been observed in most species.

Bovine and ovine genital campylobacteriosis: Abortion occurs most frequently in late pregnancy with occasional infertility. Liver shows typical gray, necrotic foci 1-2 cm in diameter; fetuses usually edematous and body cavities contain reddish fluid; fetal membranes edematous and cotyledons pale and necrotic but lesions do vary. Curved bacteria in stains of cotyledon impressions or fetal abomasal fluid. Gram negative organisms found in wet preps of abomasal fluid examined by dark-field or phase-contrast microscopy.

Diagnosis: Bacterial culture of fresh feces; darkfield examination of abomasal contents or culture of placenta or abomasal contents or in uterine discharge

Material required for laboratory analysis: Fresh fecal samples in enteric transport kit and storage at 4°C

Relevant diagnostic laboratories: Laboratories capable of bacteriologic culturing on selective culture media incubated under microaerobic conditions, and temperatures allowing growth of non-thermotolerant species. *Campylobacter* species are difficult to isolate and suboptimal conditions for isolation will yield false-negative results. Variations in laboratory practices have been reported, also suggesting variations in specimen handling and processing, which likely influence recovery and detection of *Campylobacter* species.

Treatment: Usually no medical treatment is necessary. Rehydrating levels of fluids should be administered during diarrheal disease. Appropriate antibiotics, such as erythromycin, may be a consideration in some cases. Suspected genital campylobacteriosis should be confirmed by isolation of organisms from herd bulls, selected infertile non-pregnant cows or aborted fetuses or cotyledons.

Prevention and control: Raw poultry meat should not be prepared on a cutting board then used unwashed for other food items, especially when not cooked after handling. Appropriate hygiene in food preparation should include separate cutting boards for proteins and produce. Unpasteurized milk and untreated surface water and mountain streams should be avoided. Wash hands using soap and running water after contact with animals, their enclosures, and other surfaces that are in contact with feces from animals. Animals infected with genital campylobacteriosis should not be utilized for breeding.

Suggested disinfectant for housing facilities: After cleaning gross contamination, diluted bleach (15ml in one quart of water) applied to dry or wiped dry after 10 minutes. Other disinfectants may be used; check disinfectant label to verify its effectiveness against *Campylobacter* spp.

Notification: Report cases to the local health department if zoonotic transmission occurs, depending on the state.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Maintain infected animal in a quarantine situation until the infection is cleared. Do not house infected animals with immune compromised animals.

Conditions for restoring disease-free status after an outbreak: Improvements in personal and environmental hygiene can be directed at animal husbandry and health staff. Education efforts can be directed toward proper food handling techniques, and toward avoiding consumption of potentially contaminated food, milk or water.

Experts who may be consulted:

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CAMPYLOBACTERIOSIS

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CANINE DISTEMPER VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All terrestrial families in the order Carnivora. Also known cases in marmots, phocids, primates, tamandua, tayassuids, and sloths.	Highly contagious! Aerosol of respiratory exudate is primary mode but other body excretions and secretions may be infective. Vaccinal, or vaccine-induced, distemper possible.	Respiratory, gastrointestinal, integumentary, ophthalmic, CNS. Hyperkeratosis of footpads and myoclonus.	Variable. Inapparent to peracute death.	Secondary infections. Supportive care	Vaccination! Keep infected animals isolated. Exclusion of potential reservoirs (e.g., domestic dogs, raccoons).	No. However, evidence of correlation of CDV with some human diseases.

Fact Sheet compiled by: Sharon L. Deem

Sheet completed on: updated 31 July 2018

Fact Sheet Reviewed by: AJ Marlar, Cara Field

Susceptible animal groups: Species within all terrestrial families of the order Carnivora (Canidae, Mustelidae, Procyonidae, Mephitidae, Hyaenidae, Ursidae, Viverridae, Herpestidae, and Felidae). Phocids also infected with CDV. Pinnipeds, sea otters, and cetaceans susceptible to closely related viruses (e.g., PDV, PMV, and DMV). Additionally, CDV disease has been confirmed in primates, marmots, tayassuids, tamandua and sloths. Mustelids are exquisitely susceptible, with mortality approaching 100%.

Causative organism: Canine distemper virus. Single-stranded, enveloped RNA virus within the family Paramyxoviridae, subfamily Paramyxovirinae, and genus Morbillivirus. Related to measles, rinderpest, and peste des petits ruminants.

Zoonotic potential: No. Some correlation with human diseases and growing concern with the mutability and changing epidemiology of CDV.

Distribution: Worldwide.

Incubation period: 7-18 days in domestic dogs. Variable with species and across individuals but estimated 1 week to 1 month.

Clinical signs: Signs associated with respiratory, gastrointestinal, integumentary, ophthalmic, and the central nervous systems are commonly seen. Which system(s) is/are affected depends on species, as well as strain virulence and environmental conditions. Animals are often depressed with mucopurulent, oculonasal exudates. Nasal and digital hyperkeratosis (hard pad) and involuntary muscle twitching are characteristic in domestic dogs. Acute conjunctivitis and occasionally uveitis, but in less severe cases, keratoconjunctivitis sicca and chorioretinal lesions are common. Differential diagnoses must include rabies and other viral encephalitides, respiratory infections, toxoplasmosis, canine parvovirus, lead poisoning, and bacterial enteritides.

Post mortem, gross, or histologic findings: Most significant gross lesions are pneumonia, depletion of lymphopoietic organs, and hyperkeratosis of the nose, foot pads, and eyelids. Common histologic findings are hyperkeratosis of the nose, foot pads, and eyelids. Eosinophilic inclusion bodies are present in many organs (most commonly cytoplasmic but occasionally intranuclear) including the CNS, urinary bladder, and bronchial epithelium. Cytoplasmic inclusion bodies in the gastric mucosa and bile ducts and diffuse interstitial giant cell pneumonia often followed by suppurative bronchopneumonia. Often lymphoid depletion, diffuse interstitial pneumonia, and perivascular lymphoplasmacytic infiltration in areas of demyelination and neuronal degeneration of the CNS. Syncytial giant cells in the lungs and CNS white matter, anterior uvea, and lymph nodes may also be present. In contrast to histologic lesions identified in the domestic dog, lungs of large felids may show diffuse alveolar type 2 cell hyperplasia with intracytoplasmic and intranuclear viral inclusion

CANINE DISTEMPER VIRUS

bodies. Additionally, feline brain histopathology may lack the typical canid pattern of demyelination with astrocytosis and vascular cuffing. Most cats have had mild, patchy CNS lesions compared with those of canids.

Diagnosis: Clinical signs, especially hyperkeratosis of foot pads and nose, and myoclonus are highly suggestive of CDV. Clinical pathologic changes including absolute lymphopenia, thrombocytopenia, regenerative anemia, decreased albumin, and increased alpha and gamma globulin concentrations may be present. Cytologic evaluation and/or immunofluorescence of conjunctival scrapes, buffy coat smears, CSF, skin or foot pads may also demonstrate intracytoplasmic inclusion bodies. Paired sera by viral neutralization or the indirect fluorescent antibody test to show a four-fold rise in antibody titer may be of value although often unrewarding as many animals die before building measurable antibody titers. Antibodies in CSF may be more diagnostic than serum. Newer ELISAs have been developed to detect IgM and IgG antibodies allowing determination of recent infection or vaccination.

Material required for laboratory analysis: Unfixed lung, liver, lymph nodes, brain, and spleen of dead animals with suspected CDV infection should be collected for viral isolation, fluorescent antibody and/or RT-PCR. RT-PCR assays are the test of choice for antemortem testing on oral swabs, blood, skin biopsies or urine samples. Immunohistochemistry on formalin-fixed tissues or FA on frozen sections provides definitive evidence of CDV infection. Vaccine virus may be differentiated from street virus by different target cell susceptibility, but sequencing of PCR products is the most definitive test to differentiate between vaccine and wild type viruses.

Relevant diagnostic laboratories: In the US, the Animal Health Diagnostic Center at Cornell, Michigan State Diagnostic Laboratory, and Colorado State Diagnostic Laboratory all routinely perform diagnostic tests for CDV. In Canada, biomaterials can be sent to Ontario Veterinary College. Other provincial laboratories in Canada should also be able to run CDV diagnostics.

Treatment: No specific therapy for animals with clinical canine distemper is available. Nonspecific treatment is supportive and includes fluids, antibiotics (for secondary bacterial infections), and medications to minimize CNS inflammation and seizure activity.

Prevention and control: Vaccination is the mainstay of prevention. In non-domestic species, recombinant vaccines are the safest. Exclusion of reservoir species from zoo sites, whenever possible, is important. Quarantine all animals suspected of being infected with CDV. Paired CDV titers should be used to monitor potentially naïve carnivores particularly when in quarantine before putting with others, or with breeding females to enhance pup titers.

Suggested disinfectant for housing facilities: CDV, being an enveloped virus, is fairly labile in the environment. Extremely susceptible to ultraviolet light, heat, desiccation, and common disinfectants (e.g., formaldehyde, ether, chloroform, phenolic compounds, and quaternary ammonium compounds.)

Notification: None required.

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: Maintain infected animal in a quarantine situation until asymptomatic. May be necessary to cull animals with residual CNS complications.

Conditions for restoring disease-free status after an outbreak: Clean infected environment with any of the common disinfectants. Vaccination of susceptible species is imperative. Vaccines available are modified live (MLV), killed and recombinant. MLV in species safe to vaccinate and probably promotes life-long immunity but many vaccinal or vaccine-induced, infections have resulted from MLV vaccines in wildlife species. Currently the Purevax® Canine Distemper (Merial) recombinant vaccine is recommended for non-domestic carnivore species, but other products (Recombitek® C3 by Merial) have been used in zoological species but often multivalent and use may not be indicated in some species.

Experts who may be consulted:

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CANINE DISTEMPER VIRUS

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***Chrysosporium* anamorph of *Nannizziopsis vriesii*: *Nannizziopsis*,
Paranannizziopsis, and *Ophidiomyces ophidiicola* (Under reclassification)**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Reptiles	-Direct -Indirect (via fomites and environmental contamination)	Variable dermatitis; Cellulitis and edema may be present. Internal organ invasion with <i>O. ophidiicola</i>	Mild to severe but high mortality is possible	Itraconazole; Voriconazole, Terbinafine (nebulization/ SQ implants/ injection)	Proper disinfection of housing areas; avoid contaminated fomites; prevent contact with infected animals	No direct transmission from animals reported but humans can be infected

Fact Sheet compiled by: E. Marie Rush

Sheet completed on: updated 1 May 2018

Fact Sheet Reviewed by: Bonnie Raphael, Tim Georoff

Susceptible animal groups: Reptiles

Causative organism: *Nannizziopsis* spp., *Ophidiomyces* spp., *Paranannizziopsis* spp.

Formerly, this grouping was *Chrysosporium* anamorph of *Nannizziopsis vriesii* (CANV) fungus. Recent taxonomic publications have identified new epidemiological information about these fungi grouped under the CANV appellation. While *Nannizziopsis vriesii* does produce a *Chrysosporium* anamorph in culture, all CANV-like isolates differ so that an overarching CANV appellation is discouraged.

For example, the “CANV” isolates that caused fatal disease in tentacled snakes have been reclassified as two species of *Paranannizziopsis*, which has not been isolated from other reptile species. *Paranannizziopsis* includes four species that infect squamates and tuataras. *Ophidiomyces* (belonging to the Order Onygenales) is a potent pathogen of snakes and associated with “Snake Fungal Disease,” but it has not yet been recovered from ill lizards or crocodiles so may not be a threat to these taxa. *Nannizziopsis guarroi* is the main causative agent of “Yellow Fungus Disease,” a common infection in bearded dragons, green iguanas, and other lizards. Classically dermatomycoses in reptiles are linked with stress and substandard husbandry in captive animals however experimental challenge of veiled chameleons (*Chamaeleo calytratus*) with *Nannizziopsis dermatitidis* confirmed the organism can act as a primary pathogen.

Zoonotic potential: While it is not directly transmitted from animals to humans, infection has been reported in two human cases where pre-existing immunosuppression was present. There are multiple subspecies of reptile infective organisms. Human species of these organisms have not been recovered in reptiles.

Distribution: Worldwide.

Incubation period: 2-5 weeks

Clinical signs: Infection is often through a breach in the skin. *Ophidiomyces* is the likely causative agent of “Snake Fungal Disease”, however the two have not always been found in tandem. Slow progression occurs from dry, hyperkeratotic plaques or vesicles to exudative lesions with excessive crusting that may later darken and slough. Snakes with *Ophidiomyces*, may have increased ecdysis frequency, abnormal resting in areas of the enclosure and anorexia. Skin may have fissures or thickening and upon pressure or incision into these areas, exudate may be expelled. Cellulitis can present concurrently. In advanced disease, general debilitation of the animal may be noted and deeper tissues – including muscle and bone – become affected. Hemogram and chemistry panels may be normal during this infection. In pygmy rattlesnakes, corticosterone levels were proportionately increased in direct correlation with severity of clinical SFD, recapture, decreased reproduction in females and decreased body condition score. Clinical signs correlated

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with decreased environmental temperatures have been established for SFD. *Paranannizziopsis australasiensis* has been reported in a tuatara with clinical signs and positive testing on PCR.

Post mortem, gross, or histologic findings: Initially, there will be hyphae proliferating in the epidermis (stratum corneum) with subsequent deeper invasion in most cases. Progression to liquefactive necrosis of the epidermis with or without granulomatous inflammation of the dermis is noted over time. Terminal chains of arthroconidia may be seen on hyphae, and with *Ophidiomyces*, granulocytic change at sites of microinvasion.

Diagnosis: Clinical signs are suggestive. Fungal culture of the organism on Mycosel™ Agar with incubation at 25-28°C. Histopathology can be performed. PAS stained sections of tissues will reveal hyphae in the keratin layer, epidermis, dermis and occasionally skeletal muscle layers depending on severity of disease. Fungus can be identified by PCR. Although Taqman RT PCR and PCR are considered the most sensitive and specific of the diagnostic tests, not all tissues may be positive, even if grossly visible lesions are present. In a study with *O. ophidiicola*, 98% of culture positive (and 40% of culture negative) snakes were found to be positive on RT-PCR. Of these snakes, 20 showed clinical signs and 16 had no clinical signs of infection. This study suggests that some asymptomatic snakes (~6%) may harbor low levels of fungus, and PCR paired with histopathology is recommended for definitive diagnosis. Massasaugas in Illinois with *Ophidiomyces* showed changes in WBC counts, lymphocytes and basophils noted retrospectively over peak years.

Material required for laboratory analysis: Frozen and formalin-fixed representative tissue samples from multiple organ systems (including skin, muscle, and bone) of necropsy specimens. Biopsies from live animals should be divided and submitted chilled for culture and fixed for histopathology.

Relevant diagnostic laboratories: Most diagnostic laboratories are capable of culturing of this organism. Pre-emptive contact with microbiologist prior to sample submission greatly increases the chance of diagnosis.

Treatment: Itraconazole and voriconazole can be used systemically. Terbinafine (10mg/kg PO SID x 7 days; pulse repeat Q3wk until one-week past resolution of signs) pulsed with itraconazole or voriconazole. In a study with timber rattlesnakes and massasaugas, voriconazole via SQ pump led to adequate levels in timber rattlesnakes, but not massasaugas. Levels post injection in cottonmouths were maintained for 12-24 hours. Cloacal administration in cottonmouths did not reach adequate levels, and several snakes died after a single injection without further treatment. In cottonmouths, terbinafine reached peak concentrations at 0.5-4 hours post nebulization, and on day 1 using subcutaneous implants (which maintained therapeutic levels for over 6 weeks).

Although topical disinfection of skin lesions with chlorhexidine solution may be helpful, alone it is not likely to be successful so combined approach is needed. Cutaneous lesions can be debrided aggressively along with topical antifungal and antibacterial dressings. Mycetomas should be considered for surgical excision in addition to systemic treatment. Prognosis for deeper structure involvement (e.g., bone) is guarded to poor.

Prevention and control: Optimization of husbandry conditions is critical for most reptiles to prevent disease as the problem is exacerbated by suboptimal husbandry. Housing areas should be thoroughly disinfected between individuals and any porous material from the enclosures should be discarded if unable to be sterilized or properly disinfected (i.e. substrate, drift wood furniture, etc). Proper quarantine measures for new animals should be followed. Separation of infected animals from healthy animals should be done until infection is completely cleared, based on biopsies and culture.

Suggested disinfectant for housing facilities: Bleach and chlorhexidine for CANV. For *Ophidiomyces*: 3% bleach for at least 2 minutes, 70% ethanol, 0.16% Roccal®-D for 10 minutes, Lysol® products, CLR® bath and kitchen cleaner (5-10% lactic acid), Process NPD® or Formula 409® household cleaners. Chlorhexidine, Simple Green® and spectracide are NOT effective for *Ophidiomyces*. Ten minutes of contact time is recommended for most cleaners to reach optimal spore removal.

Notification: None required

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Measures required under the Animal Disease Surveillance Plan: None required
Measures required for introducing animals to infected animal: It is not recommended to introduce non-infected animals to infected animals until confirmation that infection is completely cleared based on culture of biopsy of the originally affected areas.
Conditions for restoring disease-free status after an outbreak: It must be assured no residual carrier animals in remaining group of animals.
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CAPRINE ARTHRITIS-ENCEPHALITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Goats	<p>Vertical</p> <ul style="list-style-type: none"> - Infected colostrum or milk - Possibly <i>in utero</i> or during parturition <p>Horizontal</p> <ul style="list-style-type: none"> - Aerosolization - Unsanitary milking practices - Possible venereal transmission 	<ul style="list-style-type: none"> -Progressive lameness -Neurologic signs -Interstitial pneumonia -Mastitis -Chronic weight loss <p>(animals may have one or more forms of the disease)</p>	<ul style="list-style-type: none"> -Asymptomatic carrier state to chronic debilitating arthritis -Rapidly progressing neurologic disease 	<ul style="list-style-type: none"> -Supportive care -Analgesics -Antibiotics for 2^o infections 	<ul style="list-style-type: none"> -Quarantine or cull infected animals -Serologic testing of herd every 6 months beginning at 6 months of age 	No

Fact Sheet compiled by: Andrea Goodnight
Sheet completed on: 15 April 2011; updated 21 July 2013
Fact Sheet Reviewed by: Kimberly Rainwater, Eric Klaphake
Susceptible animal groups: Domestic goats and more common in dairy goat breeds. Domestic sheep may be infected, and non-clinical but possibly carriers.
Causative organism: Caprine arthritis-encephalitis virus (CAEV) is a small ruminant Lentivirus in the family Retroviridae that is related closely to OPP and Maedi-Visna viruses of sheep, and diagnostically difficult to differentiate.
Zoonotic potential: None
Distribution: Worldwide; more prevalent in herds with animals imported from long-established dairy herds. US, Canada, Europe – up to 80% seroprevalence (especially in long-established dairy herds); Southern Africa – “relatively free” of CAE.
Incubation period: Seroconversion occurs in 2-8 weeks, but disease may be clinically latent for years. Once an animal is infected, it remains infected for life.
<p>Clinical signs: Five syndromes:</p> <p><u>Arthritis</u> – Chronic, goats > 6 mo of age; progressive lameness. Swelling of carpal joints most common and preferentially may affect hocks, stifles, hips, and atlantooccipital joints. Radiographs show soft tissue swelling and periarticular calcification.</p> <p><u>Leukomyeloencephalitis</u> – Typically kids 1-4 mo of age, but may be seen in adults; ataxia progressing to tetraparesis; blindness, head tilt, facial paralysis, opisthotonos may occur. Clinical course 1-2 weeks. Very poor prognosis for recovery.</p> <p><u>Interstitial pneumonia</u> – chronic, more common in adults</p> <p><u>Mastitis</u> – interstitial (“hard udder”), hypogalactia or agalactia around parturition in young does</p> <p><u>Chronic wasting</u> – poor body condition, rough hair coat</p>
Post mortem, gross, or histologic findings:

CAPRINE ARTHRITIS-ENCEPHALITIS

Arthritis – thickened joint capsule, periarticular mineralization; chronic proliferative synovitis with subsynovial mononuclear infiltrates.

Leukomyeloencephalitis – increased protein concentration in CSF with mononuclear pleocytosis. Asymmetrical foci of discoloration in the brain and/or spinal cord. Widespread perivascular infiltration by mononuclear cells. Coagulative necrosis and demyelination of white matter.

Interstitial pneumonia – nodular lymphoid aggregates, proliferation of smooth muscle, massive infiltration of the alveolar walls by lymphoid cells

Mastitis – Inflammatory cell foci within interstitium. Extensive nodular lymphoid proliferation can be observed around the alveolar ducts. In chronic cases, inflammatory cells and connective tissue replace the normal parenchyma.

Diagnosis: Clinical signs: CAEV history in herd: Serology (ELISA or AGID); PCR; synovial fluid analysis – red/brown color with low viscosity; increased cell count, with the majority mononuclear cells (lymphocytes); synovial biopsy for histopathology. Positive test results in kids <90 days old usually reflect colostral antibody transfer. However, negative test results do not reliably rule out CAE virus infection, because the time for post-infection seroconversion is variable and occasional goats have a very low titer that may not be detectable. Low antibody titers are common in late pregnancy.

Material required for laboratory analysis: ship samples cool, on ice; serum (2 ml); whole blood in EDTA (5 mL)

Relevant diagnostic laboratories:

National Veterinary Services Laboratories (NVSL) – ELISA and AGID
1920 Dayton Avenue, Ames, Iowa, 50010, USA

Tel: (515) 337-7266

Email: NVSL_Concerns@aphis.usda.gov

Website: http://www.aphis.usda.gov/animal_health/lab_info_services/diagnos_tests.shtml

Washington Animal Disease Diagnostic Laboratory (WADDL) – cELISA

Bustad Hall Room 155N, Pullman, Washington, 99164, USA

Tel: (509) 335-9696

Email: waddl@vetmed.wsu.edu

Website: http://www.vetmed.wsu.edu/depts_waddl/index.aspx

Colorado State University Veterinary Diagnostic Laboratory – PCR, AGID, cELISA

200 West Lake Street, 1644 Campus Delivery, Fort Collins, Colorado, 80526, USA

Tel: (970) 297-0320

Email: dlab@colostate.edu

Website: <http://www.dlab.colostate.edu/webdocs/services/index.htm>

Treatment: Supportive care with analgesics (NSAIDs); physical therapy; antibiotics and antifungas for secondary infections. Antiviral medications may lessen severity and slow progression of disease but are not routinely used.

Frequent proper foot trimming, soft bedding, good pasture management

Prevention and control: Quarantine or cull affected and seropositive animals. Remove kids from affected dams immediately after parturition and feed heat-treated (56°C) colostrum and feed kids pasteurized goat's milk, milk from CAEV-negative goats, or milk replacer. Caesarean section may help prevent vertical transmission. Chemical disinfection of equipment. Serologic testing of herd recommended every 6 mo, beginning with kids at 6 mo of age.

CAPRINE ARTHRITIS-ENCEPHALITIS

Suggested disinfectant for housing facilities: Phenolic and quaternary ammonium compounds
Notification: Reportable for disease monitoring to the World Organisation for Animal Health (OIE), USDA APHIS, and many state veterinarians.
Measures required under the Animal Disease Surveillance Plan: None required
Measures required for introducing animals to infected animal: Not recommended
Conditions for restoring disease-free status after an outbreak: No seropositive animals remaining in herd after two successive testing periods. Testing performed twice yearly. Hand raise newborn kids on colostrum/milk from unadulterated source.
Experts who may be consulted: Dr. James Evermann Washington Animal Disease Diagnostic Lab Pullman, Washington 99164 USA Tel: (509) 335-3044 Email: jfe@vetmed.wsu.edu Dr. Donald P. Knowles Animal Diseases Research Unit (USDA/ARS) College of Veterinary Medicine, Washington State University 3003 ABBF Pullman, Washington 99164 USA Tel: (509) 335-6001 Email: dknowles@vetmed.wsu.edu Dr. Stephen Valas Laboratoire de Niort, Laboratoire d'étude et de recherches caprines 60 rue du Pied de Fond, B.P. 3081, 79000 Niort, FRANCE Tel: 33 (0)5 49.79.61.28 Fax: 33 (0)5 49.79.42.19 Email: stephen.valas@anses.fr
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ENZOOTIC ABORTION OF EWES/OVINE ENZOOTIC ABORTION
(Chlamydophila abortus)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Sheep and goat; less commonly cattle, pigs, horses and deer.	Oral transmission. Organism shed in aborted fetuses, placenta, vaginal secretions (during estrus and up to 9 days prior to and weeks to months post abortion) and in infected semen. Birds, i.e. pigeons and sparrows, may be reservoirs.	Can see nonspecific malaise in pregnant animals. Late term abortions, stillbirths, and birth of weak offspring.	Can see high rate of abortion, >30%, in naïve flock or yearly rates up to 5% in enzootic form. Abortion storms can be seen in intensively managed flocks.	Tetracycline or oxytetracycline. Supportive care for complications of infection such as retained placenta, metritis, pneumonia or keratoconjunctivitis.	Remove infected or contaminated materials. Keep feed sources free of fecal material. Separate first lambing ewes from rest of flock. Animals that abort develop natural immunity (~3 year duration). Vaccination.	Yes

Fact Sheet compiled by: Denise McAloose

Sheet completed on: 13 January 2011; updated 26 March 2013

Fact Sheet Reviewed by: Bonnie Raphael, Carlos Rodriguez

Susceptible animal groups: Sheep, goat

Causative organism: *Chlamydophila abortus* (previously *Chlamydia psittaci* serotype 1) is the causative Gram negative intracellular bacterium and has two genera and 9 species. Antigenic strains in sheep and goat appear to be related. Antigenic type 1 is implicated in abortion, stillbirth and the birth of weak offspring.

Zoonotic potential: Yes, and it can cause serious infection in pregnant women and lead to miscarriage. Pregnant women are discouraged from having contact with the flock during lambing/kidding season. In non-pregnant humans, infection can produce flu-like symptoms.

Distribution: World-wide distribution

Incubation period: Infection occurs through ingestion. Organisms colonize the intestinal tract, invade the bloodstream and subsequently infect the placenta and developing fetus. Incubation can be as short as 2 weeks, although typically proliferation of the organism occurs at about day 90 of gestation. Infection is latent in lambs and non-pregnant ewes and becomes activated at conception.

Clinical signs: Non-specific malaise in dam may be seen. Abortion, stillbirth or birth of weak offspring does occur. Final trimester abortions occur in ewes infected at 5-6 weeks gestation; abortion in the subsequent pregnancy occurs in ewes that were infected after this time. Abortion can occur at any time during gestation for goats. In both species, retained placenta can occur. The infection in rams may cause orchitis.

Post mortem, gross, or histologic findings:

Gross: Placental tissues contain multifocal to coalescing areas of red discoloration and edema; tissues can have a leathery appearance. Changes are typically diffuse but more significant changes may be noted in the

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cotyledonary than intercotyledonary areas. The aborted fetus is often well preserved although can be autolyzed; it may have multifocal areas of hemorrhage in muscle, lymphoid tissues and the subcutis and/or pinpoint yellow areas of discoloration on the surface of the liver.

Histology: Placental changes are characterized by fibrinoid vascular necrosis, thrombosis, and severe neutrophilic placentitis with superficial necrosis of the chorion. Trophoblasts often contain numerous intracytoplasmic organisms that distend the cells, are difficult to see with routine hematoxylin/eosin staining, and are positive with special staining using a modified Ziehl-Neelsen, Gimenez or Giemsa stain. Changes in the aborted fetus are few and characterized by foci of coagulative necrosis in the liver and spleen that may be associated with peripheral mononuclear cell inflammation. Mild subacute inflammation can also be seen in the lungs and mild meningoencephalitis has also been reported.

Diagnosis: History of abortion provides suspicion to perform testing.

Serology: Complement fixation tests can present some cross reactivity and doesn't distinguish between vaccination and natural infection; so should be paired at 2-3 weeks apart. High and rising titers in ewes and fetal serum antibodies aid in diagnosis of disease

Tissue sections: Histology; electron microscopy

Special staining: Positive staining of organisms with modified Ziehl-Neelsen, Gimenez or Giemsa in cytologic preparations or placentitis (confirmed histologically) with intralesional/intracellular positive organisms; alternatively can try to id organism on cytology of vaginal swab.

Immunologic tests: ELISA, IHC, FA

Definitive diagnosis: PCR and real-time PCR, PCR microarray hybridization, indirect inclusion fluorescent antibody test, immunohistochemical staining, tissue culture or egg inoculation

Material required for laboratory analysis:

Placenta (preferred) or fetus: Fresh tissue for cytologic preps; 10% neutral buffered formalin fixed paraffin embedded (FFPE) tissue for histology or immunohistochemical staining; fresh or FFPE for PCR; contact laboratory for tissue storage/fixation for fluorescent antibody test;

Vaginal swab: For cytology or culture

Serum: *C. abortus* antibodies are confirmatory in the fetus; paired titers used diagnostically in adults

Relevant diagnostic laboratories:

Any laboratory capable of bacteriologic culturing is capable of diagnosing *C. abortus*.

National Veterinary Services Laboratories (NVSL)

P.O. Box 844

1920 Dayton Ave

Ames, IA 50010

515-337-7514

Treatment: *C. abortus* is sensitive to tetracyclines although sensitivity testing on cultured organism may aid treatment strategy. In face of outbreak, recommendations include treating all pregnant females during final 4-6 weeks of gestation. For disease prevention, two week treatment with tetracycline in feed (400 to 500 mg/head/day) in fiber-producing animals or injection of long-acting oxytetracycline (20 mg/kg IM or SC) every 10-14 days or twice a week treatment in the last 4-6 weeks gestation in dairy herds has been reported; alternately, can treat with one injection of long-acting oxytetracycline 6 to 8 weeks prior to parturition and 3 weeks post parturition.

Prevention and control: As high numbers of organisms are shed in aborted or stillborn fetuses and in infected placental tissues or uterine discharge; and organisms remain viable for several days or longer in cold or freezing

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temperatures, isolate aborting dams and separate first lambing ewes from rest of flock. Animals that abort develop natural immunity of ~3 year duration.

Infected or contaminated materials should be removed and feed sources kept free of fecal material. Pest control should be practiced as transmission can occur via rodent and birds.

Vaccination: Live and inactivated vaccines are available for use in areas where vaccination is permitted; vaccination reportedly can prevent abortion and reduce excretion; can assist in control but will not eradicate it.

From Terrestrial Animal Health Code (<http://www.oie> Chapter 14.5):

Prevention: For importation for breeding: International veterinary certificate ensuring 1. animal has been housed for previous two years or since birth in facility with no EAE positive tests for previous two years 2. no clinical signs of EAE on day of shipment 3. was test negative for EAE within 30 days of shipment. **For importation of semen:** International veterinary certificate ensuring donor animals 1. are from facilities that have been EAE test negative for previous two years and have not been in contact with animals of lower health status and were test negative for EAE for 2-3 weeks post semen collection and 2. an aliquot of the semen for export was culture negative for *C. abortus*.

Control: Separate first lambing ewes from rest of flock. Segregate aborting animals from herd for minimum of 3 weeks, burn or bury aborted materials, disinfect the area. Prevent contamination of food and water. Control can also include culling of live kids born to infected dams. Ewes that abort develop natural immunity to infection after the first abortion (waned after ~ 3 years). Vaccine is available and licensed in some countries. One recommendation is for IM or SC vaccination 8 weeks prior to breeding and once again 4 weeks later; though immunity is thought to be protective for 3 years, annual boosters prior to breeding season are suggested. Animals that abort develop natural immunity (~3 year duration). Note: Immune or vaccinated animals can shed organism.

Suggested disinfectant for housing facilities: Susceptible to disinfection with quaternary ammoniums.

Notification: Reportable to State and Federal agencies in United States and to OIE.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak:

The following are requirements of the Terrestrial Animal Health Code (<http://www.oie>; Chapter 14):

1. Sheep flock or goat herd is under official veterinary surveillance.
2. No sheep or goats have shown clinical evidence of infection for past 2 years.
3. A statistically appropriate number of sheep, goats > 6 months of age were test negative for EAE within past 6 months.
4. All sheep, goats are permanently identified.
5. No sheep, goat additions since 30 days prior to test in #3 unless
 - EITHER the additions were isolated from other animals in flock/herd in the flock/herd of origin for a minimum of 30 days and then were test negative for EAE prior to entry in the new flock/herd.
 - OR the animal originated from a flock/herd of equal health status.

Experts who may be consulted:

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services, Emergency Management
4700 River Road, Unit 41

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(*Chlamydophila abortus*)

Riverdale, MD 20737-1231

Phone: 301-734-8073

Fax: 301-734-7817

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AVIAN CHLAMYDIOSIS/CHLAMYDOPHILOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Birds, humans, other mammals uncommon	Inhalation of aerosolized fecal matter and nasal discharges primarily; also oral secretions and feathers Fomites Mechanical transmission may occur – rodents and insects Vertical transmission is infrequent	Non-specific, oculo-nasal discharge, respiratory signs, conjunctivitis, diarrhea, weight loss, anorexia, depression, green to yellow green urates Some birds may have subclinical infections	Morbidity and mortality rates vary with the affected species, condition of infected individual, and strain/genotype involved.	For most avian species: Doxycycline for 30-60 days. See treatment section for details	Screen and quarantine new birds. Good hygiene practices. PCR testing for antigen testing. See Diagnosis section for details on other testing.	Yes

Fact Sheet compiled by: Danelle M. Okeson

Sheet completed on: 22 July 2010; updated 8 October 2012

Fact Sheet Reviewed by: Thomas N. Tully, Jr.; Robert D. Dahlhausen

Susceptible animal groups: Birds – reported in more than 30 orders of birds; but more common in Psittaciformes and Columbiformes (doves and pigeons). It is sometimes seen in ducks and turkeys but rarely in chickens. Some wild bird species may act as reservoirs, and egrets and gulls can be subclinical carriers for strains that are highly virulent for other birds.

Humans and less commonly other mammals such as occasionally in dogs, cats, horses, cattle, sheep, and muskrats.

Causative organism: *Chlamydophila (Chlamydia) psittaci*

Zoonotic potential: Yes

Distribution: World wide

Incubation period: Birds – 3 days to several weeks. Some birds may remain subclinical until stressed. Some birds may shed the infectious organism 10 days before clinical signs are observed.

Clinical signs:

Birds – lethargy, ruffled feathers, anorexia, oculonasal discharge, conjunctivitis, diarrhea, weight loss. Some birds may manifest respiratory signs ranging from sneezing to respiratory distress. Neurologic signs such as tremors, torticollis, or leg paresis may be found in subacute to chronic cases. Infected carriers may not have overt clinical signs.

Humans – fever, chills, myalgia, malaise, nonproductive cough sometimes with chest tightness and/or breathing difficulty; sometimes a nonspecific rash and enlarged spleen are also present.

Other mammals - linked to abortions in horses, cattle, and sheep; variety of clinical disease presentations in dogs including respiratory and reproductive signs.

AVIAN CHLAMYDIOSIS/CHLAMYDOPHILOSIS

Post mortem, gross, or histologic findings: Nasal inflammation, pneumonia, fibrinous air sacculitis, hepatomegaly with multifocal hepatic necrosis, splenomegaly, pericarditis. Infected birds exhibiting no signs of illness often have no gross lesions

Diagnosis: Based on clinical signs, a combination of antigen and antibody tests, and clinical tests including hemogram, chemistry panel, and radiographs. The Compendium of Measures to Control *C. psittaci* lists case definitions for suspect, probable, and confirmed cases. PCR testing may be performed on whole blood samples and/or swabs of the choana or swabs of both the choana and cloaca. Sensitivity is improved if both blood samples and swabs are tested.

Serology is available but results must be interpreted appropriately. A positive serologic test result is evidence that the bird may have been exposed to *C. psittaci* in the past, but does not prove the bird is currently infected. Conversely, a negative serologic test result is not proof that the bird is free of infection. The Direct Complement Fixation test has been historically the most commonly used serology assay.

Material required for laboratory analysis: Varies with type of testing, see Diagnosis section.

Relevant diagnostic laboratories: Several state/university and private labs offer testing, a few are listed below; see individual labs for types of tests offered.

Diagnostic Center for Population and Animal Health, Michigan State University www.dcpah.msu.edu

Comparative Pathology Laboratory, University of Miami, Miami, Florida www.pathology.med.miami.edu

Infectious Diseases Laboratory, University of Georgia College of Veterinary Medicine

www.vet.uga.edu/sams/idl

Texas Veterinary Medical Diagnostic Laboratory, College Station, Texas

<http://tvmdlweb.tamu.edu>

Veterinary Molecular Diagnostics, Inc., Milford, Ohio

<http://www.vmdlabs.com/>

Treatment: Recommended treatment period for most avian species has traditionally been 45 days with doxycycline, however some birds may require treatment for as long as 60 days. Birds should be re-checked by swab and blood PCR, two weeks after cessation of treatment. Treatment protocols of 30 days length can be effective in budgerigars. However, "...no single protocol ensures safe treatment or complete elimination of infection in every bird. Therefore, treatment for avian chlamydiosis should be supervised by a licensed veterinarian after consultation with an experienced avian veterinarian." (Compendium of Measures to Control *C. psittaci*)

Prevention and control: Quarantine for at least 30 days and test birds* entering the existing bird collection; avoid obtaining birds from multiple sources; quarantine any sick birds; screen birds* with frequent public contact; practice preventative husbandry/good hygiene.

*No test or combination of tests can declare a bird "disease free" of *C. psittaci*. Subclinical carriers exist. Intermittent shedding of the organism may also complicate testing. Further details outlined in the Compendium of Measures to Control *C. psittaci* – see references, updated yearly and available online.

Suggested disinfectant for housing facilities: The organism is susceptible to many detergents and disinfectants as well as heat. However, it is resistant to acid and alkali. Effective disinfectants include 1% Lysol, a 1:1,000 dilution of quaternary ammonium compounds (e.g., Roccal, Zephiran), or freshly prepared 1:32 dilution of household bleach (½ cup/gallon).

Notification: Reportable disease under USDA-APHIS-VS National Animal Health Reporting System. Psittacosis in humans is a Nationally Notifiable Disease – and most states require physicians to report to appropriate local or state health authorities.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Infected birds should be isolated and treated. Other birds should not be introduced until treatment is completed and the infected bird's facility is

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thoroughly cleaned and disinfected.

Conditions for restoring disease-free status after an outbreak: Infected pet birds or other valuable birds and their contacts may be isolated and treated. Poultry may be euthanized rather than treated, often due to economic constraints. Premises should be thoroughly cleaned and disinfected. New birds entering a facility can be tested for the disease but cannot truly be declared “disease-free”.

Experts who may be consulted: Consult the list of contributors to the Compendium of Measures to Control *Chlamydophila psittaci* (www.nasphv.org/Documents/Psittacosis.pdf).

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CHRONIC WASTING DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Natural infection: mule deer (<i>Odocoileus hemionus</i>), white-tailed deer (<i>Odocoileus virginianus</i>), Rocky mountain elk (<i>Cervus elaphus nelsoni</i>), and moose (<i>Alces alces</i>)	Direct animal to animal contact and contact with contaminated environment. Agent shed in feces, saliva, urine, perhaps milk. Contagious among susceptible species but mechanism unclear.	Early: subtle and non-specific. Progressive emaciation, abnormal behavior, excessive salivation, and ending in mortality.	Progressive and fatal	None	Early detection and removal of infected individuals. Depopulation of infected captive herds unless regular ante-mortem testing of remaining animals is possible. Regulation of international and interstate movements of cervids and cervid tissues.	No evidence of transmission to humans under natural conditions. Human health advisory to avoid consumption of food derived from any animal with evidence of CWD or containing potentially infectious cervid tissues.

Fact Sheet compiled by: Roy Burns

Sheet completed on: 22 March 2011; updated 23 August 2013

Fact Sheet Reviewed by: Terry Spraker; Mark Drew; Bryan J. Richards

Susceptible animal groups: Natural infection: mule deer (*Odocoileus hemionus*), white-tailed deer (*Odocoileus virginianus*), Rocky mountain elk (*Cervus elaphus nelsoni*), red deer (*Cervus elaphus elaphus*), moose (*Alces alces*), and sika deer (*Cervus nippon*). Experimental infection (intracerebral inoculation; variable transmission success across experimentally susceptible species): muntjac (*Muntiacus* sp.), domestic cattle (*Bos taurus*), sheep (*Ovis aries*), fallow deer (*Dama dama*), domestic ferrets (*Mustela putorius furo*), mink (*Mustela vison*), hamsters, mice, squirrel monkeys (*Saimiri sciureus*), and voles of the genera *Mictotus* and *Myodes*.

Causative organism: A transmissible spongiform encephalopathy produced by a prion (protein infectious agent). Two prevalent strains with divergent biochemical characteristics have been identified and strain variation may occur.

Zoonotic potential: To date, no strong evidence of CWD transmission to humans has been reported. Several epidemiologic studies provide evidence that, to date, CWD has not been transmitted to humans. Specific studies have focused on identifying human prion disease in hunter population presumed at increased risk for exposure to potentially CWD-infected deer or elk meat. In the last 60 or so years in regions where CWD is present and such exposure is known, no evidence of an increase in any neurodegenerative disease condition of humans has been identified. CWD prions have been found in muscle (meat), as well as other tissues of cervids, and could enter the food supply. Although the evidence so far suggests that CWD probably does not affect humans, the possibility that it could be zoonotic has not been eliminated although may be increasing over time.

Distribution: Maps of the current distribution of CWD in captive and free roaming herds suggest gradual

CHRONIC WASTING DISEASE

spread has occurred from original identification in Colorado:

http://www.nwhc.usgs.gov/disease_information/chronic_wasting_disease/. As of this update:

- Wild herds and captive facilities infected: Colorado, Kansas, Minnesota, Missouri, Nebraska, New York, Pennsylvania, South Dakota, Wisconsin, Alberta, Saskatchewan.
- Wild herds only infected: Illinois, Maryland, North Dakota, New Mexico, Texas, Utah, Virginia, West Virginia, Wyoming.
- Captive facilities only infected: Iowa, Michigan, Montana, Oklahoma, South Korea.

Incubation period: Infected deer and elk can appear robust and healthy in the early stages of CWD and may take two or more years before they show clinical signs of the disease. Minimum incubation time in experimental infection: 15 mo but can exceed 25 months in deer and 60 months in elk. Genetic polymorphisms influence the incubation time in both deer species and in elk (and probably in moose).

Direct animal-animal contact and contact via ingestion and inhalation of contaminated material from the environment is the primary route of transmission. Agent shed in feces, saliva, urine; other mechanisms (e.g., nasal secretions, milk) also are possible. Contaminated environments (soil, feces, offal, carcass) may be infective for years.

Clinical signs: Early clinical signs are subtle and nonspecific: behavior changes (response to handling, interaction with conspecifics, somnolence, periods of repetitive behavior, vacant facial expression). During period few weeks to several months prior to death, emaciation, abnormal behavior, progressive weight loss, stumbling, tremors, lack of coordination, blank facial expressions, excessive salivation, loss of appetite, excessive thirst and urination, listlessness, teeth grinding, abnormal head posture, and drooping ears are observed. Later, esophageal dilation, difficulty in swallowing, resulting in pneumonia caused by aspiration of food or saliva into the lungs. Disease is progressive and always fatal.

Post mortem, gross, or histologic findings: At necropsy, post-mortem findings of emaciation and pneumonia is are found. In suspected clinical cases, histologic spongiform change with degeneration and loss of neurons and identification of PRP (cwd) using IHC staining of the obex. For preclinical diagnosis, IHC on lymph nodes (tonsils, retropharyngeal lymph nodes) or mucosa-associated lymphoid tissue.

Diagnosis:

Ante-mortem: IHC staining for PrP (cwd) of biopsied lymphoid tissue (tonsil, rectal mucosa).

Post-mortem: Detection of PrP (cwd) via IHC in brain obex (specifically parasympathetic vagal nucleus in the dorsal portion of the medulla oblongata at the obex) (all species, later stages of disease). For detecting preclinical infections, IHC on lymph nodes (tonsils, retropharyngeal lymph nodes). While ELISA, Western Blotting, and PMCA testing is available, IHC is the only assay currently accepted officially by USDA.

Material required for laboratory analysis: Cervid post mortem: brain (obex) and lymph tissues (preferably tonsils, medial retropharyngeal lymph nodes).

Relevant diagnostic laboratories:

USDA-APHIS-VS-NVSL

1920 Dayton Ave. (for packages)

P.O. Box 844 (for letters)

Ames, IA 50010

NVSL (IHC for PRP Brain (obex), and medial retropharyngeal lymph node)

(515) 337-7266

Fax: (515) 337-7397

USDA-NAHLN (IHC and ELISA)

(515) 337-7731

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Fax: (515) 337-7397

http://www.aphis.usda.gov/animal_health/animal_diseases/cwd/diagnostics.shtml

Colorado State University Diagnostic Laboratory
College of Veterinary Medicine
300 West Drake Road
Colorado State University
Fort Collins, Colorado 80526

Treatment: While no therapeutic cures, supportive therapy may prolong course, but it is not advisable in the context of control.

Prevention and control:

Free Ranging: Testing hunter harvested and road kill cervids; local population reduction in infected areas; bans on feeding wild cervids; restrictions on transporting hunter-killed carcasses from enzootic regions; and transportation restrictions and monitoring of captive cervid operations will reduce transmission. Cooking or heat does not inactivate prions. Educational efforts is critical as management once disease has entered area has been very ineffective so prevention is paramount.

Captive: Routine surveillance and testing of cervids (all species) held in zoo collections. Early detection and removal of infected individuals. Depopulation of infected captive herds is recommended unless regular antemortem testing of remaining animals is possible and strict biocontainment is followed. Regulation of international and interstate movements of cervids and restricted importation of captive cervids. Consider antemortem testing of animals prior to movements. Prevent entrance of free-ranging cervids from zoo grounds. Post mortem sampling of free-ranging and captive cervids that die or are euthanized on zoo grounds. Although not shown to be food borne, rendered ruminant meat or bone meal should not be fed to cervids. No effective vaccine at present.

Suggested disinfectant for housing facilities: Environmental contamination is a major concern in eradication and prevention in new cases. Prions are extremely resistant to heat, pH, ultraviolet, and disinfectants. Sodium hypochlorite (household bleach, greater than 2% free chlorine) at 280 ml in 720 ml of water at room temperature, for one hour. Sodium hydroxide (caustic soda, soda lye) at 38g in one litre of water at room temperature for one hour.

Notification: Responsible state agency should be contacted. State and Federal regulations are dynamic and responsive to disease status. Some states have adopted regulations limiting or prohibiting whole carcass transportation or particular cervid tissue transportation out of CWD areas.

Measures required under the Animal Disease Surveillance Plan: USDA-APHIS established a national voluntary CWD Herd Certification program published in the Federal Register on 13 June 2012. Many states already have CWD monitoring or certification programs for captive herds. Guidelines for Chronic Wasting Disease (CWD) Surveillance in captive cervids in zoos AAZV/AZA Animal Health Committee, 2003.

Measures required for introducing animals to infected animal: See measures required under the Animal Disease Surveillance Plan.

Conditions for restoring disease-free status after an outbreak: See measures required under the Animal Disease Surveillance Plan.

Experts who may be consulted: Numerous experts are identified at <http://www.cwd-info.org>.

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August 2013.

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http://www.aphis.usda.gov/animal_health/lab_info_services/approved_labs.shtml Accessed 23 August 2013.

http://www.aphis.usda.gov/animal_health/animal_diseases/cwd/farmed.shtml Accessed 23 August 2013.

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CHYTRIDIOMYCOSIS DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Amphibians	Contact with contaminated water, moist or wet substrates, or infected animals. Crayfish may act as carriers.	Erythematous skin, excessive skin shedding, abnormal behavior, sudden death	Outcome of infection ranges from subclinical to fatal	Itraconazole or chloramphenicol baths, elevated temperatures	Isolate affected amphibians	No

Fact Sheet compiled by: Cynthia Stadler

Sheet completed on: updated 12 January 2019

Fact Sheet Reviewed by: Kathryn Tuxbury

Susceptible animal groups: Amphibians

Causative organism: *Batrachochytrium dendrobatidis* (Bd), a non-hyphal zoosporic fungus.

Zoonotic potential: No

Distribution: World-wide wherever amphibian populations are present. Chytridiomycosis has been implicated as the cause of massive amphibian population declines.

Incubation period: 14-70 days

Clinical signs: Erythematous or discolored skin, abnormal posture, neurologic signs, excessive skin shedding, behavior changes. Clinical signs may not be apparent prior to acute death.

Post mortem, gross, or histologic findings: Gross lesions are often not present but may include increased sloughing of the skin, discolored skin, erosions. Histologic lesions involve focal hyperkeratosis and epidermal hyperplasia with sloughing of the keratin layer. Fungal zoosporangia are found within the keratin layers. The fungal lesions are not evenly distributed on the skin surface. Predilection is noted for the digits, ventral aspect of the hind limbs, inguinal and pelvic regions, and in tadpoles, mouth parts.

Diagnosis: PCR (skin swab best), histopathology. Cytology requires experience. PCR is best for detecting subclinical infection, whereas histopathology and cytology are most useful for clinically significant infection.

Material required for laboratory analysis: Shed skin, skin scraping, skin swab or skin sample (preferably from the ventral pelvic patch) from adults. Mouthpart swabs from live tadpoles, mouthparts from deceased tadpoles. Using fine-tipped swabs (not wooden-handled), gently swab skin 20-30 times. Break swab 2-3 cm from tip and place in screw-top tube, avoiding contact with outside of tube. Allow to air-dry for 5 minutes. Samples can be kept at room temperature or 4 degrees C for 1- 2 weeks or frozen for longer-term storage. Avoid exposure to high temperatures and direct sunlight.

Relevant diagnostic laboratories:

For histopathology, any laboratory that routinely examines amphibian tissues.

For PCR:

Amphibian Disease Laboratory
15600 San Pasqual Valley Road
Escondido, CA 92027

(760) 291-5472 (760) 291-5470

amphibianlab@sandiegozoo.org https://www.sandiegozooglobal.org/News/Amphibian_Disease_Laboratory/

Pisces Molecular

1600 Range Street, Suite 201

Boulder, CO 80301

303-546-9300 info@pisces-molecular.com <http://pisces-molecular.com>

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Research Associates Laboratory
14556 Midway Road
Dallas, TX 75244

Phone: (972) 960-2221 Fax: (972) 960-1997 <http://vetdna.com>

Zoologix Laboratories
9811 Owensmouth Avenue, Ste. 4
Chatsworth, CA 91311-3800
Phone: 818-717-8880 Fax: 818-717-8881
info@zoologix.com <http://zoologix.com>

Treatment: Itraconazole 0.005% (50 mg/liter) diluted with 0.6% saline or amphibian Ringer's solution used as a 5 minute bath applied once daily for 6-10 days. Lower concentration of 0.0025% (25 mg/liter) also has been successful at eliminating the organism. Efficacious treatments can vary among species and life stages. Hygiene is essential during treatment and animals should be returned to a clean disinfected container after EACH treatment. Previously recommended higher concentration of 0.01% itraconazole is toxic to tadpoles and recently metamorphosed amphibians. Other treatments include chloramphenicol baths and elevated environmental temperatures of 37°C for 16 hrs, in those species that are thermo-tolerant. Also, terbinafine baths and topical voriconazole have been used with variable results to date. Animals with clinical chytridiomycosis may have issues from hyponatremia and hypokalemia so electrolyte replacement may be helpful.

Prevention and control: Newly acquired amphibians should undergo a minimum of 30 days in quarantine, preferably 60 days. Skin swab PCR testing or prophylactic itraconazole baths should be implemented prior to release from quarantine. All animals that die in quarantine should be necropsied and submitted for histopathology. Enclosures and equipment should be disinfected routinely. However, it is prudent to wear disposable gloves and use separate equipment for different enclosures.

Suggested disinfectant for housing facilities: Bleach, Virkon and quaternary ammonium compounds can be used for enclosures. For surgical instruments, 70% ethanol, glutaraldehyde, and benzalkonium chloride can be used.

Notification: Office International des Epizooties (OIE) notifiable disease.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Introductions are not recommended until numerous negative PCR tests have been completed.

Conditions for restoring disease-free status after an outbreak: It is recommended to test amphibians by PCR to confirm the fungus is no longer present. For disease-free status, there should be serial negative PCR tests over the course of 6 months to 1 year.

Experts who may be consulted:

Allan Pessier, DVM, Dipl. ACVP
Washington Animal Disease Diagnostic Laboratory
Department of Veterinary Microbiology and Pathology
College of Veterinary Medicine
Washington State University
Pullman WA 99164-7034
apessier@wsu.edu

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CLASSICAL SWINE FEVER (hog cholera)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Swine	Direct contact with body secretions, feeding uncooked infected pork products, mechanical vectors (flies, vehicles, people), <i>in utero</i> .	Acute: sudden death, ataxia, cutaneous cyanosis or hyperemia, petechiation, necrosis of ear tips. Chronic: failure to thrive, dermatitis. Congenital: fetal mummification, cerebellar hypoplasia, congenital tremors.	Highly contagious. Can range from mild disease in chronic infections to severe disease and sudden death in acute infections.	None	Prevention: Vaccination utilized in some countries, control pig movements, serosurveys, do not feed uncooked pork. Control: test, slaughter, quarantine, disinfect.	No

Fact Sheet compiled by: Cora Singleton

Sheet completed on: 1 March 2011; updated 31 October 2012; updated 8 August 2018.

Fact Sheet Reviewed by: Sarrah Kaye

Susceptible animal groups: Domestic and wild swine; endemic in wild boar in parts of Europe.

Causative organism: Classical swine fever virus (CSFV) is an RNA virus in the genus *Pestivirus* within the family *Flaviviridae*. Strains can range from low to high virulence. Related to bovine viral diarrhea virus and border disease virus of sheep. Also known as hog cholera.

Zoonotic potential: No

Distribution: CSFV is present in South-East Asia, the Caribbean, Africa, South and Central America, and parts of eastern Europe. Areas considered free of CSF in domestic pigs include North America (US, Canada, and Mexico), some countries in South America, New Zealand, Australia, Asia, and many countries in western and central Europe.

Incubation period: 2-19 days

Clinical signs: Acute disease (high virulence strains) – Sudden death, depression, pyrexia, anorexia, ataxia, constipation followed by diarrhea and vomiting, ocular discharge, cutaneous cyanosis, necrosis of ear tips, muscle tremors, convulsions.
Chronic disease (low virulence strains) – Dullness, anorexia, failure to thrive, diarrhea, dermatitis.
Congenital disease – Stillbirth, fetal mummification, cerebellar hypoplasia, congenital tremors, failure to thrive. Piglets infected with low-virulent strains in 1st trimester can be born viremic and healthy, serve as subclinical shedders with delayed onset of disease. Similar to BVD, it sounds like this is an important source of transmission

Post mortem, gross, or histologic findings: Petechial hemorrhages in kidney, urinary bladder, and larynx; enlarged hemorrhagic lymph nodes; splenic infarcts; encephalitis; button ulcers in cecum (chronic disease); cerebellar hypoplasia (congenital disease).

Diagnosis: Agent identification – Virus culture, fluorescent antibody test, immunoperoxidase procedure, ELISA, RT-PCR.

Serology – Neutralization peroxidase-linked assay, fluorescent antibody virus neutralization, ELISA.

Material required for laboratory analysis: Tissues (tonsil, lymph node, spleen, kidney, distal ileum), blood, serum.

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CLASSICAL SWINE FEVER (hog cholera)

Relevant diagnostic laboratories:

Foreign Animal Disease Diagnostic Laboratory, Plum Island
40550 Route 25 (for packages)
Orient Point, NY 11957
P.O. Box 848 (for letters)
Greenport, NY 11944-0848
(631) 323-3256 Fax: (631) 323-3366

Treatment: No effective treatment.

Prevention and control: Prevention – USDA/APHIS has a surveillance program to prevent reintroduction of the disease. Vaccination is utilized in some countries. Control pig movements and implement serological surveys to detect carrier pigs. Do not feed uncooked pork products.

Control – Depopulation of infected pigs, disinfection of premises, quarantine of the area and control of pig movement.

Suggested disinfectant for housing facilities: 2% sodium hydroxide (considered most suitable), 1% formalin, sodium carbonate, strong iodophors.

Notification: Reportable to the USDA/APHIS through the State Veterinarian or the federal Area Veterinarian in Charge. The disease is also reportable to the World Organization for Animal Health (OIE).

Measures required under the Animal Disease Surveillance Plan: Report suspicious cases to the USDA/APHIS Area Veterinarian in Charge, who will dispatch a Foreign Animal Disease Diagnostician to investigate the case and collect samples for testing.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Infections must be reported to USDA/APHIS for management.

Experts who may be consulted: USDA State Veterinarians or federal Area Veterinarians in Charge.

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American Association of Zoo Veterinarians Infectious Disease Manual
CLOSTRIDIAL DISEASE - BOTULISM TOXICOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, including humans; birds	Ingestion of toxin contaminated food or tissues. Wound contamination	Mostly neurologic, involving flaccid paralysis. Gastrointestinal signs.	Dose-related severity of mild to lethal.	Supportive care; antitoxin when appropriate.	Proper food preparation and storage. Avoid wound contamination	No

Fact Sheet compiled by: Danielle R. Graham Snyder

Sheet completed on: 17 January 2011; updated 18 October 2012

Fact Sheet Reviewed by: Melissa Kennedy; Stephanie Kottler

Susceptible animal groups: Most mammalian and avian species are susceptible to this problem. Wild fowl and mink have a high incidence of clinical disease.

Causative organism: *Clostridium botulinum* bacteria Types A-G: gram positive, slightly curved to straight, motile, spore-forming, saprophytic, anaerobic rod. Type C is the most common in animal species, and Types A, B and E most common in humans.

Zoonotic potential: No

Distribution: Soil, fresh water and sea sediments, the intestinal tracts of mammals and birds, and foods such as home-canned foods, sausages, meat products, canned vegetables and seafood products. These toxin-contaminated sources can be either ingested or contaminate a wound.

Incubation period: Normally 12-36 hrs, but can be as much as a week if a small amount is ingested.

Clinical signs:

Humans: Three types of botulism: food-borne, wound, and infant.

Food-borne botulism is caused by consumption of toxin-tainted food. In these infections, signs can include gastrointestinal issues such as nausea, vomiting and abdominal pain; symmetric, descending flaccid paralysis, and drooping palpebrae; and dry mouth, slurred speech, and muscle weakness. Descending paralysis of the respiratory muscles - potentially fatal, arms and legs may occur within 24 hours in severe cases.

Wound botulism is caused by a wound that is contaminated, usually from toxins in the soil. Signs are consistent with food-borne illness, but usually without gastrointestinal signs.

Infant botulism is seen only in infants less than one year of age and caused by spores germinating in the intestinal tract. Signs include constipation, poor suckling reflexes, peripheral weakness (“floppy baby” syndrome), and in severe cases, respiratory distress and death.

Animals: Clinical signs are mostly neurologic and caused by muscle paralysis. A symmetrical, ascending weakness starting from the rear limbs and progressing to the forelimbs is typical. Cranial nerve deficits are usually present and may include decreased palpebral reflex, decreased gag or swallowing reflex, ptialism, decreased jaw tone, mydriasis, and sluggish pupillary responses. Respiratory or cardiac paralysis can occur and usually causes death.

Post mortem, gross, or histologic findings: Most post mortem or histologic findings are the result of muscle paralysis. Mammals with wound botulism may have lesions, but the wounds are generally not obviously or grossly infected.

Diagnosis:

- History of exposure and clinical signs. The toxin can be hard to find in feed or in tissues, so most

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diagnosis is done by eliminating out other differentials.

- Cultures from wounds or tissues can be taken to potentially isolate the organism. The toxin can also be isolated from serum, feces, vomitus, or samples of food that were ingested.
- ELISA testing can detect neurotoxin, but each subtype of toxin must be evaluated individually.
- Mouse inoculation: Serum or an extract of contaminated material is injected alone and in combination with a type-specific antitoxin into the mice. Survival of the group of mice protected with antitoxin and death of the other group from signs consistent with botulism confirms the presence of botulism toxin. This test is considered the standard and most reliable method of identifying botulism toxin.

Material required for laboratory analysis: Serum, feces, vomitus, stomach or intestinal contents, contaminated food, or culture of tissues if wound botulism is suspected.

Relevant diagnostic laboratories:

National Botulism Reference Laboratory at New Bolton Center (University of Pennsylvania)
National Veterinary Services Laboratories (NVSL) Ames, Iowa

Treatment:

Supportive care is most important. Hospitalization may be necessary. Therapeutic monitoring involves intensive care of recumbent animals. Wounds or abscesses should be cleaned and debrided where possible. Selective padding and respiratory support is essential to avoid complications of recumbency. Antitoxins can be effective to improve survival rates, depending on the toxin involved and the host species. Type C antitoxin seems to work well in some birds and mink. Antibiotics are only used in cases of wound botulism or to treat secondary infections due to the paralysis. Recovery typically takes 14 - 24 days.

Prevention and control: Vaccines are available for humans and animals with high risk of exposure. To ensure food is properly stored and prepared, botulism toxin is destroyed by heating food to 80°C for 30 minutes or to 100°C for 10 minutes. Wounds should be kept clean and avoid contamination as much as possible. For wildlife, prompt removal of carcasses that could be infected is critical as decaying carcasses are known to support toxin production. Maggots feeding on decaying carcasses are sources of infection for many waterfowl as the maggots are unaffected by the toxin, but effectively concentrate it. Waterfowl consume the maggots and become infected. Stagnant water should be avoided as this creates an environment for *Clostridium botulinum* bacteria to grow and for spores to germinate.

Suggested disinfectant for housing facilities: Clean areas with diluted bleach when possible. Sunlight inactivates the toxins within 1-3 hours. Adding chlorine to water if possible will destroy toxins as well.

Notification: Notification for animals is not necessary at this time.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Infected animals should be kept in a hospital or other stable environment until they are fully recovered.

Conditions for restoring disease-free status after an outbreak: Removal of decaying vegetable matter and carcasses should be carried out and areas should be cleaned with diluted bleach if possible.

Experts who may be consulted:

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Dept of Physiology and Pharmacology
University of Georgia CVM
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Raymond Sweeney, VMD
Professor of Medicine; Director, National Botulism Reference Laboratory
University of Pennsylvania CVM

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CLOSTRIDIAL DISEASE - TETANUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Equidae; bovidae; cervidae; primates; elephant; macropods; and rodents.	Contamination of wounds from bacteria in soil.	Muscle rigidity and spasm - localized or generalized.	Up to 80 % mortality in clinically ill animals.	Penicillin, tetanus anti-toxin, supportive care to reduce signs and support of airway.	Vaccination with tetanus toxoid.	No.

Fact Sheet compiled by: Ann E. Duncan

Sheet completed on: 18 January 2011; updated 11 July 2013

Fact Sheet Reviewed by: Dalen Agnew; Sarah Woodhouse

Susceptible animal groups: The disease is infrequent in animals. All warm-blooded animals are potentially susceptible. Horses and man are most susceptible, followed by cattle and sheep. Goats, pigs, dogs, elephants, kangaroos and rodents also have been infected. Cases can occur postpartum and after surgical procedures. Neonatal tetanus is seen in animals born without passive immunity, usually through infection of the umbilical stump. Carnivores and birds are resistant.

Causative organism: *Clostridium tetani* is a slender, gram-positive, anaerobic rod that may develop a terminal spore, giving it a drumstick appearance. *C. tetani* is found in the soil and intestinal tracts of animals and man. In the presence of oxygen it forms a protective capsule and may live in the soil in spore form for months to years. In an anaerobic wound, the spores germinate and multiply, producing a potent toxin known as tetanospasmin. Toxin is disseminated via blood and lymphatics and binds in the central nervous system, interfering with neurotransmitter release and blocking inhibition impulses. This reaction to the toxin leads to unopposed muscle contraction and spasm.

Zoonotic potential: Tetanus is acquired through contact with spores in the environment and is not transmitted from animal to animal or person to person.

Distribution: Worldwide. Found in soil, dust, and animals waste. Enzootic areas exist, mainly in the tropics.

Incubation period: Varies from 3 to 21 days after contamination of a deep wound that provides anaerobic conditions.

Clinical signs: It may start with localized contraction of muscles in region of infected wound. In generalized tetanus, trismus, neck stiffness, protrusion of the nictitans, and difficulty swallowing are often seen initially. Generalized rigidity, spasms of skeletal muscle and exaggerated reflexes follow. Animals often assume a "sawhorse stance" with ears erect, tail stiff and extended. In some cases, pyrexia, sweating and tachycardia are seen. Mortality of 80% is expected.

Post mortem, gross, or histologic findings: No lesions seen. It may be possible to see secondary aspiration pneumonia.

Diagnosis: Prior existence of a wound and characteristic signs are the basis for diagnosis. Direct microscopic examination of wound material may be useful. Attempting to culture *Clostridium tetani* from the wound is generally not successful. Mouse protective bioassays were historically used, but they are no longer available.

Material required for laboratory analysis: None

Relevant diagnostic laboratories: None

Treatment: Wounds should be cleaned and debrided. Antibiotic therapy with high doses of penicillin is

CLOSTRIDIAL DISEASE - TETANUS

effective against *C. tetani*. If tetanic spasms are occurring, supportive care should be provided and an adequate airway maintained. Treatment may include muscle relaxants, tranquilizers, and barbiturate sedatives.

Animals who have previously received toxoid should be given a booster. Tetanus antitoxin is hyperimmune serum generated by either a horse or human to bind and destroy the tetanus toxin. Antitoxin can be used to neutralize unbound circulating toxin, but cannot remove toxin already bound to nerve endings. Substantial risk of anaphylactic reaction is present when using a blood product from another species. Skin testing is used to test for reactivity to antitoxin before use. Antitoxin can be given under the skin or intraperitoneally but can take up to 3 days to reach a therapeutic level. Intravenous administration is more rapid but more likely to induce anaphylaxis. For passive protection, tetanus antitoxin effects will persist for about two weeks.

Prevention and control: Active immunization with tetanus toxoid is recommended in susceptible species due to ubiquitous presence in environment. Two doses of tetanus toxoid should be given 4-8 weeks apart with boosters given one year later and every 2-5 years thereafter. Vaccination is not contraindicated in pregnant animals. Passive immunization with antitoxin should be reserved for cases with high-risk wounds and no previous active immunization, unvaccinated patients who must undergo surgical procedures, and neonates in high-risk situations. Procedures such as umbilical cord severing, dehorning, and castration should be done in the most aseptic conditions possible, and antiseptics should be applied to surgical wounds.

Suggested disinfectant for housing facilities: None.

Notification: None.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: None.

Conditions for restoring disease-free status after an outbreak: None.

Experts who may be consulted: None identified.

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COCCIDIOIDOMYCOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals	-Inhalation of arthroconidia -Wound contamination (rare) -Intrauterine transmission (rare reports in horses, humans)	Dogs: wet or dry cough, fever, anorexia, weight loss, lameness. Cats: skin lesions, fever, anorexia, weight loss, cough and lameness rare. Zoo species: various signs reported	Subclinical infection most common. Untreated disseminated disease can be fatal.	Oral antifungal (i.e., fluconazole)	Avoid endemic areas; reduce animal exposure to dusty conditions.	Not directly but humans can contract

Fact Sheet compiled by: Maria Spriggs

Sheet completed on: 3 August 2011; updated 27 September 2012

Fact Sheet Reviewed by: Joe Wheat, Tiffany Wolf

Susceptible animal groups: Mammals, including humans; reptiles (rare) – published in Sonoran gopher snake; exotic/zoo cases published in black rhino, Indochinese tiger, Przewalski’s horse, ring-tailed lemur, California sea lion, bottlenose dolphin, chimpanzees, river otter, tapir, llama, bighorn sheep, koala.

Causative organism: Disease is also known as “Valley Fever.” Causative agents: *Coccidioides immitis* (California) and *Coccidioides posadasii* (Arizona).

Zoonotic potential: No direct transmission; however, fomites (bandages, cultures) should be handled carefully. People exposed to *C. immitis* develop asymptomatic infection or mild, transient respiratory signs, but rarely severe disease. In endemic areas, 10-15% of people are skin-test positive. Organ transplantation in humans has been reported as rare route of transmission.

Distribution: Disease is found only in the western hemisphere, specifically in Southwestern US (CA, AZ, NM, UT, NV, TX), Mexico, and Central and South America. Prevalence increases in years after high rainfall as arthrospores return to surface after rain then are dispersed by wind.

Incubation period: 1-3 weeks (respiratory signs); 4 months (disseminated disease)

Clinical signs: Clinical disease in dogs is most common in young males.

Primary pulmonary form– chronic dry or moist cough, fever, anorexia, weight loss

Disseminated form– lameness due to osteomyelitis of appendicular skeleton, draining skin tracts (especially in domestic cats), regional lymphadenopathy, CNS signs, cardiac signs, ocular lesions

Primary localized skin lesions – rare from penetrating wounds contaminated with organism

Post mortem, gross, or histologic findings: Pyogranulomatous inflammation seen in affected tissues. Gross lesions may be either disseminated or limited to lungs, mediastinum and thoracic lymph nodes. The lungs are often involved, even in disseminated disease where the primary complaint is not respiratory.

Diagnosis:

Clinical pathology: Nonregenerative anemia, leukocytosis, monocytosis, hyperglobulinemia, hypoalbuminemia

COCCIDIOIDOMYCOSIS

Radiography: Diffuse interstitial lung pattern, hilar lymphadenopathy, bone lesions in distal diaphysis of long bones which are more proliferative than lytic

Serology (IgM and IgG): rising titers confirm active infection. UC Davis Lab and Greene's text have further interpretation information.

Cytology and culture: Demonstration of organism by cytology is difficult. Extracellular spherules are most commonly found in lymph node aspirates, fluid from draining masses, or pleural fluid and pericardial fluid. Periodic Acid-Schiff (PAS)-stained smears more suitable than dry mount.

Antigen detection: Sensitivity lower in dogs than humans, research is in progress.

PCR: Research is in progress for a real-time PCR method.

Material required for laboratory analysis:

Blood, urine, fluid or tissue sample for cytology/histopath/culture (do not culture in-house)

Relevant diagnostic laboratories:

Fungus Testing Laboratory <http://strl.uthscsa.edu/fungus/index.shtml>

MiraVista Diagnostics www.miravistalabs.com

UC Davis Coccidioidomycosis Serology Lab <http://www.ucdmc.ucdavis.edu/medmicro/cocci.html>

Treatment: Fluconazole or amphotericin B is drug of choice but itra- and ketoconazole are effective as well. Posaconazole and voriconazole are newer and effective drugs, but are expensive and little information available for veterinary medicine.

Bone infections may be incurable. Itraconazole may be more effective for skeletal lesions. Relapse is possible following treatment. Treatment is recommended 1-6 months past resolution of clinical signs.

Prevention and control: Avoid endemic areas. Reduce animal exposure in dusty conditions such as feedlots. Dust control measures might include planting grass and wetting soil. People should wear facemask if dust exposure is unavoidable.

No vaccine is available; however research is ongoing for a safe/effective vaccine for humans.

Suggested disinfectant for housing facilities: Halogens (such as iodine, and chlorine in the form of bleach), phenolics (such as Tek-Trol®), and quaternary ammoniums (Di Quat® 10-S and Roccal®-D Plus).

Arthroconidia are resistant to dry heat, but can be inactivated by moist heat (121 C for minimum 15 minutes).

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Organism is not transmitted from infected animal to another animal.

Conditions for restoring disease-free status after an outbreak: Outbreaks occasionally occur, particularly following earthquakes or other events that disturb large amounts of soil in endemic areas. More recent human outbreaks have occurred among military trainees and among archeological workers.

Experts who may be consulted:

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Cattle (<i>Bos</i> spp.): bison, yak, water buffalo; reindeer; sheep and goats.	Direct Aerosol Transplacental	Fever, lethargy, severe respiratory signs, weight loss. Occasionally causes joint disease	Variable, can be severe; causes death rates of up to 80% of affected animals in Africa	Not recommended	Quarantine, testing, and removal of infected animals	No

Fact Sheet compiled by: Amanda Guthrie

Sheet completed on: 16 March 2011; updated on 5 April 2013, updated on 13 Feb 2018

Fact Sheet Reviewed by: Mark Drew, Nancy Carpenter

Susceptible animal groups: Domesticated bovids (*Bos* spp.); cattle, bison, yak and water buffalo have been infected. Wild bovids and camels are resistant. Primarily young animals are affected. Sheep and goats may be infected but do not experience pathology.

Causative organism: *Mycoplasma mycoides mycoides* small colony type (MmmSC). *Mycoplasma* is a self-replicating, pleomorphic and prokaryotic organism, resistant to beta-lactam antibiotics.

Zoonotic potential: No

Distribution: Endemic in most of Africa. Not in United States since 1892, considered to be eradicated from Western Hemisphere. Occasional outbreaks in the Middle East, Asia (India and China) and parts of Europe (Spain, Portugal and Italy).

Incubation period: 1 – 3 months, typically, but can range from 5 – 207 days.

Clinical signs: Similar to other pneumonias in cattle and difficult to differentiate based on clinical signs. It can cause polyarthritis or joint disease in young animals.

Acute: Severe respiratory signs such as coughing, labored breathing, outstretched neck and wide stance, loss of appetite and weight loss and decreased milk production.

Chronic: mild cough, recurrent low-grade fever.

Carriers: few or no signs of illness

Post mortem, gross, or histologic findings: Thickening and inflammation of lung tissues, typical of pleuropneumonia. Large amounts of straw-colored fluid in the thoracic cavity. Marbled appearance of lungs in both acute and chronic cases. Fluid accumulation in the lungs, fibrosis of lung tissue and pleura, fibrin deposits throughout the thorax.

Diagnosis: Confirmed with a blood (serological) screening test, organism can be cultured/isolated and identified with several tests. Available tests include complement fixation, latex agglutination, and competitive ELISA.

Material required for laboratory analysis: Live animal: blood, nasal secretions, bronchoalveolar washes, pleural fluid. Dead animal: lung fluids, lymph nodes, joint fluid and purulent discharge from lung tissue.

Relevant diagnostic laboratories: IDEXX CBPP Ab Test available outside the US

Treatment: Not recommended but tylosin is reported to be effective. Streptomycin, oxytetracycline, fluoroquinolones, and chloramphenicol may slow progression of disease and predispose to formation of sequestra. Treatment should only be attempted in endemic areas; treatment is unlikely to eliminate organisms, and will likely result in a carrier state.

Prevention and control: Quarantine of exposed and infected animals; testing and slaughter of infected animals. Organism is transmitted via saliva, urine, fetal membranes, and uterine discharges. Vaccine is available in endemic areas; only effective if herd coverage is high.

Suggested disinfectant for housing facilities: Inactivated by common disinfectants such as bleach; may survive in the environment for a few days. Formaldehyde solution (0.5%, 30 seconds) can be applied.

CONTAGIOUS BOVINE PLEUROPNEUMONIA

Notification: Foreign animal disease; contact appropriate state and federal authorities immediately. Reportable disease in many countries.

Measures required under the Animal Disease Surveillance Plan: Reportable as a foreign animal disease.

Measures required for introducing animals to infected animal: Not recommended; culling recommended.

Conditions for restoring disease-free status after an outbreak: all animals negative 12 months after last positive animal or last vaccinated animal slaughtered.

Experts who may be consulted:

Idexx Laboratories

1-800-548-9997

<http://www.idexx.com>

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CONTAGIOUS ECTHYMA “ORF”

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Domestic sheep and goats; wild artiodactylids; humans; rarely, domestic cat and dog.	Direct contact with vesiculo-proliferative lesions or scab material.	Minor to severe: skin proliferative lesions generally confined to skin of lips and muzzle, but can affect other skin, mucocutaneous areas and digestive system.	Typically mild and self-limiting in domestic animals, but can cause fatalities in severe cases particularly in young animals and in sensitive species like musk oxen.	Generally, no treatment is required. Supportive care and treatment of secondary infections in severe cases. Treatment with cidofovir systemically or topically, may be beneficial.	Isolate infected animals. Transmitted through damaged skin; dispose of scab material and contaminated bedding. Wear latex gloves and other protective clothing when working with known infected animals. Autogenous vaccine sometimes used.	Yes.

Fact Sheet compiled by: James M. Rasmussen

Sheet completed on: updated 6 January 2018

Fact Sheet Reviewed by: Anne Burgdorf, Kristin J Torbin

Susceptible animal groups: Ruminants (ovids, caprids, cervids typically but experimental transmission to calves, monkeys), camelids and possibly dogs, cats and squirrels.

Causative organism: Highly infectious epitheliotropic double-stranded DNA enveloped virus in the family Poxviridae, subfamily *Chordopoxvirinae* genus *Parapoxvirus* which includes the closely related bovine papular stomatitis virus, pseudocowpox virus, parapox virus of reindeer, parapoxvirus of red deer in New Zealand, and parapoxvirus of seals.

Zoonotic potential: Yes, orf virus is readily transmitted to humans. Infection typically occurs when abraded skin contacts infected animals or fomites.

Distribution: Orf virus has a worldwide distribution and is a common cause of disease in domestic sheep and goats and can affect a wide range of wild artiodactylids.

Incubation period: 2-3 days experimentally, 6-8 days under natural conditions

Clinical signs:

Humans: Generally, cause wart-like lesions on the hands and arms of people handling infected animals. Lesions progress quickly from macule, papule, vesicle, pustule until they become crusty lesions. As with animals, secondary bacterial infection may occur and can cause more severe complications in immunocompromised people. In uncomplicated cases they will heal in 2-6 weeks without scarring.

Animals: Similar to humans in rapid progression from macule through crusty proliferative papillomatous growths. Generally, start on mucocutaneous regions of the muzzle around nares and lip commissures, but can affect periorbital area, udder, legs/corony region, and oral cavity. Periorbital lesions may lead to visual impairment and/or mechanical trauma to cornea. Lesions on the muzzle or presence in oral cavity may reduce feed intake particularly in young suckling animals. Lesions rarely occur in esophagus or forestomach. Secondary, bacterial infections and myiasis may also occur. In uncomplicated cases scabs generally fall off in 4-6 weeks, but may persist for months.

Post mortem, gross, or histologic findings: Histologically, mature lesions demonstrate epidermal hyperplasia with ballooning degeneration of keratinocytes of the stratum spinosum, ulceration, and intracytoplasmic inclusion

CONTAGIOUS ECTHYMA “ORF”

bodies. Oftentimes, secondary bacterial infections are present.
Diagnosis: Electron microscopy of fresh or frozen lesion biopsies will typically demonstrate morphologically distinct ovoid-shaped parapoxvirus virions approximately 260nm x 160nm. Scab is not a preferred sample as it generally does not contain large numbers of virus. PCR and sequencing of viral DNA is required to differentiate Orf virus from other parapox viruses. Real time PCR can be performed on formalin fixed, paraffin embedded samples if fresh/frozen tissue is unavailable.
Material required for laboratory analysis: Biopsies of lesion- fresh/frozen and formalin fixed.
Relevant diagnostic laboratories: Laboratories capable of performing electron microscopy of biopsy samples can identify to the level of parapox virus. Sequencing of viral DNA is required for more specific identification.
Treatment: Lesions are generally self-limiting, but in some severe cases supportive care and antibacterial therapy for secondary infections is indicated. The antiviral drug cidofovir has been used with some success in the treatment of some pox virus infections. However, little if any benefit was subjectively noted during a course of intravenous treatment of cidofovir in two musk ox calves as compared to an untreated herd mate. An experimental trial using topical spray of cidofovir, sucralfate and sodium dihydrogen phosphate has shown benefit in sheep and cidofovir cream has been useful to treat lesions in people. In severe, unresponsive cases euthanasia should be considered before secondary complications cause significant morbidity.
Prevention and control: Vaccination should not be used in areas where the disease has not occurred. In endemic areas sheep and goats may be vaccinated with live virus vaccine which can be obtained from Colorado Serum Company- P.O. Box 16428- Denver Colorado, 80216- (800)525-2065 (http://www.colorado-serum.com). The vaccine is an attenuated live virus product which can cause disease in naïve animals and in susceptible species and people. Trial work has been done with DNA vaccines in China. Neither natural infection nor vaccination confers long-term immunity, but subsequent infections are generally less severe. Vaccines are more protective when developed from virus obtained from the same species infected. Infected animals should be isolated as long as scab material is present. Virus may persist in the environment or in wool for years in cool dry areas when encrusted in scab or organic material.
Suggested disinfectant for housing facilities: Removal and incineration or burial of organic material. Sunlight, heat and humidity leads to more rapid inactivation of virus, but virus may persist for months to years if frozen or present in cool, dry locations. Fairly resistant to disinfectants, but phenolics, quaternary ammonium compounds and iodophors can be effective disinfectants with proper concentration and contact time. Organic debris will decrease disinfectant efficacy. Steam sterilization and dry heat may also be utilized for disinfection.
Notification: Public health officials may need to be notified if zoonotic transmission occurs, depending on the state.
Measures required under the Animal Disease Surveillance Plan: Currently none
Measures required for introducing animals to infected animal: Maintain infected animals in a quarantine situation until lesions have healed and scabs have been lost. If feasible may want to bathe infected animal in order to remove all virus from fur. Do not introduce infected animal to an animal with a compromised immune system.
Conditions for restoring disease-free status after an outbreak: Wait for all scabs to be lost from infected animals. Remove bedding and biological material to the extent possible and disinfect with phenolic or quaternary ammonia disinfectants.
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CORONAVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Multiple mammalian and avian taxa.	Fecal-oral, inhalation, contaminated feed or fomites.	Diarrhea (often mucoid) due to enteritis, respiratory discharge, dyspnea, lethargy, death.	Asymptomatic infections are possible (bats). However when disease occurs, it is often severe.	Supportive, antibiotics to reduce secondary infections.	Vaccines exist for certain species: Recommended for - Avian Infectious Bronchitis Virus, Bovine Corona-virus, Trans-missible Gastro-enteritis Virus.	SARS and MERS are known to be zoonotic. Other coronaviruses may gain the ability to infect a new host, including humans.

Fact Sheet compiled by: Meredith M. Clancy

Sheet completed on: 17 Jan 2018

Fact Sheet Reviewed by: Kirsten Gilardi

Susceptible animal groups:

Birds: Avian Infectious Bronchitis Virus (IBV) – poultry; Turkey Coronaviral Enteritis (TCE) – turkeys; multiple other less pathogenic avian coronaviruses in other species

Mammals: Nearly ever mammal family has an endemic coronavirus.

In Hoofstock:

- Bovine Coronavirus (BCV) – domestic cattle; multiple ruminant species, including cervids, nondomestic bovids, and giraffids
- Equine Coronavirus (ECoV) – equids
- Porcine Deltacoronavirus (PDCoV), Porcine Epidemic Diarrhea Virus (PEDV), Porcine Respiratory Coronavirus (PRCoV), and Transmissible Gastroenteritis (TGEV) – suids

In Carnivores:

- Canine Enteric Coronavirus (CCV), emerging canine respiratory coronavirus (CRCoV) – canids
- Feline Coronavirus (FCoV) – felids, including both wild and captive exotic felids
- [Note: biotype that develops into feline infectious peritonitis (FIP) covered separately in this manual]
- Ferret enteric coronavirus (FECV, formerly Epizootic Catarrhal Enteritis or ECE) and Ferret Systemic Coronavirus (FRSCV) – ferrets
- Middle East Respiratory Syndrome coronavirus (MERS-CoV) – humans, suspected reservoir in bats, camels

In other mammals/multiple species:

- Severe Acute Respiratory Syndrome-associated Coronavirus (SARS-CoV) – humans, possibly other primates, carnivores—including palm civets, raccoon dogs, ferret badgers and domestic cats—and bats

Causative organism: Each disease caused by specific coronavirus (family Coronaviridae)

Zoonotic potential: Both SARS and MERS are confirmed zoonotic diseases.

Distribution:

Avian coronavirus distribution worldwide.

BCoV, CCV, FCoV – worldwide

MERS-CoV – Middle East'

SARS CoV - Asia

PEDV – discovered in UK in 1971, spread to Europe and Asia by 2013; first case in US in 2013.

TGEV – worldwide though less frequently in Europe, epidemic form; its deletion mutant (PCRv) first noted in 1980s, exists in endemic form

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FRECV – first noted in 1993 in US, FRSCV – first noted in Spain in 2004; also present in US
 PDCoV – first noted in US in 2014

Incubation period: generally very short, ranging from 18-24 hours to 3-4 days

Clinical signs: One of three disease manifestations:

- Enteric coronaviruses (BCoV, CCV, ECoV, FCoV, FECV, PDCoV, PEDV, TGEV) with tropism for GI epithelial cells cause malabsorptive, maldigestive diarrhea with possible dehydration, metabolic acidosis, and death; generally seen in young animals, especially BCV, TGE and PED. In BCV and TGE, the diarrhea is often mucoid and yellow in color with possible milk clots. In FECV, the diarrhea begins as green and mucoid progressing to a rice-water, granular stool.
- Respiratory coronaviruses (PRCoV, MERS-CoV, SARS-CoV) are adapted to enter and reproduce in the upper respiratory mucosa causing fever, nasal discharge, cough, pneumonia, and possibly death. Of note, BCoV has been implicated as a part of bovine respiratory disease complex, although whether this is due to the same or different virus remains unclear.
- Systemic coronaviruses infect and persist in macrophages, causing lethargy, weight loss, anorexia, abdominal masses, anemia, peritonitis, vasculitis, peritoneal effusions, and death.

Post mortem, gross, or histologic findings:

Enteric coronaviruses – gross lesions include thin-walled, flaccid small intestine, often with yellowish contents; fluid in the colon and/or cecum; microscopically, villous atrophy and blunting, with club-shaped, stumpy villi, often fused; hyperplastic crypt epithelium.

Respiratory coronaviruses, specifically SARS and MERS – gross lesions include pulmonary edema and consolidation; microscopically, diffuse alveolar damage with acute exudates with edema, hyaline membranes, and fibrosis with mixed cellular infiltration.

Systemic coronaviruses, specifically FSCV – gross lesions include whitish nodules through peritoneal viscera ± peritoneal effusion; microscopically, pyogranulomatous inflammation of visceral peritoneum, mesenteric adipose tissue, liver, lungs, kidneys, lymph nodes, spleen, pancreas, and other peritoneal viscera.

Diagnosis:

Avian IBV - ELISA available for flock screening; qPCR also available

Electron microscopy (EM) can be used as screening test for enteric coronaviruses

Molecular diagnostics (e.g. PCR) most widely used for antemortem diagnosis of coronaviruses; PCR generally cross reacts among the alpha-coronaviruses (FCoV, FECV, CCV, TGEV), and beta-coronaviruses (BCV); PCR confirmation in presence of clinical suspicion, performed by CDC approved lab for MERS, SARS

Indirect fluorescent antibodies (IFA) are often used on affected tissue in post-mortem samples, but can be used on antemortem swabs of nasal discharge or feces; IFA available for BCV, TGE, CCV

Material required for laboratory analysis:

ELISA – blood, serum, or eggs (poultry)

EM – feces, tissue

IFA – intestinal or respiratory tissue, nasal/pharyngeal swab or tracheal wash/bronchoalveolar lavage

IHC – formalin-fixed tissue

PCR – blood (serum or EDTA), mucosal (oropharyngeal, nasal, rectal) swabs peritoneal fluid, feces, fresh tissue

Relevant diagnostic laboratories:

Many tests widely available in state diagnostic labs in US. Specific testing:

IDEXX: ELISA (IBV)

Cornell Animal Health Diagnostic Laboratory: IFA (multiple); PCR (individual, and generic alpha- and beta-coronavirus); viral isolation [<https://ahdc.vet.cornell.edu/>]

Iowa State University Veterinary Diagnostic Laboratory: PCR (BCoV, CCV, all porcine), IHC (BCoV), ELISA (TGE, PRCoV), whole genome sequencing (PDCoV) [<http://vetmed.iastate.edu/diagnostic-lab/>]

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Michigan State University Diagnostic Center for Population and Animal Health: PCR (BCoV, CCV, ECoV, FeCoV, FECV, PDCoV, PEDV); [<https://www.animalhealth.msu.edu/>]

Texas A&M Veterinary Medical Diagnostic Laboratory: PCR (porcine, BCoV, CRCoV); IFA (BCoV, FeCV); EM [<http://tvmdl.tamu.edu/>]

Treatment: Treatment is supportive. In the case of enteric coronaviruses, treatment of the dehydration and electrolyte abnormalities is often accompanied by antibiotics to control secondary bacterial infections. Respiratory coronaviruses are often self-limiting, except in the case of the rare zoonotic SARS and MERS. Treatment of systemic coronaviruses is generally not successful, but rather focuses on controlling clinical signs.

Prevention and Control: Enteric coronaviruses are best prevented in similar fashion: by reducing fecal contamination of environment through routine cleaning and removal of feces, disinfection of enclosures, bowls, and other material with bleach once weekly. For respiratory coronaviruses, isolation of sick individuals and quarantine of new animals is important to reduce exposure of naïve animals to shed virus. In production animals, the all-in/all-out technique is used to reduce exposure and contamination. Vaccinations are available in many species and recommended to prevent IBV, BCoV, and TGEV. Vaccines often are combination rotavirus and coronavirus products and have been used in exotic hoofstock, although efficacy is variable and vaccine reactions have been reported in giraffids (*Okapia johnstonii*). Coronavirus vaccination is not currently recommended in domestic carnivores.

Suggested disinfectant for housing facilities: Coronaviruses are enveloped and labile in the environment. They are generally vulnerable to sunlight and basic disinfectants, like bleach, iodine, and quaternary ammonium compounds.

Notification: MERS and SARS are reportable to CDC. TGE reportable to USDA.

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Asymptomatic carriers are considered common in ferret coronaviruses. Pigs and cattle that have recovered from coronaviruses are not considered at high risk for repeat disease.

Conditions for restoring disease-free status after an outbreak: N/A

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COWPOX VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Rodent reservoir with large affected host range including: Felidae, Bovidae, Elephantidae, Equidae, Canidae, Mustelidae, Ailuridae, Herpestidae, Suidae, Camelidae, Tapiridae, Rhinocerotidae (black and white), Myrmecophagidae, Soricidae, Cercopithecidae, Callitrichidae, Humans	Most likely direct contact with infected animal or scabs. Poxviruses are fairly resistant to environmental inactivation.	From mild skin lesions to severe skin, oral/esophageal and respiratory lesions. Skin lesions may be absent in the pulmonary form. Lymphadenopathy and conjunctivitis may occur.	From mild to fatal although human fatalities are rare. Severity may depend on virus strain as well as species infected and individual immune status.	Generally self-limiting. Supportive care in more severe cases with antibiotics for secondary infections. Systemic or topical antiviral therapy with cidofovir may be beneficial.	Isolation of infected animals. Protective equipment including latex gloves and face shield to prevent cutaneous and mucous membrane exposure. Rodent control in endemic areas. Vaccinia virus vaccines available for zoo animals in some countries.	Yes

Fact Sheet compiled by: James M. Rasmussen

Sheet completed on: updated 20 January 2018

Fact Sheet Reviewed by: Sarah A Cannizzo

Susceptible animal groups: Rodents (voles, mice, rats, gerbils, ground squirrels, beaver, cavy), shrew, felids (domestic cat, cheetah, lynx, African lion, spotted leopard, ocelot, jaguar, puma, jaguarundi, Asian leopard cats), cattle, canids (dog, red fox, arctic fox), banded mongoose, marten, red panda, wild boar, okapi, llama, alpaca, horse, Malayan tapir, black rhinoceros, white rhinoceros, Asian elephant, African elephant, anteater, Barbary macaque, common marmoset, and human.

Causative organism: Double-stranded DNA enveloped virus in the family Poxviridae, subfamily *Chordopoxvirinae*, genus *Orthopoxvirus* which includes smallpox (Variola virus), monkeypox, buffalopox (vaccinia virus), ectromelia, camelpox, horsepox, raccoonpox, skunkpox, volepox, and Uasin Gishu disease. Multiple strains of cowpox exist.

Zoonotic potential: Yes. Smallpox vaccination confers protection against cowpox as well. Cats are the most common source of human infection. Less common sources include cattle, pet rats and an Asian elephant.

Distribution: Endemic in various rodent reservoir hosts in Great Britain, Scandinavia, European mainland and adjacent western Asiatic countries.

Incubation period: 3-10 days.

Clinical signs: Skin lesions usually progress through characteristic macule, papule, vesicle, and pustule phase before becoming scabbed. Generally mild self-limiting cutaneous pox lesions in humans and most animal species, but can become generalized and/or cause necrotizing pneumonia in certain species or immune-compromised individuals. Strain, route and dose of virus causing infection may influence course of disease.

Humans: Generally localized lesions on hands, face, arms or other points of contact with infected animal. Infection may cause lymphadenopathy and flu-like symptoms. Lesions typically resolve in 6-8 weeks without secondary bacterial infections which can extend the process by several weeks. Systemic infections and fatalities

COWPOX VIRUS

may occur in immune-compromised individuals. Previous vaccinia vaccination for smallpox should confer at least partial immunity.

Animals: Mild localized pox lesions to generalized lesions with ulcerations, including the conjunctiva, oral cavity and esophagus. Oral lesions may cause anorexia. An uncommon disease in cattle, but lesions most typical on udder and teats of cows and mouths of suckling calves. Infection may cause pyrexia and lymphadenopathy. Pulmonary disease is rare in most species, but is more common in felid species. Pulmonary involvement has also been seen in giant anteaters.

Postmortem, gross, or histologic findings: Epitheliotropic virus. The lesions undergo the classical poxvirus cascade of macules, papules and later collapse of the lesion from the center giving the lesion a targetoid appearance. The lesions then scab over and are slow to heal. Histologically, affected epithelial cells demonstrate ballooning degeneration and may have eosinophilic homogenous intracytoplasmic inclusions. The affected cells often swell and rupture leaving spaces filled with neutrophils and debris (pustules). These lesions with intraepithelial intracytoplasmic inclusions have also been identified in the pulmonary tract and oral cavity. In an outbreak in captive banded mongooses inclusions were also present in hepatocytes, enterocytes as well as in cells with histiocytic and fibroblastic morphology.

Diagnosis: Histopathology shows characteristic large homogenous eosinophilic cytoplasmic inclusion bodies in epithelial cells undergoing ballooning degeneration. Electron microscopy of fresh or frozen lesion material will typically demonstrate morphologically distinct orthopoxvirus (approximately 220nm x 280nm, brick-shaped virions with tubular surface projections). Cell culture. PCR and DNA sequence analysis. Serologic testing is available to determine if exposure occurred.

Material required for laboratory analysis: Biopsies of lesions- fresh/frozen and formalin fixed.

Relevant diagnostic laboratories: Laboratories capable of performing electron microscopy of biopsy samples can identify to the level of Orthopox virus. Sequencing of viral DNA is required for more specific identification.

Treatment: Lesions are generally self-limiting, but in some severe cases supportive care and antibiotics for secondary infections are indicated. Systemic treatment with the antiviral drug cidofovir has been used with some success in the treatment of some pox virus infections, but severe side effects have been reported in humans (e.g. nephrotoxicity). A compounded topical cream preparation of cidofovir is available as well. In severe, unresponsive cases euthanasia should be considered before secondary complications cause significant morbidity.

Prevention and control: Control of rodents to the extent possible in endemic areas. Isolated affected animals if possible. Vaccination of susceptible zoo animals with modified vaccinia virus Ankara (MVA) is authorized in some European countries.

Suggested disinfectant for housing facilities: Removal and incineration or burial of organic material. Sunlight, heat and humidity leads to more rapid inactivation of virus, but virus may persist for months or longer in scabs or crusts, if frozen, or present in cool, dry locations. Fairly resistant to disinfectants, but phenolics, quaternary ammonium compounds and iodophors can be effective disinfectants with proper concentration and contact time. Organic debris will decrease disinfectant efficacy. Steam sterilization may also be utilized for disinfection.

Notification: May be reportable in some jurisdictions.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Maintain infected animals in a quarantine situation until lesions have healed and scabs have been lost. If feasible may want to bathe infected animal in order to remove all virus from fur. In endemic areas may want to vaccinate susceptible species. Recovered animals should have immunity to the virus. Surviving infected brown rats have demonstrated continued viral shedding in feces and urine for more than a month after recovery.

Conditions for restoring disease-free status after an outbreak: Wait for all scabs to be lost from an infected animal. Remove bedding and biological material and incinerate or dispose of with other appropriate method. Disinfect environment with phenolic or quaternary ammonia disinfectants to the extent possible.

Experts who may be consulted:

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COWPOX VIRUS

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Q FEVER (*Coxiella burnetii*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ruminants, cats, dogs, lagomorphs, birds, marsupials, marine mammals, human	Two patterns: 1) wild animals and ticks, 2) domestic ruminants independent of wildlife cycles. Shed in high numbers within amniotic fluid and placenta. Excreted in milk, urine, feces. It also may be spread through wind and dust.	Mammal infections may be sub-clinical or lead to fever, anorexia, late term abortions, infertility, retained placenta, metritis.	Highly infectious. Humans – acute form has moderate morbidity (50%), generally low mortality (1-2%). Mortality with endocarditis is up to 65%.	Tetracycline antibiotics if showing clinical signs	Appropriate disposal of placenta, aborted fetuses	Yes; most often an acute febrile illness, but chronic manifestation, such as endocarditis can occur.

Fact Sheet compiled by: Diana Boon

Sheet completed on: 22 November 2010; updated 15 November 2012

Fact Sheet Reviewed by: Betsy Stringer, Jane Sykes

Susceptible animal groups: Peri-parturient ruminants (goats, sheep, cattle, pigs), cats, dogs, and wild animals (lagomorphs, and birds). Host range includes wild and domestic mammals, arthropods (ixodid and argasid ticks), and birds.

Causative organism: *Coxiella burnetii* (obligate intracellular Gram-negative bacteria)

Zoonotic potential: Yes, with acute and chronic presentations

Distribution: Global

Incubation period: Depends on number of infective organisms, but usually 2-3 weeks. Two patterns of transmission: via free-ranging animals and ticks, or between domestic animals with no wild animal involved. Tick bites are important for spread to animals, but rarely spread infections to humans. Human to human transmission is rare.

Clinical signs: Peri-parturient ruminants present subclinical disease, infertility, or anorexia, retained placenta, metritis, or late term abortion. Often sporadic abortions in herds can be seen that are followed by recovery without complications. In humans, acute Q fever is characterized by marked pyrexia, severe headache, myalgia, pneumonia, and similar flu-like signs while the chronic form is manifested as endocarditis, granulomatous hepatitis, optic neuritis, osteomyelitis and /or prolonged fever and chronic fatigue syndrome.

Post mortem, gross, or histologic findings: Necrotizing placentitis with large number of organisms in trophoblasts, but otherwise it is non-specific. Immunohistochemistry for *C. burnetii* can be performed on affected tissue(s) – mammary glands, supramammary lymph nodes, placenta, uterus, aborted fetus. The organism has a predilection for macrophages and monocytes.

Q FEVER (*Coxiella burnetii*)

<p>Diagnosis: IFA antibody tests can be used to screen for exposure or to identify recent infection using paired sera. Antibodies to phase 1 antigens predominate in chronic infection, whereas those to phase 2 antigens predominate in acute infection. A complement fixation test is also available but is less sensitive. Antibodies to both phase 1 and phase 2 antigens can persist for several years after the initial infection. Other means of diagnosis include: direct isolation using cell culture (which requires highly specialized facilities, PCR, and immunohistochemical staining of placenta/aborted tissues for organisms. Smears of placental cotyledon, vaginal discharge, and lung, liver, or stomach contents of aborted fetus stained with Stamp, modified Ziehl-Neelson, Gimenez, Giemsa, or modified Koster stain in order to detect organisms, but diagnosis using this method should be supported with serologic test results and clinical findings.</p>
<p>Material required for laboratory analysis: Placenta, vaginal discharges, and liver, lung, or stomach contents of aborted fetuses, and from milk, colostrum, and feces. At risk personnel (contact with reproductive organs, infected carcasses, and fur or wool) should wear adequate protective equipment to protect against small droplet and aerosol exposure.</p>
<p>Relevant diagnostic laboratories: State diagnostic laboratories or NVSL (Ames, Iowa) but submit to CDC (Atlanta, GA) for confirmation as needed. Positive test results are automatically reported to CDC if human case(s) involved.</p>
<p>Treatment: Tetracyclines are generally used to treat animals if showing clinical signs. Other active antimicrobials include azithromycin, fluoroquinolones, or trimethoprim-sulfa drugs. In humans, prolonged combination antimicrobial drug therapy is required for treatment of chronic Q fever.</p>
<p>Prevention and control: Vaccination is not commercially available in US. In wildlife settings, precautions against tick bites should be taken. Ruminants – particularly those in guest contact roles or domestic animals - can be screened for antibodies to <i>C. burnetii</i>, especially if in a breeding program. Obtain history of recent abortions if acquiring new animals from sending facility. Segregation of pregnant and periparturient animals from any new acquisitions for several weeks post-partum and appropriate quarantine of newly acquired animals and appropriate disposal of birth tissues and aborted fetuses by incineration or burying are recommended. At risk personnel (contact with reproductive organs, infected carcasses, and fur or wool) should wear adequate protective equipment to protect against small droplet and aerosol exposure. Pasteurization of milk products inactivates the organism.</p>
<p>Suggested disinfectant for housing facilities: Susceptible to ethanol, glutaraldehyde, gaseous formaldehyde, 10% bleach solution but bacteria are extremely hardy and resistant to heat, drying, and many common disinfectants.</p>
<p>Notification: Notifiable within the US if associated with human infection. The organism also is considered a potential bioterrorism agent due to heat resistance, high infectivity, and ability to aerosolize.</p>
<p>Measures required under the Animal Disease Surveillance Plan: Currently reported as present and sporadic to OIE every 6 months.</p>
<p>Measures required for introducing animals to infected animal: Infected animal(s) should be kept separated until the birth process is complete or acutely affected clinical animals have completed antibiotic therapy. The key is to maintain a properly cleaned facility and dispose of placental tissue, aborted fetuses, and feces appropriately by incineration or burying.</p>
<p>Conditions for restoring disease-free status after an outbreak: The organism is enzootic in most areas where domestic ruminants (cattle, sheep, goat) are found, and because of environmental persistence, eradication is difficult. The highest seroprevalence appears to be in sheep (~42%). If eradication is desired, repeated testing should be performed, potentially over several years, as the antibodies can persist for an extended period of time after the initial infection. Approximately 20% of seronegative animals will</p>

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continue to shed, so testing for restoring disease free status becomes problematic.

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CRYPTOCOCCOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, birds, rare in reptiles and amphibians	Inhalation of airborne organisms	Typically respiratory, central nervous system, ocular, or cutaneous signs; possibly in combination.	Moderate to severe; guarded prognosis with neurologic signs.	Antifungal drugs; in some cases, surgical excision of granulomas may be helpful	Avoid contact with pigeon droppings.	Not directly transmissible from animals to humans. Common source exposure can occur.

Fact Sheet compiled by: Cynthia Stadler

Sheet completed on: 4 May 2011; updated 30 May 2013

Fact Sheet Reviewed by: Jane Sykes; Julie Harris

Susceptible animal groups: Mammals, birds, rarely in reptiles and amphibians

Causative organism: Most often it is associated with *Cryptococcus neoformans* or *C. gattii*.

Zoonotic potential: Animals and people may become infected by the same environmental source. Humans with HIV are at a greater risk for acquiring infection. Pet bird feces have been implicated as a possible source of *C. neoformans* infection for immunocompromised people but and no mammal-to-mammal transmission has been documented.

Distribution: Worldwide, but especially southeastern and western Australia, British Columbia in Canada, and the west coast of the US. In specific, *C. neoformans* is considered global and ubiquitous while *C. gattii* likely is present in hotspots around the world, and recently associated with an outbreak in the Pacific Northwest US and British Columbia. Some implication has been made with *Eucalyptus* trees although other hardwood tree species have been implicated.

Incubation period: Unknown. May be a few months to many years in some circumstances.

Clinical signs: Rhinitis, sneezing, pulmonary granulomas (cryptococcomas); chorioretinitis; CNS signs include ataxia, circling, and blindness; cutaneous nodules or ulceration; lymphadenopathy, weight loss, lethargy, vomiting if disease is widely disseminated.

Post mortem, gross, or histologic findings: Gross lesions may include gelatinous masses and granulomas. Histopathology reveals pyogranulomatous to granulomatous inflammation in affected organs with intralosomal encapsulated yeasts that are round to oval with a distinctive capsule.

Diagnosis: Cytology, fungal culture, tissue biopsy, antigen testing (serum and cerebrospinal fluid), PCR (not currently widely used). Distinction of *C. gattii* from *C. neoformans* requires specialized canavanine glycine bromothymol blue agar (Hardy Diagnostics).

Material required for laboratory analysis: Samples of the tissue affected, serum, cerebrospinal fluid

Relevant diagnostic laboratories: Many state, university and commercial laboratories run specific testing for cryptococcosis, although results of antigen tests may vary between laboratories. Culture is not hazardous for laboratory personnel and allows antifungal susceptibility testing and molecular typing.

Treatment: Long term treatment (months to years) with fluconazole, itraconazole, voriconazole, ketoconazole, and/or amphotericin B. Flucytosine can be used in combination with one of these antifungal agents but should never be used alone due to rapid development of resistance and it may be prohibitively expensive. Surgical excision of cutaneous nodules can assist with drug penetration into poorly perfused tissues.

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<p>Prevention and control: Avoid accumulations of bird droppings (especially from pigeons) for <i>C. neoformans</i>. Prevention difficult to achieve for <i>C. gattii</i> due to implications of contact with contaminated soil and tree bark.</p>
<p>Suggested disinfectant for housing facilities: accelerated hydrogen peroxide, potassium peroxydisulfate, 1% sodium hypochlorite, iodine, chlorhexidine.</p>
<p>Notification: None</p>
<p>Measures required under the Animal Disease Surveillance Plan: None</p>
<p>Measures required for introducing animals to infected animal: N/A</p>
<p>Conditions for restoring disease-free status after an outbreak: N/A</p>
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<p>References:</p> <ol style="list-style-type: none"> 1. Aiello, SE and MA Moses (eds). Merck Veterinary Manual Online. 2013. http://www.merckmanuals.com/vet/generalized_conditions/fungal_infections/mycoses/cryptococcosis.html. Accessed 2 August 2013. 2. Cryptococcus neoformans- Material Safety Data Sheets. 2010. Public Health Agency of Canada. http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/cryptococcus-eng.php. Accessed 2 August 2013. 3. Jones, T.C., R.D. Hunt, and N.W. King (eds). 1997. Veterinary Pathology. 6th edition. Lippincott Williams & Wilkins, Baltimore, MD. Pp. 516–517. 4. Lagrou, K., J. Van Eldere, S. Keuleers, et al. 2005. Zoonotic transmission of Cryptococcus neoformans from a magpie to an immunocompetent patient. J. Int. Med. 257: 385-388. 5. Lester, S.J., R. Malik, K.H. Barlett, and C.G. Duncan. 2011. Cryptococcosis: update and emergence of Cryptococcus gattii. Vet. Clin. Pathol. 40: 4-17. 6. Nosanchuk, J.D., S. Shoham, B.C. Fries et al. 2000. Evidence of zoonotic transmission of Cryptococcus neoformans from a pet cockatoo to an immunocompromised patient. Ann. Intern. Med. 132:205-208. 7. Okabayashi, K., M. Imaji, T. Osumi et al. 2009. Antifungal activity of itraconazole and voriconazole against clinical isolates obtained from animals with mycoses. Jpn. J. Med. Mycol. 50:91-94. 8. Spickler, A.R. Cryptococcosis. 2013. http://www.cfsph.iastate.edu/Factsheets/pdfs/cryptococcosis.pdf. Accessed 2 August 2013. 9. Sykes, J. 2012. Treatment of fungal infections: the which, why and how of antifungal drug therapy. Proc of ACVIM Forum. New Orleans, Louisiana. 10. Sykes, J.E., and R.M. Malik. 2014. Cryptococcosis. In: Sykes, J.E. (ed.). Canine and Feline Infectious Diseases. Elsevier Saunders, St. Louis, Missouri. Pp. 599-612.

CRYPTOSPORIDIOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals Avians Herptiles Fish Humans	Direct: fecal to oral. Waterborne transmission, possible paratenic host transmission, possible aerosol transmission in birds.	Gastrointestinal: Diarrhea, vomiting. Respiratory disease documented in birds.	Depending on the affected species and organ system, severity can vary from a mild, transient, self-limiting disease to a severe and fatal disease. Severe disease is typical of immune suppressed patients, and reptiles.	Nitazoxanide (Alinia) is licensed and approved for use in humans. Oral bovine hyper-immune serum is reported to be effective in reptiles. Paromomycin (Humatin) is effective against some stages of the disease but will not eliminate infection.	Strict quarantine, testing of new specimens, biosecurity, personal and environmental hygiene.	Yes. <i>C. parvum</i> is known to affect both animals and humans. Other species (<i>C. felis</i> , <i>C. canis</i> , <i>C. meleagridis</i> , <i>C. fayeri</i> , etc.) are occasionally isolated from immune compromised.

Fact Sheet compiled by: Christopher J. Bonar

Sheet completed on: 3 August 2011; updated 9 April 2013; updated 2018

Fact Sheet Reviewed by: David Lindsay, Christie Hicks

Susceptible animal groups: Mammals, avian, herptiles, fishes

Causative organism: *Cryptosporidium* sp. of which at least 20 different species exist. *Cryptosporidium parvum* in mammals and humans, *C. ubiquitum* in man and many species, *C. saurophilum* in lizards, and *C. serpentis* in snakes are the most commonly encountered species in zoological medicine, but there are many others. *C. meleagridis*, *C. baileyi* and *C. galli* are reported in birds. Some species are being debated (eg. *C. parvum* = *C. pestis*), but clearly there are many and molecular techniques may define still more.

Zoonotic potential: Yes, at least for mammalian forms.

Distribution: Common in domestic dairy calves, and often transmitted to humans. Virtually, all dairy calves become infected if sampled repeatedly during life. *C. andersoni* and *C. bovis* are found in weaned cattle. *Cryptosporidium* spp. are present in free-ranging wildlife. *C. serpentis* apparently affects both free-ranging and captive squamates. The reptile form is common in zoological parks and serpentariums. Avian forms are found in both exotic and domestic species. A human form, *C. hominis* has been transmitted to lemur species in Madagascar from the increased exposure of humans into their wild habitats.

Incubation period: This period is not well defined in zoological specimens. Reptiles can show gradual, progressive illness. Inapparent carriers are suspected. Humans often become acutely infected, and incubation time is approximately 2 to 10 days, although often the exact time between exposure and onset of disease is often unknown.

Clinical signs:

Humans: Diarrhea, intestinal cramping, low grade fever, nausea, vomiting, malabsorption, dehydration. Occasionally respiratory, ocular, pancreatitis, choecystitis, cholangitis.

Animals: Vomiting, anorexia, and mid-body (gastric) swelling in snakes. Diarrhea in lizards, chelonians, exotic and domestic mammals, and birds. Respiratory disease is also reported in birds.

Post mortem, gross, or histologic findings:

This coccidian parasite can cause a variety of pathology in different taxonomic groups. In mammals, enteritis is the most common. In reptiles, proliferative gastritis is the most common manifestation in snakes, often yielding a firm, mid abdominal swelling. In lizards, enteritis with hyperplasia and mononuclear cell infiltrate in the small intestine is more common. In both gastritis and enteritis, the organism can often be seen attached to the luminal

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surface or within a parasitivoracious vacuole within the host cells. Aural and pharyngeal cell polyps are reported in iguanas. Birds are often diagnosed with either enteric or respiratory tract infections. In humans, infections of the bile ducts, respiratory tract, and conjunctiva are found in immunosuppressed patients.

Diagnosis: Diagnosis is by histopathology, ELISA test on feces, Meriflour IHC of gastric washings or gastric biopsies. Acid-fast stain of gastric wash, fecal smear, or cytologic preparations. Low sensitivity and specificity of acid-fast stains on gastric washes, fecal smears, and cytologic preparations makes confirmation by more sophisticated tests (of both positives and negatives) important. FLOTAC has been shown to detect *Cryptosporidium* in reptiles. Sheather's flotation sedimentation staining can also be used and is 83% sensitive and 99% specific.

Material required for laboratory analysis: Fecal sample, gastric wash, gastric or intestinal biopsy.

Relevant diagnostic laboratories: Many laboratories can perform these tests, although some are more experienced or have more capabilities than others. Much of the pioneering work on this disease in exotic animals has been performed at the University of Florida and Johns Hopkins University.

Treatment: Nitazoxanide (Alinia) is licensed and approved in the U.S. for treatment of immune-suppressed humans with clinical disease from cryptosporidiosis. It is not documented to shorten the course of disease in immunologically normal humans. Its effectiveness in exotic animals is not published. Oral bovine hyper-immune serum has been demonstrated to be effective in reptiles. Paromomycin (Humatin) has been used to suppress the organism, but it is not effective against all stages of the organism and is unable to eliminate the infection. Other drugs similar to Nitazoxanide are in pre-clinical testing for use in humans and may show promise for exotic animals as well. Drugs such as tizoxanide, tizoxanide-glucuronide, D-eritadenine, and (S)-DHPA all have shown promise in in-vitro testing.

Prevention and control: Strict quarantine and testing of reptiles for *Cryptosporidium* has long been considered an important part of biosecurity for serpentariums. Good hygiene and disinfection are essential to prevent zoonotic transmission of mammalian *Cryptosporidium* to human caregivers. Testing of symptomatic birds, reptiles, and mammals should be performed, and appropriate biosecurity and hygienic practices implemented to prevent spread to other animals and caregivers when positive cases are detected.

Suggested disinfectant for housing facilities: *Cryptosporidium* is notoriously resistant to most common disinfectants, especially chlorine-based disinfectants. Heat sterilization of implements is most reliably effective, as well as having separate implements and tools to prevent spread from one enclosure or exhibit to another.

Notification: Public health officials may need to be notified if zoonotic transmission occurs. In humans, it is a reportable disease.

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: An infected animal should not be introduced to others of the same taxonomic group. However, mammalian *Cryptosporidium parvum* has been shown to be non-infective to some reptiles. *Cryptosporidium* has been shown to be transmissible between squamates and chelonians, and wild mammals have been shown to carry *C. parvum*.

Conditions for restoring disease-free status after an outbreak: Heat disinfection is the only method known to destroy oocysts and can be used to disinfect utensils, cleaning equipment and surfaces.

Experts who may be consulted:

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CYTAUXZOOONOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Felids, wild and domestic	Tick-borne (<i>Amblyomma americanum</i> and <i>Dermacentor variabilis</i>)	Domestic cats and some exotic felids: some cats develop no clinical signs while others may develop high fever, lethargy, dyspnea, depression, dehydration, anorexia, anemia, hepatosplenomegaly, and/or jaundice. Others die acutely. Exotic felids: often no clinical signs.	Non-clinical or mild to severe including death; could depend on numerous factors such as species of felid, strain or genotype of parasite, or other unknown factors.	Mortality can be high even with treatment. A combination of atovaquone and azithromycin seems to have the highest success rates. Supportive care also should be provided.	Avoid contact with ticks by keeping cats indoors. Outside cats should have effective acaricides applied. No vaccine available.	No

Fact Sheet compiled by: Michael J. Yabsley

Sheet completed on: 1 August 2013; updated 2018

Fact Sheet Reviewed by: Adam Birkenheuer

Susceptible animal groups: Felids. *Cytauxzoon felis* has been reported from domestic cats, bobcats (*Lynx rufus*), puma (*Puma concolor*), and captive exotic felids (e.g., tigers [*Panthera tigris*]) in the United States. *Cytauxzoon manul* infects the Pallas cat. *Cytauxzoon* spp., some genetically similar to *C. felis*, have been reported from domestic cats and numerous free-ranging and/or captive exotic felids in South America, Africa and Europe.

Related *Cytauxzoon* spp. has been reported from meerkats (*Suricata suricatta*) from South Africa and Formosan pangolins (*Manis pentadactyla pentadactyla*) from Taiwan.

Causative organism: *Cytauxzoon* spp. are Apicomplexan parasites in the Order- Piroplasmida which are related to important human and veterinary pathogens in the genera *Babesia* and *Theileria* spp. In the US, *Cytauxzoon felis* is the causative agent of cytauxzoonosis in domestic cats and some exotic felids. Bobcats, and other wild felids (e.g., cougars), are the natural reservoir but chronically infected domestic cats can serve as a source of infection for ticks. Outside of the US, other *Cytauxzoon* species or genetic variants of *C. felis* infect wild and domestic felids; however, clinical cytauxzoonosis is rare.

Zoonotic potential: None

Distribution: *C. felis* has been reported from numerous states in the eastern US but is likely found throughout the range of the vector(s) and the main wildlife reservoir (bobcats). Other species of *Cytauxzoon* have been reported in parts of South America, Africa, Europe and Asia. Some of the *Cytauxzoon* likely represent novel species or have been described as separate species (e.g., *C. manul*), but genetic data indicates that *Cytauxzoon* from Brazil is closely related to *C. felis* from the US. Exotic felids kept in enclosures that allow tick exposure within the natural range of any *Cytauxzoon* spp. are at risk of infection.

Incubation period: *C. felis* can typically be detected in erythrocytes of infected cats approximately 1-3 weeks after an infected tick bite. Clinical signs typically occur 5-16 days after infected tick bite.

Clinical signs:

Domestic cats: The majority of domestic cats develop severe clinical disease but some never develop clinical signs, but remain chronic carriers. Those with clinical signs may develop high fever, lethargy, dyspnea, depression, dehydration, anorexia, anemia, hepatosplenomegaly, and/or jaundice.

Captive wild/exotic felids: Development of clinical signs is highly variable and may depend on felid species,

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strain of parasite, or some other factor. Fatal cases have been reported in a tiger housed in Florida and lions in Brazil; however, asymptomatic infections have been detected in tigers in US and ocelots (*Leopardus pardalis*), oncilla (*Leopardus tigrinus*), jaguar (*Panthera onca*), and puma in Brazil.

Wild felids (natural reservoirs): Wild reservoir species rarely develop clinical signs but very rare acute mortality has been reported among young bobcats. In addition, three infected cougar in the US developed a transient anemia and increased serum bilirubin concentrations and increased alanine aminotransferase and aspartate aminotransferase activities soon after infection; however, all recovered rapidly without treatment.

Clinical pathological, gross, and histopathological findings: Parasitemias of *C. felis* on blood smears are generally low (<5%), even for clinically ill felids. Leukopenia or pancytopenia may be present as well as thrombocytopenia and normocytic, normochromic anemia. Gross lesions are typically severe as death occurs due to severe occlusions of vessels by developing parasites. Felids may have pale or icteric mucous membranes, petechiae and ecchymoses in the lung, heart, lymph nodes and on mucous membranes, splenomegaly, lymphadenomegaly, and hydropericardium. Numerous large schizonts will be noted in the cytoplasm of infected macrophages that often occlude the lumens of numerous vessels of many tissues, especially the lungs. Despite the large numbers and size of schizonts, a lack of inflammatory reaction generally is present.

Diagnosis: Piroplasms may be detected in stained thin blood smears if sufficiently high parasitemias are present; however, subclinical chronic carriers generally have very low parasitemias. Although feline babesiosis has not been reported in domestic cats in the US, *C. felis* trophozoites are morphologically similar to other small piroplasms so PCR testing is necessary to definitively identify *C. felis*. If possible, a fine needle aspiration of a peripheral lymph node, spleen, or liver should be performed to identify schizonts in macrophages. These intracellular schizonts are not found in babesiosis cases so can be used to definitively identify *Cytauxzoon* infections. Several PCR protocols have been developed for the detection of *C. felis*. If PCR assays are not specific to *C. felis*, amplicons should be sequenced to confirm identification as other piroplasms can infect felids, especially wild felids.

Material required for laboratory analysis: Thin blood smears fixed and stained for detection of piroplasms and anticoagulated whole blood (for PCR testing and preparation of thin blood smears). Formalin fixed needle biopsies of tissues for histologic evaluation for schizonts.

Relevant diagnostic laboratories: Many diagnostic laboratories have PCR based assays for *C. felis*.

Treatment: Despite treatment, mortality rates can be high. The greatest success has been obtained using atovaquone (15 mg/kg, PO, tid for 10 days) and azithromycin (10 mg/kg, PO, sid for 10 days) with supportive care (fluid therapy and heparin). Limited success has been obtained using imidocarb and diminazene diaceturate while even less success has been obtained using parvaquone, buparvaquone, trimethoprim/sulfadiazine, and sodium thiacetarsamide.

Prevention and control: Because *Cytauxzoon* is tick-borne, limiting exposure of felids to ticks is necessary to prevent transmission. For domestic cats, the best prevention is to keep cats indoors. For exotic or wild felids or domestic cats that are allowed outdoors, an effective acaricide or acaricide-treated collar should be used to prevent or limit tick infestation. If possible, tick checks can also decrease risk by finding and removing ticks prior to transmission. Habitat modification can also be used around a premise to decrease local habits for ticks which should decrease tick infestation rates of animals.

Suggested disinfectant for housing facilities: Prevent tick-exposure

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: This parasite is tick-borne so direct contact between animals is not a risk factor for infection. However, tick prevention should be implemented.

Conditions for restoring disease-free status after an outbreak: Not applicable

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CYTOMEGALOVIRUS (CMV)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mice, rats	In mice, urine, vertical transmission; in mice/rats, tears, saliva	None in natural infections; immune, reproductive, and hematopoietic effects when experimentally inoculated in mice	Usually subclinical	Depopulation and restocking with MCMV-free animals	Isolate wild individuals from laboratory colonies	No
Guinea pig	Saliva, urine, vertical	Pneumonia, fetal death; neonatal runting, neurologic deficits, deafness	Subclinical to severe	None effective	Separate infected from GpCMV-free	
Swine	Ocular/nasal discharges, urine, cervical fluids	Abortion, neonatal piglet losses, runting, poor weight gain, inclusion body rhinitis, pneumonia	Subclinical when >3 wks old	Antibiotics for secondary bacterial invaders	“All-in-all-out” farrowing and weaning management	
Cattle	Possibly milk; not well documented	Rare to absent. Possible abortion; respiratory/genital diseases produced experimentally only	Subclinical	None	None needed	
Horses	Probably respiratory secretions	Immunosuppression; corneal ulcers; pharyngitis; lymphadenopathy; fever in foals.	Subclinical to moderate, possible foal death	Symptomatic	None	
Non-human primates	Bodily secretions	SIV-infected macaques similar to HIV infected humans; necro-	Majority subclinical	Symptomatic	Screen prior to introduction if necessary	

CYTOMEGALOVIRUS (CMV)

		tizing enteritis, encephalitis, lymphadenitis, pneumonitis				
Humans	Intrauterine, sexual contact, bodily fluids, trans-fusions and trans-plants, fomites	Congenital: childhood deafness. Acute acquired: mononucleosis-like fever, malaise, myalgia, arthralgia. Immunocompromised: retinitis, esophogitis, pancreatitis, pneumonia.	Majority subclinical; fatalities in transplant patients	Antivirals—ganciclovir or Foscarnet	Good hygiene, hand washing, limiting transfusions, screening donors	
Australian finches	Respiratory	Depression, anorexia, conjunctivitis, dyspnea	High mortality rate	Symptomatic	Isolate captive from wild birds	

Fact Sheet compiled by: Eleanor C. Manela Newcomb; updated by Jan Ramer

Sheet completed on: 1 April 2011; updated 1 September 2013

Fact Sheet Reviewed by: Meredith Clancy; Kyoung-Jin Yoon; Hayley Murphy

Susceptible animal groups: Rodents, swine, cattle, non-human and human primates, some other mammals, some marsupials, some passerine birds.

Causative organism: Family *Herpesviridae*, Subfamily *Betaherpesvirinae*, except in cattle and horses, where it is *Gammaherpesvirinae*.

Zoonotic potential: Although the virus has a restricted host range, interspecies transmission does occur in non-human primates. No natural transmission to humans from other species documented.

Distribution:

Rodents: The virus is widespread through reservoirs in wild populations. Specifically in guinea pigs, the virus is common in pets and laboratory populations but its distribution in the wild is unknown.

Swine: Worldwide, with >90% herd prevalence in North America, Europe, and Japan.

Cattle: Worldwide.

Equine: Widespread.

Non-human primates: Widespread.

Humans: 85% of population worldwide and in US, 50-85% adults are infected by age 40. If infection is acquired by mother during pregnancy, then up to 20% neonates severely affected.

Finches: reported mostly in Europe.

Incubation period: Unknown in most species. Swine: 10-20 days. Humans: 3-12 weeks. Lifelong latent infection occurs commonly, may produce periodic episodes of reactivation, viral replication and shedding.

CYTOMEGALOVIRUS (CMV)

Clinical signs: In rats, mice, and squirrels, no clinical signs are presented in natural infections. Guinea pigs, however, present weight loss, ruffled coat, abortion, and neonatal abnormalities. Swine present signs of respiratory, neurologic, and reproductive systems. Cattle present no correlation between presence of virus and specific lesions. Horses present conjunctivitis, oculonasal discharge, and cough. In finches, affected birds present respiratory disease and death. Humans and non-human primates are usually subclinical. Immunocompromised non-human primates can present diarrhea, melena, dyspnea, and terminal opportunistic infection. In humans, severe permanent disabilities in children can occur when primary infection occurs during pregnancy, or when acquired in AIDS patients, organ transplant and cancer chemotherapy. These clinical signs range from malaise to permanent hearing loss, and include mental retardation; gastrointestinal, pulmonary, and auto-immune disease; and death.

Post mortem, gross, or histologic findings: Marked enlargement (6x normal) of nucleus and cytoplasm of infected cells (cytomegaly) is observed with large intranuclear (“owl’s-eye”) and smaller basophilic intracytoplasmic inclusions.

Affected organs by rodent species include: mice: submandibular salivary gland; rats: salivary/lacrimal glands; European ground squirrels: salivary gland; guinea pigs: salivary glands/renal tubules.

Swine: macrophages in lungs, nasal mucosa, turbinates, and upper respiratory tract.

Sheep: cytomegaly with virus has been detected in lung tissue of lamb with *Mycoplasma pneumonia*.

Cattle: monocytes/macrophages in multiple organ sites.

Horses: leukocytes and respiratory tract and, kidneys.

Non-human primates: inclusion bodies in alveolar septa and septal lining, liver, CNS, spleen, kidney, testes; meningoencephalitis, necrotizing vasculitis; neutrophilic infiltrates may be prominent in CNS and gastrointestinal tract. Several other species (e.g. hamster, chimpanzee, and gorilla) have been diagnosed based on characteristic cytomegaly in the absence of virus isolation.

Diagnosis: Virus isolation from bodily fluids, macrophages, or affected tissues can be performed. Horses can have nasal swabs submitted. Serologic or molecular testing options (ELISA, IFA, PCR) are available.

Material required for laboratory analysis: Tissues and bodily fluids for virus isolation include biopsies or post-mortem samples, or urine, cervical secretions, semen, saliva, lung lavage, or blood.

Relevant diagnostic laboratories:

Pathogen Detection Laboratory
California National Primate Research Center
University of California
Road 98 & Hutchison,
Davis, California 95616
(530) 752-8242
Fax: (530) 752-4816
PDL@primate.ucdavis.edu
<http://pdl.primare.ucdavis.edu/>

VRL Laboratories-San Antonio
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Treatment: Most species are recommended to receive symptomatic treatment. If severely debilitated from disease, cull may be recommended and entire groups can be depopulated if virus will interfere with laboratory studies.

In humans, several weeks course of intravenous antivirals (e.g., ganciclovir or Foscarnet) are administered and treatment is usually lifelong for AIDS patients.

Prevention and control: Separate wild from captive populations to minimize transmission. Test individuals prior to introduction if applicable. All-in-all-out in production facilities used with all individuals moved out as a group and premises disinfected thoroughly between groups.

In humans, blood and blood-product transfusions should be limited and CMV-seronegative donors selected. High-titer CMV immunoglobulins may be prophylactic for bone marrow or renal transplant recipients.

Suggested disinfectant for housing facilities: Disinfectants or detergents should be utilized that are effective against herpesviruses,.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Do not introduce infected animal to pregnant or immunocompromised individuals, or to group-housed research animals.

Conditions for restoring disease-free status after an outbreak: Disinfect environment, depopulate and restock with CMV-free animals

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Most vertebrates including mammals, reptiles, and avian species	Direct contact with infected hairs, fomites, infected animals, or environment (rare). Contact does not always result in infection.	Lesions can appear different in each species, but most consist is a well-demarcated area of alopecia with grey-white scaling, crusting, and mild erythema. Depending on the species and complicating factors lesions may or may not be pruritic.	Generalized dermatophytosis can be difficult to cure, but mortality is low. In healthy hosts and low burden of disease dermatophytosis can be self-limiting.	Standard of care for treatment involves topical antifungal agents in combination with systemic therapies (see below for details).	Holding period for newly introduced animals. Isolation of affected animals until mycological cure. Protective clothing and good personal hygiene after handling infected animals. Decontamination of all fomites (brushes, blankets, toys, cages, etc.) and environment. Vaccines do not protect against exposure in dogs and cats, though may show some efficacy in cattle and horses (see below).	Yes-Specific species carry higher potential for zoonosis than others (see details below).

Fact Sheet compiled by: Samantha Lockwood

Sheet completed on: 8 February 2018

Fact Sheet Reviewed by: Ryan Colburn

Susceptible animal groups: All mammals can be affected.

- Avian species-rare, mostly seen in domestic fowl.
- Reptiles-uncommonly affected; reports in lizards, snakes (green anacondas, *Eunectes murinus*), chameleons, and one report in an iguana.
- Pocket Pets-rabbits, chinchillas, ferrets, guinea pigs, hedgehogs, less commonly rats and mice.
- Often animals with compromised or underdeveloped immune systems will be more commonly affected. Young, stressed, elderly, or sick (neoplasia, underlying metabolic disease) animals are more likely to develop infection after exposure. Animals with compromised skin barriers such as allergic patients, genetic predispositions (Yorkshire terriers and Persian cats), or long coats may also be more likely to develop infection after exposure.

Causative organism: Three genera- *Microsporum*, *Epidermophyton*, and *Trichophyton*. The three genera can be categorized into anthrophilic (adapted to humans), zoophilic (adapted to animals), and geophilic (normally live in environment, but occasionally are infectious). Both *Microsporum* and *Trichophyton* are anthrophilic and zoophilic, whereas only one species of *Epidermophyton* (*E. floccosum*) has been known to cause disease in humans (anthrophilic). The most common geophilic species that can cause disease are species from the *M. gypsum* complex.

- Overall, the most common species that affect domestic animals are *Microsporum canis* (dogs and cats), *M. equinum* (horses), *M. nanum* (pigs), *M. gypsum* (dogs and cats), *T. mentagrophytes* (horses, dogs, and cats), *M. persicolor* (voles), and several species of *T. mentagrophytes* complex (rodents, rabbits, hedgehogs).
- Microsporum canis* is the most common causative agent in dogs and cats, but can be routinely found in horses, rabbits, cattle, sheep, goats, camelids, and swine.
- Microsporum gallinae* occurs in domestic birds (chickens), uncommon in wild birds.
- Microsporum nanum* is the most commonly isolated species in swine, though *T. mentagrophytes*, *T. verrucosum*, and *M. canis* have also been isolated.
- Microsporum gypsum* has been found in many different species of animals, but is mostly geophilic. It has been reported in cats, dogs, ruminants, camelids, horses, pigs, birds, and rodents.

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- *Trichophyton spp.* is most commonly isolated from reptiles.
- *Trichophyton equinum* is the most common cause of dermatophytosis in horses. It has also been reported in dogs, cats, goats, and sheep.
- *Trichophyton mentagrophytes* is common in many species, such as cattle, horses, pigs, dogs, cats, and especially rabbits, rodents-including guinea pigs. *T. mentagrophytes* var. *erinacei* occurs in the European and African hedgehog.
- *Trichophyton verrucosum* is the most common cause of dermatophytosis in cattle, goats, and sheep. It has been reported in horses, donkeys, and South American camelids.
- *Trichophyton simmii* affects non-human primates (monkeys), poultry and dogs.

Zoonotic potential: Dermatophytosis poses a risk to humans especially when working in shelters or multi-animal facilities. *M. canis* (dogs and cats), *T. verrucosum* (cattle), *Arthroderma benhamiae* (guinea pigs) and *Arthroderma vanbreuseghemii* (cats, dogs, rabbits, mice, and chinchillas) appear to have a more common frequency in humans. Occasionally *T. equinum* can be transmitted to humans. *T. rubrum*, the cause of athlete's foot in humans has been reported to cause reverse zoonosis in dogs and cats.

Distribution: Most species of dermatophyte are worldwide.

- *T. simii* is rarely seen outside of India
- *T. erinacei* geographical distribution includes Europe, East Asia, and New Zealand
- *M. persicolor* geographical distribution includes Europe and the USA
- The causative agent that predominates in any one particular area can vary depending on the climate, geographic locations, and other factors such as concentration or livestock, pets, or exotic animals present.
- Humid, warm, tropical, and subtropical areas appear to have higher incidence.

Incubation period: The infective form of dermatophyte is the arthrospore. Infection can be established within hours after exposure, though clinical signs generally occur 1-3 weeks after exposure in animals and 4-14 days in humans.

Clinical signs:

Canine/Feline- Variable pruritus, though when complicated by secondary bacterial infection pruritus is more commonly noted. *Trichophyton mentagrophytes* can be extremely pruritic and mistakenly diagnosed as allergic disease. Focal to locally extensive areas well demarcated expanding alopecia with scale, crust, and follicular papules; these lesions are often expansive with chronicity. Facial lesions are common, though any of area of the body can be affected. Fungal kerions are another manifestation of disease characterized by an exudative, well circumscribed nodular furunculosis seen more commonly on limbs and the face of animals; these are often associated with dermatophytosis in dogs.

Equine- One or more circular patches of erythematous alopecia with scaling and crusting. Early lesions can appear as papular urticaria. Lesions are most often seen in the saddle and tack areas (thorax, head, and shoulders). Pruritus is usually minimal, but occasionally severe (suggestive of ectoparasitism).

Bovine- Non-pruritic periocular lesions (mostly in calves), discrete patches of alopecia with scaling white-grey crusts; papules and nodules can be present as well. Fungal kerions can be seen in cattle. Lesions are most commonly seen on the head, neck, and pelvis. For bulls the dewlap and intermaxillary space will often be affected.

Caprine/Ovine- Pruritus is rare. Alopecia, scale, erythema, and yellowish-grey crusting most often seen on the face, pinna, neck, and limbs. Udders and teats can be affected.

Porcine- Lesions are often diffuse, but seen mostly behind the ears and on the trunk. Annular areas of red to brown discoloration with superficial orange-brown crusting are noted. Alopecia and pruritus are rare.

Pocket Pets- Pruritus is common. The face, neck and limbs are commonly affected by areas of alopecia with scale and crust. Ears are often affected in rabbits.

Reptiles- Blisters that rupture into brown/yellow crusts can be seen. Other manifestations include proliferative growths or nodules that are often described as appearing 'necrotic'.

Avian- Alopecia (loss of feathers, though feathers are not infected) with scale and white crusts. Hyperkeratosis

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or white plaque formation can be seen. Occasionally feather plucking and self-mutilation occur.

Post mortem, gross, or histologic findings: Post-mortem and gross findings are the same as clinical signs and skin lesions present ante-mortem.

•The most common histopathological findings include:

- 1) perifolliculitis, folliculitis, and furunculosis more specifically, infiltrative lymphocytic mural folliculitis, suppurative luminal folliculitis, and pyogranulomatous furunculosis
- 2) hyperplastic or spongiotic superficial perivascular or interstitial dermatitis with prominent parakeratotic or orthokeratotic hyperkeratosis of the epidermis and hair follicles
- 3) intraepidermal pustular dermatitis (suppurative, neutrophilic epidermitis)

•Arthroconidia and hyphae can be detected in hair shafts with H&E staining, but special staining such as Periodic acid-Schiff (PAS) and Grocott methenamine silver (GMS) lends for an easier detection.

Diagnosis: There is no one gold standard diagnostic test for dermatophytosis; diagnosis includes multiple complementary techniques.

•Woods lamp examination will not provide a definitive diagnosis for dermatophytosis. This technique is helpful in identification of infected hairs, thus allowing a clinician to pinpoint the best area to pluck hairs or brush lesions for cytological evaluation and culture sampling.

•Trichogram and cytological evaluation of infected hairs and/or scale can reveal arthrospores and hyphae in 40-70% of cases and provides a preliminary diagnosis.

•Dermoscopy has recently been used in cats as a non-invasive diagnostic tool. On evaluation variably amounts of yellow to brown crusts are common and slightly curved or broken hairs with a homogenous thickens named “comma hairs” are common.

•Fungal culture is commonly used for diagnosis of dermatophyte. A Mackenzie brush technique utilizing a soft bristle toothbrush is ideal for collection of samples; 20 brush strokes, 2-3 minutes of brushing, or until the brush is full of hair should be achieved when collecting samples. Sabouraud’s dextrose agar or Dermatophyte Test Media (DTM) are the most reliable culture plates to confirm dermatophytosis. Specific dermatophyte species can be determined by assessment of macroconidia on cytological evaluation of colony growth from culture plates.

•It is important to note that *T. equinum* requires nicotinic acid (vitamin B3) for growth on fungal cultures.

•Polymerase Chain Reaction (PCR) has become a common and expedient diagnostic tool for evaluation of dermatophytosis. False positives may occur due to fomite carriage or detection of non-viable dermatophyte organisms after recent treatment. False negative results may be due to poor sample collection or marker used for detection does not correlate to specific dermatophyte species (i.e. only *Microsporum* and *Trichophyton* spp. are currently available for PCR testing).

•Skin biopsy is not often used, but can be helpful when fungal kerions are present, negative culture or PCR results occur.

*It is important to note that ectoparasites, bacterial pyoderma, and *Malassezia* dermatitis should be ruled out when approaching diagnostic testing for dermatophytosis.*

Material required for laboratory analysis: Infected hairs and/or scale; samples for infected hairs/scale can be detected using Wood’s lamp evaluation, trichogram, or cytological evaluation.

•Test media such as Sabouraud’s dextrose agar, DTM, Mycobiotic Agar (Difco, Detroit, MI), Mycosel Agar (BBL, Cockeysville, MD), Sab-duets (Hardy Diagnostics, Mountainview, CA), and Derm Duet (Hardy Diagnostics, Mountainview, CA) can all be used for culture.

Relevant diagnostic laboratories: Most clinics/clinicians will grow and review cultures in-house. Most commercial laboratories (Idexx, Antech) and veterinary schools will offer culture. Idexx laboratories offers PCR testing for *Microsporum* and *Trichophyton* spp.

Treatment: End point of treatment is considered with two or three negative culture (or PCR) results at consecutive weekly (2-4 weeks) intervals occur, this is considered a mycological cure.

•Clipping the hair, especially in long coated cats or severely infected animals, should be performed. This is

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particularly recommended in multi-cat facilities. In single animal households it is not necessarily needed. This decreases the burden of infection and allows for easier topical application of shampoos. Clipping the hair can also reduce chances of false positive results on culture and PCR.

- Effective topical agents include lime sulfur (1:16 dilution), 2% combined miconazole/chlorhexidine (1:1 ratio) formulations, and 0.2% enilconazole formulations. The animal's entire body should be treated twice weekly with topical therapies, allowing contact time for 10 minutes each treatment.
- In large animals 0.5% sodium hypochlorite (1:10 dilution of household bleach) can be used as topical therapy, but can be caustic to the skin.
- Systemic antifungal therapy includes griseofulvin, itraconazole, terbinafine, fluconazole, and ketoconazole. When considering systemic antifungal treatment it is imperative to note that compounded antifungal drugs have been shown to be inconsistent in dose, stability, and efficacy, therefore are not recommended. Itraconazole and terbinafine appear to have the best efficacy in treating dermatophytosis. Other systemic treatment options can be used though close monitoring for side effects, such as hepatic toxicity, is recommended.
- In horses, other than griseofulvin, no other antifungals are approved for oral use in the United States.
- The dose of griseofulvin for large animal species is widely variable. There is high evidence of spontaneous resolution; therefore, often in large animal species dermatophytosis will be left untreated.
- Itraconazole, fluconazole, ketoconazole, and terbinafine are often used in small animals (dogs and cats). Itraconazole is the treatment of choice for cats. There are varying dosages, duration, and regimens available for all the azole drugs. Pulse regimens with itraconazole and terbinafine have also been shown to be effective in some species. Itraconazole and terbinafine are most commonly used for pulse therapy such as one week on and one week off, or 2 days/week.
- Overall, treatment for generalized or severe dermatophytosis in all animals should include a combination of systemic and topical antifungals. If lesions are minimal, less than 2-3 lesions, then considering topical therapy alone is valid., or spontaneous resolution.

Prevention and control: Arthroconidia can remain viable in the environment and be infective for months to years, though studies have shown over time viability decreases.

- With the exception in cattle and horses, vaccines are not efficacious at preventing disease in other species. In Europe (Soviet Union and Scandinavia), a modified live *Trichophyton verrucosum* vaccine for cattle and modified live *Trichophyton equinum* vaccine for horses have shown to be effective. The vaccine is administered intramuscularly in calves at one and three weeks of age and in horses intramuscularly twice at 14 day intervals. The vaccine for cattle can have protection against *T. verrucosum* for up to 4-5 years.
- Control of dermatophytosis includes proper hygiene, routine disinfection of facilities, tools, housing, bedding, and toys, reducing fomites by using proper protective gear, limit handling and number of people handling infected patients, and isolation of infected patients.
- Vacuuming/sweeping facilities helps to remove any dander, scale, or infected hairs that could be lingering in the environment. This is considered a mechanical clean to remove any organic debris harboring arthroconidia. After removal of debris, disinfection is necessary to kill remaining arthroconidia.
- In equine medicine, tack and riding gear used in infected horses should be solely used in those individuals and properly disinfected or disposed of after use.
- Isolation of newly acquired animals for 2-4 weeks is recommended. Fungal culture and/or fungal PCR of all newly acquired animals are recommended to reduce asymptomatic carriers.
- Clipping the fur of infected animals and proper disposal can help reduce the amount of arthrospores introduced into the environment.

Suggested disinfectant for housing facilities:

- Environmental cleaning is aimed at reducing transmission of disease to animals and humans, minimizing fomite carriage, and shortening the course of unnecessary treatment.
- There are three major steps in decontaminating housing facilities:
 - 1) Mechanically remove all debris, fur, and fomites from facility (as described above).

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- 2) Thoroughly wash all surfaces with water and detergent.
- 3) Disinfect with 5.25% sodium hypochlorite (chlorine bleach) at a 1:100 dilution is effective and less irritating to humans and animals. This should be allowed to sit for 10 minutes. The diluted bleach solution does not retain efficacy over time and is recommended to be made new weekly.

•Other topical disinfectants include accelerated hydrogen peroxide, 1% Formaldehyde Solution (Formalin) and Enilconazole Environmental Spray (concentrate diluted to 0.2%). Household cleaners labeled to be effective against *Trichophyton* spp. can also be effective with 10 minute contact time.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Healthy non-infected animals should never be introduced to infected animals. As stated above, contact alone does not always result in disease, but exposing a healthy animal to an infected animal is not recommended.

•Animals should only be allowed to interact once mycological cure has been achieved.

•*Trichophyton verrucosum* and *Trichophyton equinum* modified live vaccines in Europe both show efficacy at preventing disease in cattle and horses respectively. All other vaccines have not been shown to be efficacious in other species.

Conditions for restoring disease-free status after an outbreak:

•Treating all infected animals and achieving mycological cure.

•Disinfecting housing facilities adequately (see above).

•Following isolation protocols for newly acquired animals.

Experts who may be consulted:

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DIROFILARIA IMMITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Many mammal species: Dogs Cats Ferrets	Female mosquito vector: >70 out of 3000 mosquito species world-wide; 16 species east of the Mississippi, three on the California coast, including <i>Aedes</i> , <i>Anopheles</i> , <i>Culex</i> , <i>Mansonia</i> , and <i>Psorophora</i> spp.	Dogs: Tricuspid regurgitation murmur, right heart failure, pulmonary hypertension, pulmonary thrombo-embolism, jugular pulses, allergic pneumonitis, ascites, hemo-globinemia, and hemo-globinuria (caval syndrome). Cats: Pulmonary granulomas, dyspnea, chylothorax, blindness, tachycardia, syncope. Ferrets: Anorexia, cough, weakness, dyspnea, bilirubinuria	Dogs: Asymptomatic or mild, may progress to fatal Cats: Asymptomatic to fatal; possible spontaneous cure with no treatment Ferrets: Potentially severe, >4 worms can be fatal	Dogs: Melarsomine dihydrochloride Cats: Symptomatic treatment or surgical extraction only Ferrets: Injectable moxidectin, if available	Dogs: Macrolytic lactones—monthly oral ivermectin, milbemycin oxime, or moxidectin; or monthly topical moxidectin or selamectin; or parenteral moxidectin every 6 months. Mosquito control, keep animals indoors Cats: Same as dogs Ferrets: Liquid ivermectin, topical moxidectin	Yes, but rare

Fact Sheet compiled by: Andrew Moorhead

Sheet completed on: Updated 14 Jan 2019

Fact Sheet Reviewed by: Elizabeth Arnett-Chinn

Susceptible animal groups: Mammals. Dogs 100% susceptible. Cats 61-90% susceptible. Domestic dog and wild canids (wolf, coyote, fox), and possibly Eurasian otter, are definitive hosts. Raccoons, wolverines, coyotes, deer, and bears are wildlife reservoirs. Documented in the rabbit, ferret, river otter, muskrat, harbor seal, sea lion, red panda, Japanese raccoon dog, wild cat, black-footed cat, golden cat, bobcat, ocelot, clouded leopard, snow leopard, African leopard, tiger, African lion, American black bear, polar bear, horse.

Causative organism: *Dirofilaria immitis*, a nematode intravascular parasite, that lives in bloodstream of host, normally pulmonary vessels

Zoonotic potential: Occasionally occurs and usually causes pulmonary dirofilariasis; in Florida, 100 cases were documented in the last 40 years.

Distribution: Diagnosed in 48 contiguous states plus Hawaii and US territories and worldwide.

Incubation period: Prepatent period at least 6-7 months in definitive host, 7-8 months in cat. Temperature dependent maturation of organism in mosquito occurs >57°F. In mosquito: ingested L1 (microfilariae) molt

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into L2 in 8-10 days post-infection, molt to L3 in 2-3 days after second molt, then migrate to mouth parts in 1-2 more days. Total development time can be as short as 14-15 days. In dog: L3 injected into host by mosquito molt to L4 in 3-12 days in skin, molt to juvenile adult heartworm in subcutaneous tissue and muscle in 50-70 days, migrate to heart via vascular system by day 70-120. L1 (microfilariae) discharged by mature nematodes 6-9 months post infection and can survive up to 2-3 years in the bloodstream. Worm longevity: 5-7 years in dog, 2-3 years in cat. Clinical signs may not appear for one year after infection.

Clinical signs: Lethargy, weakness, fatigue, exercise intolerance, dyspnea, cough, anorexia, weight loss, vomiting, diarrhea, collapse, seizures, sudden death. Humans: aberrant host--worms do not reach adult stage--no microfilaremia.

Post mortem, gross, or histologic findings: Female nematodes <12", males <7", microfilariae <1/800". Worms found in lobar arteries and main pulmonary artery when mild (e.g., 10 worms); right atrium and caudal vena cava, and rarely the right ventricle when severe (e.g., >40 worms). Dogs: 1-250 worms. Cats: 1-3 worms. Rabbits: aberrant host--granulomatous lung nodule reported. Humans: "coin lesion" in lungs, can be confused radiographically with carcinoma.

Diagnosis: In dogs and exotic species, antigen test (most sensitive, nearly 100% specific) detects adult female *D. immitis* protein >5-7 months post-infection. Cats: Both antigen and antibody tests preferred. Ancillary tests: Modified Knott or filtration test for microfilariae to differentiate *D. immitis* from *Acanthocheilonema* (formerly *Dipetalonema*) *reconditum*, thoracic radiography, ultrasonographic visualization of worms.

Material required for laboratory analysis: In-house antigen blood testing simple and inexpensive for dogs. Blood tubes for both antigen and antibody testing for cats.

Relevant diagnostic laboratories: Any veterinary diagnostic laboratory that performs the diagnostic testing.

Treatment: Dogs--arsenical compound: melarsomine dihydrochloride--only effective on worms >120 days old. Maximum 98% efficacy on adult worms. Adjunct therapy: Pretreatment with macrocyclic lactone 8-12 weeks to eliminate migrating larvae <60 days old and allow larvae 60-120 days old to reach melarsomine-susceptible age. Doxycycline 10 mg/kg bid for 4 weeks to reduce inflammation from filarial-associated *Wolbachia*. Surgical extraction of adult heartworms in acute caval syndrome. Cats: adulticide treatment not recommended. Symptomatic: prednisolone, bronchodilators. Surgical removal via right jugular venotomy or right ventriculotomy. Extreme caution must be exercised with melarsomine in exotic carnivores due to narrow margin of safety.

Prevention and control: Dogs and cats-- macrocyclic lactones: ivermectin/pyrantel (Heartgard Plus--Merial, dog and cat; Iverhart Plus--Virbac; Tri-Heart--Merck); milbemycin oxime/lufenuron (Sentinel--Novartis); moxidectin (Advantage Multi--Bayer, dog and cat; ProHeart 6;--Zoetis, dogs only); selamectin (Revolution--Zoetis)--all against L3, early L4; microfilariae--milbemycin oxime (off-label at preventive dose). Preventives have some efficacy against adult heartworms, but studies have mostly been performed with ivermectin/pyrantel compounds. Efficacy declines in late stages of L4. As of this writing, resistance to heartworm preventives has been proven; however, it does not appear to be a concern except in the Mississippi Delta region.

Suggested disinfectant for housing facilities: Not applicable

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: None, although presence of mosquitoes will increase risk to non-infected individuals.

Conditions for restoring disease-free status after an outbreak:

Treat affected individuals, eliminate microfilariae pre-treatment (Topical moxidectin/imidicloprid- FDA-labelled for microfilariae elimination. Milbemycin oxime 500 ug/kg or ivermectin at 50 ug/kg will also result in clearance of microfilariae.), mosquito control

Experts who may be consulted:

American Heartworm Society

DIROFILARIA IMMITIS

P.O. Box 8266
Wilmington, DE 19803-8266
info@heartwormsociety.org
<http://www.heartwormsociety.org/>

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EASTERN EQUINE ENCEPHALOMYELITIS (EEE)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Birds, equids, and occasionally other mammals	Mosquito (<i>Culiseta melanura</i>)	Febrile, altered mentation, neurologic abnormalities, seizures, paresis, paralysis, death	Equine fatality rate is up to 90%; survivors usually exhibit long-term neurologic signs; human fatality rate is 50-75%	Supportive care	Formalin-inactivated whole viral vaccine, insect control	Yes; however, not believed to transmit from horses as viremia is too low

Fact Sheet Compiled by: Erica Lipanovich

Completed on: updated 10 October 2017

Fact Sheet Reviewed by: Elizabeth Arnett-Chinn

Susceptible Animal Groups: Birds are the principal enzootic hosts. Clinical cases occur in equids and occasionally other mammals, including swine, cows, rodents and opossums. Mammals are almost always dead-end hosts. Snakes, turtles and fish are suspected to be an amplifier or over-wintering reservoir.

Causative Organism: Eastern equine encephalomyelitis virus (Family Togaviridae, genus Alphavirus). There are four lineages of EEE. Group I is endemic in North American and the Caribbean and causes most of the human cases. The other three groups (IIA, IIB, III, and IV) cause primarily equine illness in South or Central America and are now classified as the Madariaga virus.

Zoonotic Potential: Mosquito bites from *Culiseta melanura* is the important vector in the maintenance cycle in birds. The majority of isolates have been found in 27 species of mosquitoes (e.g., some *Aedes*, *Coquillettidia*, and *Culex* species).

Distribution: Western Hemisphere - North American variant is found in eastern Canada, all states east of the Mississippi River, Arkansas, Minnesota, South Dakota, Texas, and the Caribbean islands. The South American variant is confined to central and South America.

Incubation Period: 4 to 10 days, and rarely up to 3 weeks.

Clinical Signs: Equids frequently include altered mentation, impaired vision, aimless wandering, head pressing, circling, anorexia, grinding of teeth, esophageal paralysis, irregular or ataxic gait, paresis, paralysis, seizures, coma and death. Many horses progress to recumbency within 12-18 hours of onset of neurological abnormalities. Most deaths occur within 2-3 days after onset of signs. Mortality of equids with clinical signs is 50-90%.

Most people infected have no apparent illness. Two types of illness can develop in humans, systemic or encephalitic. Systemic infection has an abrupt onset of malaise, fever, chills, arthralgia and myalgia lasting one to two weeks. Recovery is complete if there is no CNS involvement. Encephalitic illness can be abrupt or become present after a few days of systemic illness, such as fever, headache, irritability, restlessness, drowsiness, anorexia, vomiting, diarrhea, cyanosis, altered reflexes, convulsions, and coma. One third of all EEE human cases usually die within 2 to 10 days after onset of symptoms. Those persons who recover have irreversible neurological damage.

Birds: Most cases are asymptomatic but fatal outbreaks have occurred in emus, game birds such as pheasants, whooping cranes, passerines and psittacines.

Post mortem, Gross or Histological Findings: Gross lesions are rare but congestion may be present in the meninges of acutely affected animals. Histologic findings are typical of encephalomyelitis which include severe gliosis with necrosis of the neuropil in the cerebrum and through the corona radiata to the thalamus and perivascular cuffing throughout the mid and hindbrain and cervical spinal cord.

Diagnosis: Clinical presentation in an endemic area, EEEV-specific IgM antibody in serum or cerebrospinal fluid samples (CSF), and confirmed by neutralizing antibody testing of acute and convalescent phase serum specimens.

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Material Required for Laboratory Analysis: Serum and CSF samples are collected from live animals to detect virus-specific IgM and neutralizing antibodies. Brain, spinal cord, and other tissues may also be collected from necropsied animals.

Relevant Diagnostic Laboratories: Only a few state laboratories or other specialized laboratories are capable of doing the testing. National Center for Emerging and Zoonotic Infectious Diseases (NCEZID) through the CDC. <https://www.cdc.gov/ncezid/dvbd/specimensub/arboviral-shipping.html>. Accessed 2 October 2017. The state health departments must be notified upon submission.

Treatment: Supportive and symptomatic care.

Prevention and Control: There is no vaccine for humans. Reducing exposure to mosquitoes, mosquito control, and vaccination in equids. Vaccination of captive, at risk bird species birds in areas high virus activity is often practiced. No cross-immunity obtained when vaccinated for other alphaviruses (e.g., western equine encephalitis virus) or flaviviruses (e.g., West Nile virus) or bunyaviruses (e.g., La Crosse virus).

Suggested Disinfectant for Housing Facilities: Clean infected environment with an approved EPA disinfectant.

Notification: Suspected cases are reported according to individual State procedure, typically by notification of the State Arboviral Coordinator or State Animal Health Official. Reports of positive equine cases of arboviral disease are reported to ArboNET, an internet-based arbovirus surveillance and reporting system managed by state health departments and the Centers for Disease Control and Prevention. ArboNET captures laboratory-confirmed positive cases in humans, horses, other mammals, birds, and mosquitoes across the U.S. Equine cases vary by state, but those reported to ArboNET are confirmed by State Veterinarians prior to reporting.

Measures Required under the Animal Disease Surveillance Plan: In most states, reporting is mandatory.

Measures Required for Introducing Animals to Infected Animal: Maintain infected animal in a quarantine situation. Do not introduce infected animal to an animal with a compromised immune system.

Conditions for Restoring Disease-Free Status after an Outbreak: Clean infected environment with diluted bleach to the extent possible. Minimize contact of infected staff with animal.

Experts Who May Be Consulted:

USDA, APHIS, Veterinary Services
4700 River Road, Unit 41
Riverdale, MD 20737-1231
Telephone (301) 734-8093
Fax (301) 734-7817

www.aphis.usda.gov/animal_health/index.shtml

Center for Disease Control and Prevention
Public Health Resources:

State or Territorial Health Departments

<https://www.cdc.gov/mmwr/international/relres.html>

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RESTON EBOLA VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Macaque, swine Fruit bats are a possible reservoir	Direct contact with infected animals, secretions or droplets Possible aerosol transmission in pigs	Anorexia, lethargy, diarrhea or melena with frank blood, bleeding from external orifices, petechial to suffusive hemorrhage	Macaque: Fatal Swine: Can vary from subclinical to severe	Isolation of unaffected animals No successful clinical treatment	Quarantine of imported primates in country of origin and in import facilities in the US	Whereas the Ebola genus viruses are known to cause disease in humans, this one is not.

Fact Sheet compiled by: Dawn Zimmerman

Sheet completed on: 27 December 2017

Fact Sheet Reviewed by: Tim Georoff

Susceptible animal groups: Cynomolgus monkeys (*Macaca fascicularis*) were identified in the index case in 1989 and subsequent outbreaks among animals imported from the Philippines in 1990, 1992 and 1996. An additional outbreak has been documented at a lab facility in the Philippines in 2015. Reston ebolavirus virus has also been isolated from swine in the Philippines and China, which were co-infected with porcine reproductive and respiratory syndrome virus (PRRSV). It is unclear if swine are an incidental host or part of the virus' transmission cycle. Bats in the Philippines, Bangladesh, and China have tested seropositive; however, their epidemiological role is unknown.

Note: African green monkeys (*Chlorocebus aethiops*) and baboons (*Papio hamadryas*) are resistant to both RESTV infection.

Causative organism: Reston virus (RESTV) species *Reston ebolavirus*, family Filoviridae.

Zoonotic potential: Humans exposed to the disease in primates and swine have become seropositive but have no apparent or clinically mild infection. It is unknown how infection would affect immune-compromised people, pregnant women, or children.

Distribution: Philippines (and animals recently imported from Philippines), China, Bangladesh. Geographic distribution may be larger depending of the reservoir distribution.

Incubation period: 7-14 days.

Clinical signs: In primates: Anorexia, lethargy or sudden death may be the only signs. Fever, cough, nasal exudates, swollen eyelids, splenomegaly, and renomegaly can occur. Animals may also show signs of hemorrhagic fever with diarrhea or melena with frank blood, bleeding from external orifices, petechial to suffusive hemorrhage.

Post mortem, gross, or histologic findings: Maculopapular rash, splenomegaly, widespread petechial hemorrhages, hemorrhage in proximal duodenum, and interstitial pneumonia are observed grossly. Lymphoid necrosis, massive fibrin deposition in spleen, hepatic necrosis, necrosis of adrenal cortex and pulmonary bronchiolar and alveolar epithelium, interstitial nephritis, and amphophilic cytoplasmic inclusion bodies in many tissues including liver, adrenal gland, and spleen are observed histologically. Extensive viral replication in tissue macrophages and interstitial fibroblasts.

Diagnosis: In blood during acute phase: ELISA, RT-PCR (rapid, more sensitive than antigen detection ELISA, and allows identification of the virus species), virus isolation (requires a BSL-4 lab), IgG/IgM, immunohistochemical staining and histopathology on post-mortem or collected tissues to localize viral antigen. Biosafety concerns during the collection and processing of the specimens.

Material required for laboratory analysis: Testing liver samples by ELISA antigen capture is the mandatory test for confirmation or ruling-out the diagnosis in suspected dead primates during quarantine (<https://www.cdc.gov/ncepid/dhcpp/vspb/pdf/primate-form-508.pdf>).

RESTON EBOLA VIRUS

Relevant diagnostic laboratories:

Viral Special Pathogens Branch
Centers for Disease Control and Prevention
1600 Clifton Rd
Atlanta, Georgia 30333
Phone: 470-312-0094

Treatment: Based on epidemiology from prior outbreaks it appears that virus spread through group-housed animals is unavoidable. Since asymptomatic animals may be present in groups during the incubation period, strict depopulation is likely the best course of action.

Prevention and control: Pre-shipment quarantine of primates to be shipped from Philippines. CDC licensed primate import quarantine facility with special permit required for *Cynomolgus* monkeys, rhesus, and African green monkeys. Diagnostic testing of potentially affected animals, personal protective equipment to prevent exposure of personnel and close coordination with importer and CDC. Strict isolation of groups of imported animals.

Suggested disinfectant for housing facilities: Hypochlorite or phenolic disinfectants are generally recommended for disinfection. Ebola virus is susceptible to 2% sodium hypochlorite, 2% glutaraldehyde, 5% peracetic acid, 1% formalin.

Notification: CDC

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Depopulation of affected group and premise disinfection.

Experts who may be consulted:

Centers for Disease Control and Prevention (CDC) Viral Special Pathogens Branch or Division of Global Migration and Quarantine (DGMQ).

May be contacted 24 hours a day through the CDC Emergency Operations Center (770-488-7100).

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RESTON EBOLA VIRUS

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ECHINOCOCCOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals	Intermediate (IH)/accidental (AH) hosts: Fecal-oral Definitive (DH) host: predation or ingestion of infected IH/AH	IH/AH: Abdominal distention and pain, lethargy, inappetence, cough, dyspnea	IH/AH: Initially subclinical but cyst growth can lead to hepatic and respiratory disease which may be fatal	IH/AH Benzimidazoles or percutaneous drainage of hepatic cysts DH: Praziquantel	Eggs susceptible to desiccation and extreme temperatures; avoid feeding potentially-infected carcasses; anthelmintic bait (praziquantel) for DH; dog population management; education; EG95 vaccine	Yes

Fact Sheet compiled by: M. Camille Harris

Sheet completed on: 19 March 2011; updated 20 March 2013

Fact Sheet reviewed by: Malika Kachani, Philip Craig, Linda Pote

Susceptible animal groups: Mammals: primates (Old World monkeys and great apes) – including significant number of primate cases in the literature; ungulates; marsupials; rodents; canids

Causative organism: Primary transmission cycles include:

E. granulosus complex (EG; wolf-cervid; canid-livestock) molecular species include:

- *E. granulosus sensu stricto* [sheep, Tasmanian sheep and buffalo strains]
- *E. equinus* [EE; horse strain]
- *E. ortleppi* [EOr; cattle strain]
- *E. canadensis* [EC; camel, pig, and cervid strains]
- *E. felidis* [EF; lion strain]

E. multilocularis (EM; fox/canid/felid/rodent)

E. oligarthrus (EOl; felid-agouti/paca)

E. vogeli (EV; bush dog-paca)

E. shiquicus (ES; Tibetan fox-pika)

Zoonotic potential: Yes. Humans are susceptible by ingesting shed *Echinococcus* eggs (EG, EC, EM, EOl, EV, EOr).

Distribution:

EG: Worldwide; EM: Northern Hemisphere

EOl and EV: Central and South America; ES: Qinghai-Tibet plateau of China

Incubation period:

IH/AH incubation period: Months (e.g., rodents) to years (e.g., primates) depending on hydatid cyst location and growth rate; DH prepatent period: EG (32-80 days); EM (28-35 days)

Clinical signs: Larval metacestode infections of IH/AH are initially subclinical and signs may not develop during the host's life span. Clinical signs are related to cyst location, which is most often the liver and lungs.

ECHINOCOCCOSIS

As cysts develop, signs may include lethargy, abdominal pain, abdominal enlargement (due to hepatomegaly), inappetance, and respiratory signs. Cyst rupture may lead to anaphylaxis. EM is most likely to eventually cause clinical disease after a 5-15 year asymptomatic period. No clinical signs in DH are noted.

Post mortem, gross, or histologic findings: Adult cestodes (body length: 1.2 - 11 mm) are found in the small intestine of DH with EG primarily in the upper third and EM in the middle third. The formation of fluid-filled cysts is primarily in hepatic and pulmonary tissues, but can occur in any organ of IH/AH. EG and EOI are usually associated with a single cyst. Similar to a metastasizing neoplasm, EM and EV form masses of small cysts. Viable protoscolices may be present within cysts.

Diagnosis:

Antemortem: Imaging may be used to identify and classify fluid-filled cysts in IH/AH (see Table 2.5, Eckert et al. 2001) along with a cytologic exam of FNA. Fecal diagnosis in DH is difficult due to the small size of gravid proglottids (1-2mm) and inability to differentiate from eggs of *Taenia* species. Fecal coproantigen ELISA and confirmatory *Echinococcus* PCRs have been developed for DH. Serum antibody ELISAs have been developed but *Taenia* false positives may occur and species validation is limited. Percutaneous drainage of cysts may reveal the presence of protoscolices.

Postmortem: Fluid-filled cysts may be seen at necropsy (primarily hepatic and pulmonary). In IH/AH, histopathology may reveal the presence of protoscolices within brood-capsules or in hydatid sand. In DH, the scraping, filtration and counting technique can be used to extract cestodes from the intestines.

Material required for laboratory analysis: cyst fluid, serum (IH/AH); feces, small intestine (DH)

Treatment: In humans, treatment options include surgical removal of cysts and ultrasound-guided partial removal of cyst fluid and injection of anthelmintic (PAIR – puncture, aspiration, injection, reaspiration). Alternatively, anthelmintics (benzimidazoles) can be used for IH/AHs. Degenerating cysts may not require treatment and monitoring would be an option.

Prevention and control: Infective material can be decontaminated by extreme temperatures (70°C for 12 hrs; -80°C for 48 hrs). When handling infective material, personal protective equipment should be worn to reduce the risk of human exposure. Ensure the DH's diet does not include potentially infected organs and carcasses. Prevent scavenging and predation by susceptible mammals. Decontaminate foliage or branches used for environmental enrichment. Pet and feral dog population management and deworming. Education of animal care workers, dog owners and other at-risk human populations. Anthelmintic baiting of foxes (50mg praziquantel/bait). EG95 vaccine has been shown to be protective against EG in sheep, goats, cattle and tammar wallabies.

Suggested disinfectant for housing facilities: Chemical disinfection is unreliable but $\geq 3.75\%$ bleach (NaOCl) solution for 1 hr (metal surface) or 2-3 hrs (concrete) may be effective for EM. Facilities can be decontaminated by 40% relative humidity and 30°C for at least 48 hrs.

Notification: Echinococcosis is an OIE reportable disease and USDA should be notified.

Experts who may be consulted:

Dr. Philip S. Craig

Cestode Zoonoses Research Group, School of Environment and Life Sciences, University of Salford
Manchester M5 4WT, UK.

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ECHINOCOCCOSIS

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EDWARDSIELLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Several teleost species, especially in warm water.	Unknown, probably oral.	Septicemia, enteritis, skin ulcers, petechiae.	Variable, can be severe with concurrent stressors.	Systemic antibiotics based on culture and sensitivity and regulations.	Remove stressors; improve disinfection.	Yes.

Fact Sheet compiled by: Catherine Hadfield

Sheet completed on: 28 November 2013; updated 6 July 2013

Fact Sheet Reviewed by: Leigh Clayton, Lester Khoo

Susceptible animal groups: Various teleost species, usually those found in warm water. Common in American, European, and Japanese eels, channel catfish, carp, bass, Japanese flounder, and many tropical marine teleosts. It can also cause disease in some invertebrates, amphibians, reptiles, and mammals.

Causative organism: Enterobacteriaceae, *Edwardsiella tarda* and *Edwardsiella piscicida*. These species cannot be differentiated phenotypically, so earlier reports of *E. tarda* may represent *E. piscicida*. Molecular diagnostics are required for differentiation. Other strains may be identified in the future.

Zoonotic potential: Yes. Usually necrotic skin wounds or gastroenteritis but it can spread systemically.

Distribution: Worldwide.

Incubation period: 5-7 days.

Clinical signs: Acute or chronic presentation may include: lethargy, inappetance, ulcers, hyperemia, petechiation, erythema, pale gills, coelomic distension, positive buoyancy, and ocular lesions (such as keratitis, uveitis, and exophthalmia). Mortalities tend to be low.

Post mortem, gross, or histologic findings: Congestion and/or focal necrosis of spleen, liver, kidney, and heart are observed. Malodorous abscesses in the viscera or skeletal muscle may be seen. Small, straight Gram negative rods which may be motile can be present. Inflammation, often suppurative but may be granulomatous, can be observed in infected organs, such as kidneys.

Diagnosis: Bacterial culture from lesions, blood or organs. PCR, DNA hybridization, or sequencing required for differentiation of *E. tarda* and *E. piscicida*. However, bacteria may be present in the gastrointestinal tract of healthy fish.

Material required for laboratory analysis: Blood culture can be performed. Tissue swabs or preferably tissue samples for bacterial culture, especially kidney. Samples should be transported at 4°C.

Relevant diagnostic laboratories: Most laboratories should be able to culture *Edwardsiella* spp., but further identification may require specialist fish laboratories.

Treatment: Removal of stressors is important for successful treatment and good supportive care should be provided. Water quality and disinfection should be improved. Systemic antibiotics, based on culture and sensitivity and relevant legislation, e.g., trimethoprim sulfa, florfenicol, can be used. However, as of June 2013, no FDA-approved medications are available for use in food fish. Immunostimulants, e.g., glucans, glycans, alginate, or ascorbic acid.

Prevention and control: Stressors (e.g., temperature, water quality, stocking density, and organic load) should be reduced in the environment. Water can be disinfected with UV or ozone. *E. tarda* vaccines are under trial.

Suggested disinfectant for housing facilities: Susceptible to most common disinfectants: 1% sodium

EDWARDSIELLOSIS

hypochlorite, 70% ethyl alcohol, iodophors, phenols.
Notification: None.
Measures required under the Animal Disease Surveillance Plan: None.
Measures required for introducing animals to infected animal: Introduction of animals should be avoided if clinical signs are present.
Conditions for restoring disease-free status after an outbreak: Not known; animals can carry the bacteria asymptotically.
Experts who may be consulted: Most fish clinicians will be familiar with Edwardsiellosis and can be consulted if an outbreak is encountered.
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***Ehrlichia ruminatum* (HEARTWATER)**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals - domestic and wild ruminants. Infections in other mammals (mice), and possibly (although not definitively proven) in reptiles, and birds.	Non-contagious tick borne disease via <i>Amblyomma</i> spp. ticks including US tick species: <i>A. maculatum</i> , <i>A. cajennense</i> , <i>A. dissimile</i> , <i>A. americanum</i> .	Acute – fever, anorexia, diarrhea, serosa or mucosa petechiae, respiratory and neurologic signs. Peracute – sudden death!	Subclinical to peracute death. Dependent on strain, host, and environment. Most common is acute clinical disease.	Remove ticks and antibiotics (e.g., tetracycline). Submit ticks for diagnosis using the pCS20 RT or nested PCR.	Tick control and test for carrier status in animals prior to translocations. “Vaccination” (see below) can be used in areas with endemic heartwater present.	No

Fact Sheet compiled by: Sharon L. Deem

Sheet completed on: updated 31 July 2018

Fact Sheet Reviewed by: Beth Bicknese

Susceptible animal groups: Ruminant species (domestic and wild) and other mammals (mice). Sheep and goats more susceptible than cattle and European breeds more susceptible than zebu type. Infections in birds and reptiles have not been confirmed.

Causative organism: *Ehrlichia ruminantium* (previously called *Cowdria ruminantium*). A small, intracellular, Gram negative, pleomorphic coccus bacteria found in endothelial cells, monocytes and neutrophils.

Zoonotic potential: Not definitively, although pCS20 sequences have been amplified in humans in South Africa.

Distribution: Endemic countries are on the African continent south of the Sahara, Madagascar, various small islands in the Indian and Atlantic Oceans, and islands in the Caribbean. A foreign animal disease for US, concern of entry is high due to illegal wildlife trade with infected ticks and the potential for domestic ruminants and white tailed deer in the US to serve as host species. All susceptible animals legally imported (e.g., zoo animals and stocking of exotic animal ranches) to the US from heartwater endemic regions may serve as a route of introduction of *E. ruminantium* to the American continents. Ticks on tortoises from Africa can carry infected ticks.

Incubation period: This period varies with species infected, route of infection, and strain of *E. ruminantium*. In domestic cattle, incubation is 12 days after intravenous injection of *E. ruminantium* “vaccine”. The period is shorter (e.g., 7 days) when more virulent strains are used. Incubation period of tick transmitted heartwater is 18-21 days.

Clinical signs: Severity ranges from subclinical infection to peracute disease. Clinical signs range from mild transient fever in subclinical cases to death without premonitory signs in peracute cases – i.e., presenting as sudden death. The acute form is characterized by rapid onset of fever (41.5° to 42°C), tachypnea, inappetence, petechiation on serosal and mucosal surfaces, and neurologic signs (e.g., hyperesthesia, high-stepping or unsteady gait, twitching eyelids, head pressing, chewing, abnormal tongue movement, individual muscle tremors). In domestic cattle and goats profuse, fetid, hemorrhagic diarrhea commonly occurs terminally.

Post mortem, gross, or histologic findings: Hydrothorax, pulmonary edema, ascites, hydropericardium (“heartwater”), cerebral edema, edema of the lymph nodes, and splenomegaly are observed. *E. ruminantium* found in brain endothelial cells lining capillaries as colonies in all animals that have died of heartwater. Rare to find colonies in brain smears of infection carrier animals.

***Ehrlichia ruminatum* (HEARTWATER)**

Diagnosis: Clinical signs in ruminants with known *Amblyomma* spp. tick infestations may be suggestive although a number of differential diagnoses must be considered due to the non-specific gastrointestinal and neurologic signs. In peracute cases, anthrax and peracute typanosomiasis are top differentials. In acute cases, rabies, cerebral babesiosis, cerebral theileriosis, tetanus, cerebral listeriosis, coccidiosis, arsenical or plant intoxication, hemorrhagic septicemia, and hemonchosis can be confused with *E. ruminantium*. Differential diagnoses are host species and geographical location dependent. Clinical pathologic changes are variable but may include progressive anemia, marked decline in thrombocytes, fluctuations in total and differential white cell counts, increased total bilirubin, and a decrease in total serum proteins. Serologic diagnostics for antibodies (e.g., ELISA and Western blot) and pathogen detection (e.g., DNA probes and pCS20 PCR) are available. It is recommended that MAP1B ELISA and the pCS20 PCR (nested or reverse transcript) be run on samples from animals with suspected *E. ruminantium* infection to detect both antibodies and the pathogen. (NB: Animals that die of heartwater will not have antibodies detected in the blood.) Definitive diagnosis (gold standard) is brain smears showing the organisms in endothelial cells that stain positive with Giemsa stain. In addition to the brain, organisms may be identified by light microscopy in kidney, lung, and heart tissue.

Material required for laboratory analysis: Brain tissue, *Amblyomma* spp. ticks, and blood or bone marrow collected in anti-coagulant.

Relevant diagnostic laboratories:

Submissions from suspect cases coming from the US:
 USDA-APHIS-VS National Veterinary Services Laboratory (NVSL)
 1920 Dayton Ave. (for packages)
 P.O. Box 844 (for letters)
 Ames, IA 50010
 Phone: (515) 337-7266
 Fax: (515) 337-7397

Submissions from suspect cases coming from foreign countries:
 Foreign Animal Disease Diagnostic Laboratory (FADDL), Plum Island, New York
 40550 Route 25 (for packages)
 Orient Point, NY 11957
 P.O. Box 848 (for letters)
 Greenport, NY 11944-0848
 Phone: (631)323-3256
 Fax: (631) 323-3366

Treatment: Limited value in clinically ill animals after the onset of neurologic or gastrointestinal signs. Administration of antibiotics (sulfonamides and tetracyclines) at the start of a febrile response may be successful.

Prevention and control: In regions free of heartwater (US), control depends on tick control (*Amblyomma* spp.) and regulation of animal movements (e.g., subclinical carriers). In endemic regions, control is dependent on maintenance of endemic stability through vaccination and strategic tick control. "Vaccination" (infection and treatment) is possible in endemic regions with intravenous injection of live *E. ruminantium* organisms and then intravenous administration of antibiotics started at first rise in body temperature. Hazard of live vaccination is that it most likely will induce carrier status. Inactivated vaccines are being developed and may soon be commercially available to minimize clinical signs but they do NOT prevent infection.

Suggested disinfectant for housing facilities: Organism is extremely fragile outside the host, losing its viability within hours. Tick control is backbone of heartwater prevention. Appropriate acaricides are important and proper quarantine periods when moving animals from heartwater endemic to non-endemic regions.

Notification: OIE list B notifiable disease.

***Ehrlichia ruminatum* (HEARTWATER)**

Measures required under the Animal Disease Surveillance Plan: Heartwater is a reportable foreign animal disease in the US to USDA-APHIS.

Measures required for introducing animals to infected animal: Although a non-contagious disease, all infected animals should be quarantined and treated with acaricides routinely. Risk of spread from infected ticks on carrier animals when animals (and their ticks) are introduced to new areas or when potential *Amblyomma* spp. vector ticks in heartwater free regions feed on carrier animals and become infectious.

Conditions for restoring disease-free status after an outbreak: Tick control and culling.

Experts who may be consulted:

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EHRlichiosis

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals	Mechanical, via vectors (tick-borne)	Non-specific: fever, depression, lethargy, thrombocytopenia, anemia, weight loss, muscle/joint pain, lymphadenopathy, hepatocellular enzyme abnormalities	Subclinical or mild illness to severe, potentially fatal disease	Tetracycline antibiotics (doxycycline is drug of choice)	Tick control, screened blood donors, inspect animals frequently in tick-infested areas	Yes

Fact Sheet compiled by: Dawn Zimmerman and Danielle R. Graham Snyder

Sheet completed on: 22 December 2017

Fact Sheet Reviewed by: Majorie Bercier

Susceptible animal groups: Mammals (reported in humans, canids, felids, bovids, camelids, cervids, equids, and rodents)

Causative organism:

Tick-borne bacteria (family: *Anaplasmataceae*): small, gram-negative, pleomorphic, obligate intracellular cocci that infect different blood cells in various animals, including humans.

- *Ehrlichia chaffeensis* (human monocytic ehrlichiosis), known reservoirs include white-tailed deer and dogs.
- *Ehrlichia ewingii* (canine granulocytic ehrlichiosis, CGE), known reservoirs include white-tailed deer and dogs.
- *Ehrlichia canis* (canine monocytic ehrlichiosis, CME), known reservoirs include dogs.
- *Ehrlichia ruminantium* (heartwater), known reservoirs include ruminants.
- *Ehrlichia muris*, known reservoirs include wild small rodents.
- Other (*Ehrlichia muris eaucloirensis*, formerly *E. muris*-like agent (EMLA), an emerging human pathogen in Midwestern US)

Note: *Ehrlichia risticii* has been reclassified as *Neorickettsia risticii* and *Ehrlichia platys* as *Anaplasma platys*. *Ehrlichia equi*, *Ehrlichia phagocytophila*, and Human Granulocytic Ehrlichial Agent are now considered to be the same species and have been reclassified as *Anaplasma phagocytophilum*.

Zoonotic potential: Yes, via vectors or mechanical transmission.

Distribution: Almost every state in the US has reported a case of ehrlichiosis. Most human cases occur in the south-central and southeastern US. *E. canis* is endemic in southern, eastern, south-central and in southwest US, and is mainly transmitted by the brown dog tick *Rhipicephalus sanguineus*. *E. ewingii* is found predominantly in southern and mideastern US and is mainly transmitted by the lone star tick *Amblyomma americanum*. *E. chaffeensis* occurs predominantly in the southeastern US and is also transmitted by *A. americanum*. Globally, *Ehrlichia* has been reported in South America, Asia, Africa, and Europe.

Incubation period:

Humans: 5-10 days after a tick bite.

Dogs: 8-20 days.

It is estimated that the infected tick must be attached to the host for 24-48 hours for transmission to occur. *Ehrlichia* can remain alive in the developing tick for up to 5 months. Acute infection develops 1-3 weeks after transmission and lasts ~2-4 weeks. After ~6-9 weeks, the organism is eliminated in an immunocompetent animal or a parasitemia develops with no clinical signs in the subclinical phase which can last from weeks to years or mild to severe clinical signs. If the animal cannot mount an effective immune response, the animal becomes chronically infected.

Clinical signs: Generally non-specific, multi-systemic: fever, depression, lethargy, thrombocytopenia, anemia,

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anorexia, weight loss, lymphadenopathy; hepatocellular enzyme abnormalities, possibly gastrointestinal signs (vomiting, diarrhea), polymyositis, polyarthritis, rash, ocular signs (uveitis or retinal petechiae), reproductive disorders, and neuropathies.

Clinical signs depend on the strain of *Ehrlichia*, dose of infection, species, immunological status of host, and concurrent infections with other tick-borne parasites.

In dogs, the acute phase is generally mild and causes immune-mediated platelet destruction and manifesting in lethargy, anorexia, lymphadenopathy, fever, and is often associated with the presence of ticks. In the subclinical phase, dogs appear normal with a somewhat reduced platelet count and elevated globulin levels; this phase can last months to years. In the chronic phase, clinical signs recur with up to 60% of infected dogs presenting with abnormal bleeding due to reduced platelet numbers; elevated globulin levels are almost always seen; uveitis, neurological effects, and glomerulonephritis can also result; most dogs do not show full pancytopenia.

Infections with *E. ewingii*, which primarily causes disease in the immunocompromised, tend to additionally produce arthritis.

Post mortem, gross, or histologic findings:

Gross: splenomegaly, hepatomegaly, and lymphadenopathy during acute phase.

Histologic: extensive plasma cell infiltration of parenchymal organs; perivascular cuffing particularly of the lungs, kidneys, spleen, meninges, and eyes.

Diagnosis:

- History of exposure and clinical signs (diagnosis of subclinical disease based on anamnesis, geographic location, persistent antibody titers, mild thrombocytopenia, and hypergammaglobulinemia).
- Morulae (intracytoplasmic bacterial aggregates) in monocytes on blood and buffy coat smears (Romanowski stain); however, often only seen in a small percentage of blood smears of infected dogs, and only found in the bloodstream for a few days in the acute stage.
- Enzyme-Linked Immunosorbent Assay (ELISA), e.g. IDEXX “snap 4DX” (includes Lyme disease and heartworm tests; detects *E. canis*, not *E. ewingii*) - not quantitative.
- Detection of *E. canis* serum antibodies with indirect Immunofluorescence Antibody Test (IFA), antibodies can be detected as early as 7 days post-infection, although animals may not be seropositive until 28 days post-infection. It takes 6-9 months after infection for titers to drop. Serologic cross-reactions may occur with other rickettsial agents.

With ELISA and IFA, a positive test only indicates exposure and does not imply active infection. A titer >1:80 is considered positive. If <1:80, considered suspect and should retest in 2-3 weeks (titers will increase rapidly in the acute stage, look for four-fold increase between paired serum samples or test again using PCR or Western blot). IFA and ELISA tests detect *Ehrlichia* species other than *E. canis*.

- Polymerase Chain Reaction (PCR, e.g. Antech FastPanel™ PCR Canine Ehrlichiosis/Anaplasmosis Profile for *E. canis*, *E. chafeensis*, and *E. ewingii*, cross-reacts with *Anaplasma*). PCR can detect *E. canis* in dogs within 4-10 days of exposure, *before* they become seropositive. PCR remains positive for several weeks after infection has cleared, as it does not distinguish between live and dead organisms. Peptide and recombinant antigens are available for *E. ewingii*; however, CGE diagnosis is usually made via visualization of morulae within neutrophils, PCR, ELISA, or Western immunoblot.

- Western immunoblot
- Demonstration of ehrlichial antigen in tissue sample by immunohistochemical methods, or *in situ* hybridization.
- Isolation of ehrlichial species from a clinical specimen in cell culture.

Material required for laboratory analysis:

Serology: serum taken within first week of illness, with second sample taken 2-3 weeks later. Retain acute-phase serum sample and submit two samples together at same time.

PCR: 0.5ml whole blood (EDTA), or biopsy specimens from organs such as lymph nodes, spleen, liver, or bone marrow

Sample blood prior to starting antimicrobial therapy to avoid false negative test results.

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Relevant diagnostic laboratories:

Antech (FastPanel™ PCR Canine Ehrlichiosis/Anaplasmosis Profile) and Zoologix (PCR, two tests: one is *E. canis* specific, other detects but does not differentiate most common *Ehrlichia* species). PCR panel for tickborne diseases which includes common *Ehrlichia* species.

NCSU diagnostic PCR—<https://www.cvm.ncsu.edu/vth/ticklab.html>

OSU diagnostic PCR and serology- <http://riki-lb2.vet.ohio-state.edu/ehrlichia/>

Treatment: Tetracycline antibiotic for at least one month - usually doxycycline which allows for a more convenient dosing schedule. Dramatic initial improvement usually observed within 24-48 hours. Treatment success should be based on remission of clinical signs, decline in *E. canis* antibody titers, and concurrent decrease in gamma globulins. Rifampin and Levofloxacin may also be effective. Imidocarb is sometimes used in conjunction with antibiotics, usually for co-infections with *Babesia* and *Hepatozoon*. With severe disease, blood transfusions or intravenous fluids may be necessary. Corticosteroids (prednisone) can be used to palliate immune-mediated secondary reactions such as immune-mediated arthritis or platelet loss. Generally, the prognosis during the acute phase is good if the animal is treated properly. Animals in the chronic stage have a poorer prognosis.

Prevention and control: Exposure to ticks should be limited and use of preventatives (e.g. permethrin) considered. Animals should be examined for ticks in tick-infested areas and at peak time of year (April through September). Vegetation can be modified to discourage tick and wild host habitation. Seronegative blood donors should be used for transfusions. Vaccine development against CME shows promise.

Suggested disinfectant for housing facilities: Area-wide application of acaricides and removal of leaf litter and brush are effective. Consider least-toxic pesticide for use on targeted barriers.

Notification: Not required

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Animals may be carriers but ticks are still needed for transmission. Note that transmission can occur through a blood transfusion when the donor is infected.

Conditions for restoring disease-free status after an outbreak: Tick control in the environment is essential. Infected ticks can transmit the disease for 155 days, and after treatment, an animal is still susceptible to re-infection with the same, or another *Ehrlichia* species. However, short-term protection has been described with some *Ehrlichia* infections, waning after about one year. Prophylactic administration of tetracycline at a lower dose is effective in preventing *E. canis* infection in situations where disease is endemic. Treatment must be extended for many months through at least one tick season if the endemic cycle is to be successfully eliminated. PCR, conducted several weeks after termination of treatment, can provide confidence that a treatment has been effective versus an animal entering a subclinical phase.

Experts who may be consulted:

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ENCEPHALITOOZONOSIS / ENCEPHALITOOZON CUNICULI

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Rabbits; rodents; canids; and sporadic cases in a variety of mammals	Ingestion of environmentally resistant spores passed in urine/feces of infected host; transplacental.	Asymptomatic; neurologic; nephritis to end-stage renal failure; uveitis	Frequently asymptomatic in immunocompetent adult animals. However, progressive disease can be fatal.	Variably successful; prolonged benzimidazoles.	Environmental sanitation to prevent spore contamination.	Yes.

Fact Sheet compiled by: Karen Snowden

Sheet completed on: 26 September 2013

Fact Sheet Reviewed by: Elizabeth Didier; Susan Rohrer; Meredith M. Clancy

Susceptible animal groups: Domestic rabbits, rodents (mice, rats, muskrats, guinea pigs, hamsters, ground shrews), domestic dog; sporadic cases reported in a variety of wild carnivores including farmed blue fox (*Alopex lagopus*), wild red fox (*Vulpes vulpes*), martens (*Martes* spp.) and mink (*Mustela vison*). Sporadic natural infections reported in several species of non-human primates, including squirrel monkeys (*Saimiri sciureus*), emperor tamarins (*Saguinus imperator*), Goeldi's monkeys (*Callimico goeldii*), and experimental infections reported in vervet monkeys (*Cercopithecus pygerythrus*).

Causative organism: *Encephalitozoon cuniculi*; phylum Microsporidia (intracellular eukaryotic single-celled organism; classified by some as protozoa, by others as fungi)

Zoonotic potential: Yes, immunocompromised human cases reported. Direct animal to human transmission has not been reported although molecular characterization shows animal and human genotypes identical.

Distribution: Ubiquitous; worldwide from tropical to temperate to cold climates.

Incubation period: Poorly defined in natural infections and dependent on spore dose. Death in experimentally infected puppies in 2-8 weeks and in experimentally infected immune deficient mice 10-27 days.

Clinical signs: Most frequently, the infection is asymptomatic in immunocompetent adult animals. Progressive neurologic signs including ataxia, head tilt, circling, head pressing, can present in rabbits and canids. Progressive glomerulonephritis to end-stage renal failure can occur in dogs. Uveitis, sometimes with cataract development, can occur in rabbits.

Post mortem, gross or histologic findings: Encephalitis with multifocal to disseminated mononuclear or granulomatous inflammatory infiltrates and perivascular cuffing in the brain; glomerulonephritis; uveitis with cataract formation; intracellular organisms commonly seen in vascular endothelium of brain, glomeruli and renal tubular epithelium of kidney.

Diagnosis: Microscopically, the Gram positive organisms can be visualized in histologic sections; microscopically visualized spores in body secretions such as urine sediment or CSF using modified trichrome stain or chitin-binding Calcofluor or Fungi-Fluor stain; PCR of tissue samples; detect parasite-specific antibodies using IFA or ELISA.

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Material required for laboratory analysis: tissue, body fluids for staining and microscopy to visualize intracellular organisms or spores; tissue, body fluids for PCR; serum for antibody detection (IFA, ELISA).

Relevant diagnostic laboratories: Serologic screening is available for rodent/rabbit species through major

laboratory research animal vendors. Molecular diagnostic testing is available only through research labs, not commercially available.

Charles River Laboratories
1-877-274-8371

A list of locations can be found at: <http://www.criver.com/about-us/locations>

IDEXX Reference Laboratories
One IDEXX Drive
Westbrook, Maine 04092
1-888-433-9987

A list of locations can be found at: <http://www.idexx.com/>

Treatment: Prolonged administration of albendazole has been used in humans and anecdotally used successfully in dogs. Prolonged administration of fenbendazole has been reported in rabbits.

Prevention and control: Environmental sanitation very important to prevent contamination with environmentally resistant spores; transmission of spores via fomites is probable. Research rodent/rabbit colonies use a serologic test and cull approach to eliminate carrier animals.

Suggested disinfectant for housing facilities: Environmentally resistant spores can be inactivated by chlorine, peroxide and other disinfectants with adequate contact time.

Notification: Not reportable in animals or humans.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Not recommended. Asymptomatic seropositive animals can shed parasite spores intermittently for months/years, posing risk of exposure of introduced uninfected animals to environmentally resistant spores.

Conditions for restoring disease-free status after an outbreak: Remove seropositive animals from population; rigorous environmental cleanup and disinfection

Experts who may be consulted:

Karen Snowden
Texas A&M University
Dept. of Veterinary Pathobiology, #4467
College Station, TX 77843-4467
(979) 862-4999 ksnowden@cvm.tamu.edu

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ENCEPHALOMYOCARDITIS VIRUS (EMC)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals	Feco-oral, urine, or carcass ingestion. Rodents are asymptomatic carriers.	Range from non-clinical signs to nonspecific (lethargy, anorexia) to cardiac failure and sudden death	Many infections are asymptomatic, but may manifest as sudden death	Supportive care for cardiac failure if possible	Rodent and rodent feces control Hygienic feeding practices +/- vaccine	Yes

Fact Sheet compiled by: Kay Backues

Sheet completed on: 9 Jan 2019

Fact Sheet Reviewed by: Gretchen Cole

Susceptible animal groups: Mammals, including humans

Causative organism: Encephalomyocarditis virus which belongs to genus *Cardiovirus* in the family Picornavirus. The traditional virus should be labeled EMCV-1 as a new strain isolated from a wood mouse is being called EMCV-2. The new strain can be distinguished serologically and by molecular testing. The host range of EMCV-2 remains to be determined.

Zoonotic potential: Yes. Infection in humans is common although many are asymptomatic and unrecognized.

Distribution: Worldwide, free ranging and captive. In US, disease primarily seen in states bordering the Gulf of Mexico.

Incubation period: Viremia may occur within 24 hours of infection.

Clinical signs: Many infections are nonlethal and probably subclinical. Subtle nonspecific clinical signs include lethargy, anorexia, listlessness or dyspnea. Signs of acute heart failure may occur, especially in primates and artiodactylids. Typical presentation is death without any prior signs of illness. Neurologic signs are not common except in smaller non-human primates and some rodents.

Post mortem, gross, or histologic findings: Gross findings primary limited to cardiovascular system: myocardium severely marked with pale streaks, petechiae or ecchymosis on the epicardial surface. Sequelae to heart failure such as pulmonary edema, hydrothorax, hydropericardium, froth in trachea or bronchi, and fibrin in the body cavities. Pulmonary edema is often severe and dramatic, lungs are wet and heavy. Histologic findings include lymphocytic, plasmacytic necrotizing myocarditis; congested and markedly edematous lungs. Encephalitis is frequently seen in rodents, and may be seen in larger animals but the CNS infrequently is submitted for larger animals.

Diagnosis: Histologic appearance of affected tissues is very suggestive of disease. Further diagnostics to be considered include polymerase chain reaction (PCR); virus isolation from tissues – fresh or frozen; serologic testing via virus neutralization (VN), hemagglutination-inhibition, or ELISA for paired titer, although this route is not very helpful in acute cases. If animals survive, antibody testing may be helpful.

Material required for laboratory analysis: PCR on whole blood, serum, plasma, or tissue (fresh or frozen) so at necropsy liver, heart, and spleen should be collected in most animals. Tissues for virus isolation: heart muscle, spleen, liver and brain from wildlife species. In addition, take intestine from rodent species.

ENCEPHALOMYOCARDITIS VIRUS (EMC)

Relevant diagnostic laboratories:

Zoologix Inc. (PCR)
9811 Owensmouth Avenue, Suite 4
Chatsworth CA 91311-3800
Phone: 818-717-8880
Fax: 818-717-8881
Email: info@zoologix.com
www.zoologix.com

Texas A&M Veterinary Medical Diagnostic Laboratory (Virus neutralization)
College Station Laboratory
PO Box Drawer 3040
College Station, TX 77841-3040
Phone: (979) 845-3414
Toll Free: (888) 646-5623
Fax: (979) 845-1794
<http://tvmdl.tamu.edu>

USDA-APHIS-VS-NVSL
P.O. Box 844 (letters)
1920 Dayton Ave. (packages)
Ames, IA 50010
Phone: (515) 337-7266
Fax: (515) 337-7397
http://www.aphis.usda.gov/animal_health/lab_info_services/

Treatment: Generally no treatment is performed because animal are often asymptomatic for infection or found dead. Supportive care for cardiac failure can be provided in less than acute cases.

Prevention and control: Consistent and long term rodent control and prevention of rodent access to animal enclosures and food sources is critical for prevention. Hygienic feeding practices are important. If rodent feces are detected, increase rodent control measures and change feeding practices such as not leaving food bowls available overnight. Enclosure surfaces and food bowls should be cleaned with appropriate disinfectants. In enclosures with heavy contamination of rodent feces, removal of soil and substrate should be considered.

Commercial vaccines are not available in the US. Vaccine research is ongoing and recent trials have shown some promise in producing antibodies in tested species. However, USDA allows the production and use of autogenous product from an affected institution's viral isolates at that institution only. To discuss this possibility, available contact is : Dr. Mark Titus, Newport Laboratories, Worthington, MN 56187, (800) 220 2522, direct phone: 507-372-3563, www.newportlabs.com, mtitus@newportlabs.com.

Suggested disinfectant for housing facilities: 5.25% sodium hypochlorite or household bleach at 3% dilution can be used by adding 3 gallons bleach to 2 gallons water and mixing thoroughly. This combination can be corrosive and damage clothing. Potassium peroxymonosulfate and sodium chloride (Virkon-S) - 1% dilution, Follow label directions. Sodium carbonate (soda ash) – 4% dilution, Add 5.33oz sodium carbonate to 1 gallon hot water (mildly caustic).

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: None

Conditions for restoring disease-free status after an outbreak: Remove all evidence of rodent feces in the environment, clean the affected animal's living spaces with an approved disinfectant (see above), increase rodent control, re-evaluate animal feeding strategies. A zoo collection animal (non-rodent species) that recovers from disease is not considered a carrier.

ENCEPHALOMYOCARDITIS VIRUS (EMC)

Experts who may be consulted:

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Tulsa, Oklahoma 74115
Phone: (918) 669-6243
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EQUINE INFECTIOUS ANEMIA

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Family Equidae	Hematophagus insects; iatrogenic (blood transfusion, needles, surgical instruments, teeth floating, etc.)	Variable: Fever, weight loss, icterus, anemia, edema and weakness	Variable	No treatment	No vaccine; keep ~200m from antibody positive animals; control insects	No

Fact Sheet compiled by: Amanda Guthrie

Sheet completed on: 16 March 2011; updated 5 April 2013; updated 12 Feb 2018

Fact Sheet Reviewed by: Mark Drew, Nancy Carpenter

Susceptible animal groups: All members of Equidae although donkeys and mules are less likely to develop severe clinical signs.

Causative organism: Lentivirus in family Retroviridae (subfamily Orthoretrovirinae)

Zoonotic potential: No

Distribution: It is distributed nearly worldwide, except a few countries including Iceland and Japan.

Incubation period: Although usually a week to 45 days or longer is required for infection, it typically takes 45 days or more for sufficient antibody to be produced to cause positive test result.

Clinical signs: Nonspecific; equid may have fever and transient inappetence. Severity of disease depends on strain and dose of virus and health of the animal.

Acute: weakness, depression, inappetence, jaundice, tachypnea, tachycardia, ventral pitting edema, thrombocytopenia, petechiae on mucus membranes, epistaxis or blood stained feces.

Chronic: recurring clinical signs that vary from mild illness and failure to thrive to fever, depression, petechial hemorrhages on mucus membranes, weight loss, anemia and dependent edema.

Asymptomatic: Carriers with no clinical signs.

Post mortem, gross, or histologic findings: Findings during febrile illness include generalized lymph node enlargement, an enlarged liver with a prominent lobular pattern, an enlarged spleen, mucosal and visceral hemorrhages, ventral subcutaneous edema, and vascular thrombosis. Histopathology of these tissues reveals accumulations of lymphocytes and macrophages in liver, lymph nodes, adrenal glands, spleen, meninges, and lung. Extramedullary hematopoiesis and proliferation of reticuloendothelial cells is evident. Pathology of infected animals with no clinical signs are generally unremarkable, although some may have glomerulitis, retinal depigmentation and choroiditis.

Diagnosis: Agar Gel Immunodiffusion (Coggins test) is only legally recognized test, but now at least three rapid ELISA tests are available; a positive ELISA must be verified with a Coggins test. Positive animals are infected for life.

Material required for laboratory analysis: Blood drawn by accredited veterinarian and must be submitted to an approved lab.

Relevant diagnostic laboratories: Labs are widely available in each state.

Treatment: None.

Prevention and control: As no vaccine is available, uninfected animals must be maintained ~200m from antibody positive animals. Coggins test is used for surveillance for asymptomatic carriers and at preshipment and quarantine. All horses should be tested annually; interstate travel requires a negative EIA test as do most horse shows or public sales. Control by decreasing risk through effective fly control and proper disinfection of equipment between animals.

Note: EIA virus can be passed from mare to foal *in utero*, is present in milk and semen and can be transmitted venereally, may be transmitted via aerosols.

EQUINE INFECTIOUS ANEMIA

Suggested disinfectant for housing facilities: None; virus is only transmitted via contact with blood or other bodily secretions.

Notification: EIA is reportable to state veterinarian and federal APHIS office; check state and local laws. Some jurisdictions require permanent identification of positive animals with brands or tattoos.

Measures required under the Animal Disease Surveillance Plan: Federally reportable disease.

Measures required for introducing animals to infected animal: While not recommended, positive animals should remain ~200m away from uninfected animals.

Conditions for restoring disease-free status after an outbreak: It is recommended that positive animals be removed from the population

Experts who may be consulted:

National Veterinary Services Laboratory

USDA-APHIS-VS-NVSL

1920 Dayton Ave. (for packages)

P.O. Box 844 (for letters)

Ames, IA 50010

Phone: (515) 337-7266

Fax: (515) 337-7397

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/lab-info-services/sa_diagnostic_tests/ct_diagnostic_tests

Resources:

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3. Equine Infectious Anemia (EIA) [Internet]. United States Department of Agriculture Animal and Plant Health Inspection Services: 2017 [cited 2018 February 12]. Available from <https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/nvap/NVAP-Reference-Guide/Equine/Equine-Infectious-Anemia>
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ERYSIPELAS (*Erysipelothrix rhusiopathiae*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Swine, sheep, turkeys, multiple other vertebrate species	Bacteria shed in urine, saliva, nasal secretions, and feces. Transmitted by direct contact with infected animals or body discharges (ingestion, transcutaneous). Apparently healthy swine can be carriers.	Acute disease – pyrexia, anorexia, depression, stilted gait, diamond skin lesions, death. Chronic – exercise intolerance, lameness, enlarged joints	Mild to severe. High mortality in untreated animals.	Acute disease – penicillin. Chronic disease – no treatment.	Vaccinate herd, practice good sanitation, avoid overcrowding, quarantine new animals, eliminate chronic carriers.	Yes

Fact Sheet compiled by: Cora Singleton

Sheet completed on: 1 March 2011, updated 31 October 2012.

Fact Sheet Reviewed by: Pat Morris; Alex Ramirez

Susceptible animal groups: Swine, sheep, turkeys, multiple other vertebrate species

Causative organism: *Erysipelothrix rhusiopathiae*, a facultative anaerobic, weak gram-positive bacillus.

Zoonotic potential: *E. rhusiopathiae* causes local skin lesions (erysipeloid) in humans as an occupational disease of people who handle and process meat, veterinarians, game handlers, leather workers, and laboratory workers.

Distribution: Worldwide

Incubation period: Bacteremia usually develops within 24 hours of exposure. Bacteria may persist in joints and lymphoid tissue for months.

Clinical signs: Acute disease – Pyrexia, anorexia, depression, stilted gait, raised rhomboid light pink to purple skin lesions (“diamond-skin” lesions), abortion, and sudden death.

Chronic disease – Animals that survive acute disease may show exercise intolerance and cyanosis (valvular endocarditis), swollen joints and lameness (arthritis).

Post mortem, gross, or histologic findings:

Acute disease – Widespread congestion, petechial and ecchymotic hemorrhages, microthrombi and focal necrosis, mononuclear inflammation.

Chronic disease – Proliferative nonsuppurative arthritis, vegetative endocarditis.

Diagnosis: Clinical signs and necropsy lesions (especially “diamond-skin” lesions), bacterial culture, and serology. A variety of serologic tests are available, which are more valuable for detection of chronic infection on a herd basis than for detection of acute disease in individual animals.

Material required for laboratory analysis: Swab or tissue sample (blood, organs, joints) for culture.

Relevant diagnostic laboratories: Multiple laboratories available.

Treatment: Penicillin is the antibiotic of choice for acute disease but macrolides, streptogramins (eg., quinupristin/dalfopristin, pristinamycin, virginiamycin), tetracyclines, lincomycin and tylosin may also be

ERYSIPELAS (*Erysipelothrix rhusiopathiae*)

effective. Hyperimmune serum may be useful early in the course of disease. No practical treatment for chronic erysipelas is available.

Prevention and control: Vaccinate herd, practice good sanitation, avoid overcrowding, quarantine new animals, and eliminate chronic carriers.

Suggested disinfectant for housing facilities: Phenolic, alkali, hypochlorite, or quaternary ammonium disinfectants are effective.

Notification: Erysipelas is not reportable to USDA/APHIS or OIE but may be reportable to local or state agencies.

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Eliminate chronic carrier animals.

Experts who may be consulted:

Veterinary Diagnostic and Production Animal Medicine Department

Iowa State University College of Veterinary Medicine

Phone: 515-294-1950

Fax: 515-295-3564

<http://vetmed.iastate.edu/vdpam/>

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***Escherichia coli* – STEC/EPEC**

Animal Groups Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Shiga-toxigenic <i>E. coli</i> (STEC): ruminants, swine	Fecal-oral; animal to animal; Direct contact with contaminated surfaces; contaminated feed.	Asymptomatic infection without clinical disease in animals	Non-pathogen transient predominantly but not exclusively; summer commensal of ileum and large colon	Oral neomycin (experimental)	Two commercial vaccines for cattle; environmental hygiene; gloves when working with known colonized animals	Yes; young children <5 years of age at highest risk especially from direct animal contact, e.g. at children's zoos although STEC-induced disease can occur in people of all ages.
Enteropathogenic <i>E. coli</i> (EPEC): various serotypes based on pili typing or enteropathogenesis: ruminants, swine	Fecal-oral; direct contact with infected surfaces and pastures	Severe acute diarrhea, usually seen between 1-10 days of age	Severe with death common if untreated	Fluid and electrolyte replacement, systemic antibiotics	Good colostrum transmission, birthing area management, vaccination of the dam preparturition or use of oral antibody preparation at birth	Not generally

Fact Sheet compiled by: Victor Cortese

Sheet completed on: 19 April 2011; updated 10 August 2013; November 2018

Fact Sheet Reviewed by: Guy Loneragan, Franklyn Garry

Susceptible animal groups: Ruminants, swine; also for EPEC, all species to a lesser degree should be considered susceptible. Serotype pathogenicity tends to be strongly related to animal species. Disease is almost exclusively seen in neonates.

Causative organism: Shiga-toxigenic *Escherichia coli* (STEC) O157:H7 or O157:non-motile; many STEC serogroups including O26, O45, O103, O111, O121 and O145 may also infect ruminants and may cause zoonotic disease. For EPEC, enterotoxigenic, enterohemorrhagic and attaching and effacing. Further identification based on pilus types - K99 (predominant in cattle), F4 (K88), F5 (K99), F41, F6 (987P) and F18 *E.coli*,

Zoonotic potential: STEC is zoonotic and may result in mild to severe disease which may occasionally be fatal. EPEC causes mild to severe in other species, but rarely causes disease in humans, although occasional fatal disease in infected people has occurred.

Distribution: Highly prevalent in ruminant herds in temperate regions throughout the year with very high prevalence in summer months; uncommon in swine. Variable distribution within herds is observed.

Incubation period: In STEC, the incubation period is unknowable because there is no disease. The patent period of fecal shedding is summer biased, variable (2 weeks +/- one week) and may be sporadic or episodic. For EPEC, the incubation period is very short with diarrhea often seen within 12-48 hours after exposure.

***Escherichia coli* – STEC/EPEC**

<p>Clinical signs: Asymptomatic condition is noted with STEC. However, for EPEC, severe watery diarrhea that may be blood tinged is presented and resultant severe dehydration. With some attaching and effacing <i>E. coli</i>, mucosal lining maybe be sloughed and voided in the diarrhea. Toxins may cause a hypersecretory diarrhea.</p>
<p>Post mortem, gross, or histologic findings: In STEC, no visible gross lesions; may observe rare attaching and effacing histopathological lesions in colonic mucosal of colonized animals. For EPEC, fluid filled intestinal tract and mucosal lining damage is observed with some strains.</p>
<p>Diagnosis: Culture and isolation using enrichment, immuno-concentration, selective chromogenic agar, and PCR or serologic confirmation of suspect isolates is available for STEC. Similarly for EPEC, culture and isolation can be used, with FA, EM and PCR as confirmation.</p>
<p>Material required for laboratory analysis: Fresh rectal feces or freshly ground deposited feces (10gm) for either STEC or EPEC, or for STEC, environmental samples (e.g., hide swabs, surface swabs, soil and water) can be cultured, and for EPEC, intestinal section.</p>
<p>Relevant diagnostic laboratories: Various veterinary research laboratories; any BSL-2 bacteriological laboratory if personnel are adequately trained in STEC detection methods</p>
<p>Treatment: In STEC, oral neomycin sulfate in water at label dose has been used experimentally. In EPEC, oral electrolytes and IV fluids, in severe cases, systemic antibiotics, and NSAIDs may be needed.</p>
<p>Prevention and control: One vaccine based on siderophore technology has been shown to decrease fecal shedding of O157 and is available for use in cattle. Isolate infected animal groups and prevent contact of people with animals and animal feces. Clean and disinfect animal housing areas and surfaces. Animal hides, oral cavity and feces may contain high numbers of viable STEC O157. Handling sanitation of workers and handlers is recommended. Hand-washing stations recommended for visitors. Several vaccines are available for use in cattle and swine to enhance colostral transmission of antibodies against the various types of enteropathogenic <i>E. coli</i>. Oral antibody preparation can be given to the neonate have also been shown to be helpful in controlling the disease. Isolate infected animal groups and change birthing area is important.</p>
<p>Suggested disinfectant for housing facilities: Potassium peroxymonosulfate and sodium chloride (i.e. Virkon-S); avoid bleach solutions and lime as disinfectants.</p>
<p>Notification: Reportable in all 50 US states if human disease occurs for STEC.</p>
<p>Measures required under the Animal Disease Surveillance Plan: None currently</p>
<p>Measures required for introducing animals to infected animal: Await negative fecal test results. Consider use of vaccination of known infected animals to decrease potential shedding of the bacteria.</p>
<p>Conditions for restoring disease-free status after an outbreak: For STEC, isolation from contact with other animals or public for at least two weeks, followed by serial negative fecal culture of all animals in group. Place in cleaned and disinfected housing; may wish consider permanent withdrawal from herd or euthanasia of animal having direct contact with public, especially children. For EPEC, isolation from contact with other animals or public for at least two weeks and separation of recovered animals form newborns.</p>
<p>Experts who may be consulted: Victor Cortese, DVM, PhD Dipl ABVP Zoetis Inc. 746 Veechdale Road Simpsonville, KY 40067 610-662-6505 victor.cortese@zoetis.com</p>

***Escherichia coli* – STEC/EPEC**

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FELINE CALCIVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Felids - domestic and exotic	Direct (oronasal); indirect (fomites)	Acute and chronic respiratory forms (mainly upper respiratory infection); arthritic form (lameness); virulent systemic form	Variable	Symptomatic	Prevention of exposure to infected animals; vaccination; disinfection	No

Fact Sheet compiled by: Tara M. Harrison

Sheet completed on: 2 May 2011; 7 September 2012; updated January 2018

Fact Sheet Reviewed by: Dalen Agnew; Annabel Wise; Roger Maes; Rebecca Smedley

Susceptible animal groups: Domestic and exotic felids

Causative organism: Feline calicivirus (FCV)

Zoonotic potential: None

Distribution: World-wide distribution is in all members of Felidae. Disease is most common in multi-cat environments (e.g., shelters, breeding facilities) and in feral cats. The latter has been implicated in spreading this virus to a zoological institution in North America. Reports of this infection have been made in other zoological institutions.

Incubation period: Variable (2-10 days) and recovery typically in 7-10 days in the absence of complications.

Clinical signs: Mild upper respiratory infection: ocular and nasal discharge with potential for secondary infections; oral ulceration is a common transient sign.

Systemic infection: sloughing of oral mucosa (tongue), foot pads, and other mucosal epithelia; edema; pyrexia; ulcerative dermatitis; anorexia; jaundice; and death (mortality rates up to 60%); adult cats are more severely affected than kittens with virulent systemic infections.

Lymphoplasmacytic gingivitis/stomatitis and arthritis (“limping syndrome”) are also observed in domestic cats.

Post mortem, gross, or histologic findings:

Respiratory form: oral ulceration; nasal and ocular discharge; conjunctivitis; rarely interstitial pneumonia.

Virulent systemic form: cutaneous edema and ulceration associated with vasculitis; hepatocellular necrosis; interstitial pneumonia; rarely gastrointestinal ulceration; intestinal crypt lesions and pancreatitis have been reproduced experimentally.

Lymphoplasmacytic gingivitis/stomatitis: proliferative/ulcerative lesions.

Limping syndrome: acute synovitis with thickening of the synovial membrane and increased joint fluid.

Diagnosis: Virus isolation (VI), RT-PCR, virus neutralization or ELISA on paired sera, FA, immunohistochemistry (IHC); always in conjunction with compatible clinical signs

Material required for laboratory analysis: Oropharyngeal and conjunctival swabs of lesions for VI or RT-PCR (use synthetic swabs); paired sera to quantitate virus neutralizing antibody titers; affected tissues for VI, RT-PCR, FA, or IHC.

Relevant diagnostic laboratories: Most diagnostic laboratories can identify.

Treatment: Supportive; prevention or treatment of secondary infections

Prevention and control:

Prevention: Vaccination using Fel-O-Vax PCT + CaliciVax® vaccine to minimize severity of infection, particularly of virulent systemic strains; only killed vaccines should be used in exotic felids. There have been several cases of suspected vaccine-induced calicivirus in tigers and lions in the United States (personal communication, Harrison 2012, Rivas 2015).

Control: limit access to feral cats that can carry and spread FCV and recovered animals can shed infectious virus for months to years. Proper cleaning as FCV can survive up to 14 days on inanimate objects.

FELINE CALCIVIRUS

Suggested disinfectant for housing facilities: 1:30 dilution of commercial bleach; potassium peroxymonosulfate; chlorine dioxide; substituted phenolic compounds; quaternary compounds formulated at appropriate concentration and pH.

Notification: None required

Measures required under the Animal Disease Surveillance Plan N/A

Measures required for introducing animals to infected animal: Vaccination of non-exposed animal and monitoring of shedding status of both infected and incoming animal; preferably introduce incoming non-shedding animals to infected animal only after verification that the infected animal is no longer shedding infectious FCV.

Conditions for restoring disease-free status after an outbreak: Many felids can become chronic carriers so continue to monitor shedding through VI or RT-PCR. Once absence of shedding has been verified, continue to vaccinate infected animals as immunity is waning, and vaccinate susceptible animals to minimize clinical signs.

Experts who may be consulted:

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FELINE IMMUNODEFICIENCY VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Felids - most infections are species-specific, but some evidence for interspecific transmission in captive collections.	Horizontal transmission is the most prevalent route; prevalence suggests exposure occurs concurrent with sexual maturity. Vertical transmission can occur, but is the exception.	Asymptomatic to severe, depending on the strain of the virus and stage of the disease. Most often asymptomatic in non-domestic felids, but may include moderate to severe oral cavity disease, mild to progressive anemia, skin infections, weight loss, vomiting, diarrhea, and neurologic disease	Usually asymptomatic but CD4 ⁺ cell depletion - depending on the strain – can present with increased morbidity and mortality. Infection is life-long.	No specific treatment, but supportive care indicated with clinical signs.	Testing all felids prior to introduction into a collection; controlling feral cat populations	No

Fact Sheet compiled by: Kristian J. Krause
Sheet completed on: 3 August 2011; updated 25 February 2013
Fact Sheet Reviewed by: Karen A. Terio, Susan VandeWoude, Winston Vickers
Susceptible animal groups: Felids
Causative organism: Feline Immunodeficiency Virus, a Lentivirus
Zoonotic potential: None
Distribution: Worldwide. In domestic cats, most commonly found in intact feral males. In non-domestic wild felids, an increase in seroprevalence correlates with sexual maturity.
Incubation period: 3-6 months
Clinical signs: In most non-domestic felids with naturally occurring disease, FIV positive cats are asymptomatic. However, in domestic cats and captive non-domestic felids infected with certain strains, especially older cats, signs can include mild to progressive anemia, moderate to severe oral disease, especially stomatitis, mild to significant weight loss, chronic or non-healing skin infections, vomiting, diarrhea, neurologic disease, and atypical lymphosarcoma.
Post mortem, gross, or histologic findings: Findings correlate with associated diseases, if any, present.
Diagnosis: Western blot and ELISA assays are the most commonly used method of diagnosing FIV. Western blot is available for domestic cats, cougars, and African lions and may be more sensitive than domestic cat FIV

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based ELISA. PCR is available, but is not as reliable because strain genetic variation is high.
Material required for laboratory analysis: Serum, plasma or whole blood can be used for diagnosis.
Relevant diagnostic laboratories: Any laboratory capable of running the FIV ELISA is able to diagnose FIV; however, this assay may be less sensitive than strain specific Western Blot.
Treatment: Most non-domestic felids do not need any treatment. Treatment is for any specific clinical signs that arise and is supportive.
Prevention and control: Felids should be tested by ELISA prior to introduction into a new facility with other felids. Special care should be taken to prevent interaction with feral cats.
Suggested disinfectant for housing facilities: FIV is labile outside the host animals. It is easily inactivated by detergents and routine disinfectants. Routine cleaning procedures will prevent transmission. Dental and surgical instruments, anesthesia circuits, endotracheal tubes, and other items potentially contaminated with body fluids should be thoroughly cleaned and sterilized between uses. Fluid lines, multi-dose medication containers, and food can become contaminated with body fluids (especially blood or saliva) and should not be shared.
Notification: Receiving institutions should be notified of an infected animal.
Measures required under the Animal Disease Surveillance Plan: Currently none
Measures required for introducing animals to infected animal: No specific measures need to be taken. Whether or not to introduce infected and non-infected animals should be based on a population management decision, knowing that non-infected animals may become infected. Knowledge of the strain and likely clinical disease can assist with these decisions.
Conditions for restoring disease-free status after an outbreak: Not applicable as infection is life-long
Experts who may be consulted: Sue VandeWoude, DVM (Western blot testing) Professor of Comparative Medicine Department of Microbiology, Immunology, Pathology Director, Laboratory Animal Resources Colorado State University 1619 Campus Delivery Fort Collins CO 80523-1619 Phone: (970) 491-7162 Fax: (970) 491-0523 suev@lamar.colostate.edu
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FELINE INFECTIOUS PERITONITIS (FIP)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p>Felids – domestic cats and some exotic cat species</p> <p>The disease has been documented in cheetahs.</p>	<p>Primary mode of transmission is through feces. The virus is highly infective and over 90% of cats in multi-cat households typically seroconvert.</p>	<p>Malaise, inappetance, weight loss, and fluctuating fever. Effusive form – ascites, thoracic and/or pericardial effusion. Ocular lesions and CNS signs more common in the dry form.</p>	<p>Some cats exposed to the virus remain healthy while those that develop the disease have a poor prognosis. Clinical course is a few days to several months. The course is typically rapid with the effusive form of the disease and may be longer with the dry form.</p>	<p>No treatment has yet proven effective in curing cats of FIP. The disease is considered fatal.</p>	<p>Proper management can decrease the incidence of FIP in catteries.</p> <p>There is no effective vaccine.</p>	<p>No</p>

Fact Sheet compiled by: Danelle M. Okeson

Sheet completed on: December 2017

Fact Sheet Reviewed by: Kay Backues, Beth Bicknese

Susceptible animal groups: Felids – domestic cats and African lion, mountain lion, leopard, jaguar, lynx, serval, caracal, European wild cat, sand cat, Pallas cat, and cheetah (which seem to be more susceptible than other exotic felids). Most deaths in domestic cats occur in cats 3-16 months of age and are uncommon after 5 years.

Causative organism: Only a portion of cats infected with the coronavirus develop FIP. All feline coronavirus (FCoV) types may induce systemic infection. While the precise mechanism by which FIP develops is unclear, two main hypotheses have been proposed. In both cases, the ability of an FCoV to replicate in macrophages is a key pathogenic event. In the first hypothesis, a primarily avirulent FCoV that replicates in enterocytes undergoes a mutation that allows it to replicate in macrophages. In the second hypothesis, the host's immune response and viral load determine whether a cat infected with any FCoV will develop FIP (from AAEP 2013 disease fact sheet).

Zoonotic potential: No

Distribution: FIP may occur wherever FCoV occurs – worldwide and ubiquitous among cat populations.

Incubation period: Under experimental conditions, 2-14 days is required for the effusive form of the disease while several weeks longer for experimentally induced dry/non-effusive form.

Clinical signs: Early signs of the disease may be non-specific: lethargy/malaise, fluctuating fever, loss of appetite, weight loss, and may cause failure to thrive in young cats.

The disease is categorized as two forms:

- Effusive/wet form with vasculitis and polyserositis - ascites, thoracic and/or pericardial effusion. The effusive form is the more common form of the disease.

- Non-effusive/dry form with granulomatous lesions in kidneys, intestinal tract (leading to chronic diarrhea), lymph node enlargement.

Ocular and neurologic signs occur in <9% of cats with the wet form, but are relatively frequent in cats with the dry form. Ocular signs may include chorioretinitis and retinal perivascular cuffing, keratic precipitates in the anterior eye, and uveitis. Neurologic signs may include nystagmus, cranial nerve defects, seizures, ataxia, hyperesthesia, and behavioral changes.

Post mortem, gross, or histologic findings:

- Effusive (wet) form of FIP - gross findings: viscous thoracic or abdominal fluid; pyogranulomas that tend to follow the course of the cranial mesenteric artery – leading to thickened omentum containing pyogranulomas, and pyogranulomas covering the serosal surface of the abdominal viscera. The pyogranulomas appear as small, coalescing, fibrinous plaques.

FELINE INFECTIOUS PERITONITIS (FIP)

-Dry form – gross findings: pyogranulomas that appear as raised, gray-white nodules ($\geq 0.5 - 2$ cm) in the kidneys, liver, intestines, and visceral lymph nodes. CNS lesions and ocular lesions are more common in the dry form. Eye lesions may include iridocyclitis or chorioretinitis, and anterior uveitis, retinitis with hemorrhage and/or retinal detachment, and optic neuritis. Pyogranulomas may be found in the brain and spinal cord, or CNS lesions may manifest as diffuse meningitis.

Diagnosis: The disease can be difficult to diagnose. Currently, there is no test specific for FIP. “Ultimately, FIP must be diagnosed by applying a workable knowledge of the disease with sensible weighing of signalment, history, clinical signs, clinicopathologic findings, serology, and ante-or post-mortem examination of affected tissues by histopathology and immunohistochemistry” (Pedersen).

Antibody testing: Serology - ELISA, IFA (immunofluorescent antibody), and virus-neutralization tests detect the presence of coronavirus antibodies in a cat, but these tests cannot differentiate between the various strains of feline coronavirus.

Antigen testing: Immunohistochemistry on effusions or lesions containing infected macrophages is currently the gold standard for FIP diagnosis.

A PCR test is offered by a commercial laboratory and is said to differentiate between the non-pathogenic coronavirus biotype and the virulent or pathogenic biotype for use in domestic cats (IDEXX).

Material required for laboratory analysis: Effusions or lesions (such as pyogranulomas) containing infected macrophages – for immunohistochemistry (IHC). IHC tests for viral antigen. IHC using fluorescein staining requires fresh or frozen tissue sections. IHC using horseradish peroxidase (HRPO) staining may be performed on formalin fixed and paraffin embedded tissues. Both methods may be used on cells collected from effusions that have been acetone fixed. The fluorescein staining method is 5-10 times more sensitive than the HRPO method. Test sensitivity is dependent on having infected macrophages, so random biopsies of liver or kidney (biopsies not containing macrophages) in cats with FIP will not yield positive results.

Peritoneal, pleural, CSF fluid, or tissue biopsies may be used in the PCR test.

Relevant diagnostic laboratories: Several veterinary college laboratories and commercial veterinary labs offer FIP testing or referral to the appropriate lab.

University of Tennessee www.vet.utk.edu/diagnostic/virology/index.php

University of California Davis <http://www.sockfip.info/fip-studies/114-instructions-to-veterinarians-for-sending-fip-fluid-samples.html>

Treatment: No treatment has yet proven effective in curing the disease. Supportive care can be provided. Since clinical disease is caused by the cat’s immune response to the virus, proposed treatments have been aimed at controlling that response. In one study, feline interferon omega reportedly induced complete or partial remission in two-thirds of cats with FIP. However, the treatment proved totally ineffective in a larger double blinded study.

A pilot study at the University of Tennessee using an immunostimulant on three cats with the dry form (non-effusive) of FIP showed some promise. Two of three cats were still receiving treatment and were still alive 2 years after diagnosis (Legendre).

Prevention and control: Given the constraints on testing, it may be best to manage cheetahs as if the population is endemically infected (Gaffney, et al).

In case of a suspected outbreak or a seropositive animal, clinicians should contact FIP experts and/or clinicians who have dealt with similar situations in a captive wildlife setting. It is beyond the scope of this fact sheet to provide recommendations for every possible scenario. FIP is typically a problem in group-housed cats, such as in breeding catteries or rescue groups. Since there is no readily available ante-mortem test, cats cannot be effectively tested prior to introduction to a group. Strict hygiene (especially for litter boxes) and keeping cats in small groups can help reduce viral contamination.

Although a licensed FIP vaccine available, no effective vaccine is available as this vaccine has not been proven to prevent FIP, and it is not generally recommended by the American Association of Feline Practitioners Feline Vaccine Advisory Panel.

FELINE INFECTIOUS PERITONITIS (FIP)

Suggested disinfectant for housing facilities: The virus can survive for approximately 2 months in a dry environment. However, the virus is readily inactivated by detergents and disinfectants.

Notification: Not a reportable disease

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: See information under prevention and control. While not all cats exposed to the coronavirus that causes FIP will develop the disease, it is advisable not to mix cats with known infected cats.

Conditions for restoring disease-free status after an outbreak: Difficult in a multi-cat facility when other cats in the household or facility are likely infected. While many cats will not develop FIP disease, they may still shed the virus. Shedding may follow one of three patterns: 1) persistent for 18 months or more, 2) persistent for 4-6 months and intermittent for months thereafter, or 3) cleared within 6-8 months – most cats (Pedersen).

Experts who may be consulted:

Dr. Niels C. Pedersen, University of California, Davis College of Veterinary Medicine, contact information and further information on submitting samples may be found at the www.sockfip.info web site under information for veterinarians.

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FELINE LEUKEMIA VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Felids	High quantities of virus shed in nasal secretions & saliva; also shed in urine, feces, and milk, as well as semen and vaginal fluids from infected cats; most often transmitted to exotic felids via contact with or ingestion of domestic feral cats	Early – cats may have no signs. Anorexia, enlarged lymph nodes, persistent fever, gingivitis, stomatitis, persistent diarrhea, neurologic signs, eye conditions, abortions, reproductive failures	Depends on individual cat's immune response; typically asymptomatic and transient in exotic felids	No controlled studies proving effectiveness of immune modulators and interferon against the virus.	The retrovirus does not survive long outside the body under normal conditions Vaccination Exclusion of feral cats	No

Fact Sheet compiled by: Danelle M. Okeson

Sheet completed on: updated December 2017

Fact Sheet Reviewed by: Kay Backues

Susceptible animal groups: Felids. The first confirmed case of FeLV-associated lymphoma in a non-domestic felid occurred in a cheetah. Recent evidence suggests that the critically endangered Iberian lynx (*Lynx pardinus*) may be particularly susceptible to FeLV. The virus is otherwise not considered endemic in exotic felids, although antigen-positive animals have been documented, as well as seropositive, asymptomatic animals. FeLV has been isolated in leopard cat, European wildcat, and cougar.

Causative organism: A retrovirus, more specifically an oncornavirus/

Zoonotic potential: Not a zoonosis

Distribution: Rare but documented antigen-positive exotic cats have been found worldwide. In a study of more than 18,000 domestic cats, 2.3% of cats were FeLV antigen positive on ELISA testing. Prevalence was higher (3.6%) among cats allowed outdoors. Prevalence was highest among sick feral cats; 15.2% of tested ill feral cats were FeLV positive.

Incubation period: Infected cats may experience a prolonged period of clinical latency.

Clinical signs: In domestic cats, a variety of disease conditions are associated with retroviral infection including anemia, chronic inflammatory conditions, lymphoma, susceptibility to secondary and opportunistic infections, cutaneous abscesses, oral inflammation, and reproductive problems. Knowledge and understanding of the outcome of FeLV infection in domestic cats has changed. In the past, approximately one third of cats were believed to become persistently viremic and up to two thirds to eventually clear the infection. Newer research suggests that most cats remain infected for life following exposure but may revert to an aviremic state (regressive infection). In the case of a regressive infection, no antigen or culturable virus is present in the blood, but FeLV proviral DNA can be detected in the blood by polymerase chain reaction (PCR).

Therefore, two clinically relevant outcomes of FeLV exposure can be considered:

- 1) progressive infection – domestic cats typically succumb to FeLV-associated diseases within a few years. However these retrovirus positive cats may live without related illness for several years. “A decision about euthanasia should not be made based on a positive test alone.” (AAFP)
- 2) regressive infection – cats have an effective immune response, virus replication is contained, and there is no viral shedding. These cats have little risk of developing FeLV associated disease. Exotic cats typically belong to this group

FELINE LEUKEMIA VIRUS

Post mortem, gross, or histologic findings: Cats infected with FeLV that develop progressive infection may develop FeLV-related diseases including lymphoid malignancies, non-regenerative anemia, and myeloproliferative disorders. Findings may also include diseases secondary to immunosuppression, such as severe bacterial infections and toxoplasmosis.

Diagnosis:

Antigen testing – ELISA: This screening test detects the core viral antigen p27. This antigen is produced in large quantities in most infected domestic cats and most will test positive within 30 days of exposure. However, when results of antigen testing are negative but recent infection cannot be ruled out, testing should be repeated a minimum of 30 days after the last potential exposure.

Antigen testing – IFA: Antigen testing using immunofluorescent antibody (IFA) testing also detects p27 antigen within infected blood cells via bone marrow or blood smears. However, false negatives may occur in the following scenarios with domestic cats: leukopenic cats, cats with regressive infection, or cats that resist bone marrow infection. False positives may occur with sample preparation error, when background fluorescence is high, or when results are interpreted by inexperienced lab personnel.

Confirmatory testing: Cats that test positive on screening tests should be further tested with confirmatory tests. A second soluble antigen test should be performed, preferably using a test from a different manufacturer. (Virus culture is the gold standard, but not readily available in North America.) Practitioners should be aware that cats developing regressive infection may be only transiently antigenemic and may revert to negative status on soluble antigen tests. Confirmatory testing with PCR: Polymerase chain reaction (PCR) can detect FeLV RNA or DNA within one week of viral exposure in domestic cats; even when FeLV p27 antigen is not yet detectable. PCR testing detects either viral RNA or cell-associated DNA (provirus) in blood, bone marrow, and tissues.

Material required for laboratory analysis: Whole blood for antigen testing; blood, bone marrow, or tissues for PCR testing

Relevant diagnostic laboratories: Most commercial veterinary laboratories, most state veterinary diagnostic labs, Cornell University

Treatment: Immune modulators and interferon inducers are used in retrovirus-infected domestic cats, including FeLV-infected cats. Although reports of uncontrolled studies frequently suggest dramatic clinical improvement, these effects generally have not been reproduced in controlled trials. Preliminary laboratory studies have identified four drugs with anti-FeLV activity that may warrant further study into their mechanisms of action and feasibility for veterinary use.

Prevention and control: In domestic cats, identification and segregation of infected cats is considered the single most effective method for preventing new infections with FeLV. Feral cats should be excluded from contact with exotic cats in zoos. While retroviruses are generally unstable outside their host, they can remain viable in dried biological deposits for more than a week.

As with domestic cats, zoos should determine the FeLV status of all exotic cats. Cats should be tested for FeLV infection at quarantine and routine exams. If exotics cats are to be vaccinated, testing before initial vaccination is also recommended. However, since routine screening tests detect antigen, not antibody, vaccination does not typically interfere with FeLV testing.

Several injectable inactivated vaccines with adjuvants and a recombinant vaccine without adjuvants (designed for transdermal administration) are commercially available in the United States. The vaccine is not currently recommended as a core vaccine for exotic cats in zoos, but may be used in situations of high-risk, such as extensive exposure to infected feral cats.

When FeLV vaccination is determined to be appropriate, a two-dose primary series is recommended, with the first dose administered as early as 8 weeks of age followed by a second dose administered 3-4 weeks later. A single booster vaccination should be administered 1 year following completion of the initial series and repeated annually in cats that remain at risk of exposure.

FELINE LEUKEMIA VIRUS

Suggested disinfectant for housing facilities: Common hospital disinfectants and detergents will inactivate the retrovirus.

Notification: Not a reportable disease

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: The virus can be shed through casual contact such as grooming. Exotic felids have not been shown to maintain the infection. If a zoo felid is confirmed FeLV-infected, it may infect conspecifics, but the risk may be low.

Conditions for restoring disease-free status after an outbreak: Retroviruses are unstable outside their host and are quickly inactivated by detergents and common hospital disinfectants. However, retroviruses can remain viable in dried biological deposits for more than a week.

Experts who may be consulted:

American Association of Feline Practitioners guidelines on Retrovirus management are available online:

www.catvets.com/guidelines/practice-guidelines/retrovirus-management-guidelines

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FELINE PANLEUKOPENIA

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Felidae; limited other carnivores	Oronasal exposure to virus; transplacental	Depression, anorexia, severe dehydration, leukopenia	Subclinical to fatal	Fluid therapy, antibiotics, antiemetic, analgesics	Vaccination; disinfection	No

Fact Sheet compiled by: Ray Wack; updated by Christine Molter

Sheet completed on: 1 March 2011; updated 3 November 2012; updated 2 January 2018

Fact Sheet Reviewed by: James Evermann, Ray Wack

Susceptible animal groups: Felidae, mustelidae, procyonidae, viverridae, hyaenidae

Causative organism: Feline Panleukopenia virus (FPV [parvovirus]), in rare cases, canine parvovirus 2a, 2b or 2c

Zoonotic potential: None known

Distribution: Worldwide

Incubation period: 2-7 days, rarely up to 14 days

Clinical signs: Most cases are subclinical in cats > 1yr of age or those with partial protection from maternal antibodies. Most cases with illness are < 1 yr of age. Peracute cases may result in death. Acute cases present with fever, anorexia, depression, vomiting, diarrhea, hematochezia, severe dehydration, septic shock, and DIC. In transplacental infections, ataxia and tremors with normal mentation are observed in kittens due to cerebellar hypoplasia. Retinal lesions are also possible.

Post mortem, gross, or histologic findings: Virus replicates in and destroys rapidly dividing cells especially in bone marrow, lymphoid tissue and gastrointestinal tract mucosa. Transplacental infection may result in cerebellar hypoplasia, retinal dysplasia, embryonic resorption, fetal mummification, abortion, or stillbirth.

At necropsy, signs of sepsis and dehydration. Intestinal crypts can be dilated and contain sloughed epithelial cell debris. Blunting and fusion of villi may be present. Eosinophilic intranuclear inclusion bodies are rare.

Diagnosis: Hemogram often shows panleukopenia (WBC <3,000) with neutropenia being more common than lymphopenia, and thrombocytopenia and anemia. Fecal FPV antigens may be detected through an in-house immunochromatographic test kit, but antigen is present for short duration of time and false-negatives are possible. Definitive diagnosis can be made with IFA staining of tissue samples and PCR amplification and identification of virus DNA or virus isolation.

Material required for laboratory analysis: Serum titers can be used to document successful vaccination using hemoagglutination inhibition or indirect immunofluorescence testing. Tissue samples can be tested for presence of virus using fluorescent antibody staining of histopathology sections. Virus particles can be identified in feces using virus isolation, PCR amplification and identification of virus DNA, or electron microscopy.

Relevant diagnostic laboratories:
 Washington Animal Disease Diagnostic Lab
 Bustad Hall Room 155N
 Pullman WA 99164-7034
 Phone: 509-335-9696
 waddl@vetmed.wsu.edu
<http://waddl.vetmed.wsu.edu/>

 Animal Health Diagnostic Center
 College of Veterinary Medicine, Cornell University
 PO Box 5786

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240 Farrier Rd
 Ithaca, NY 14852-5786
 Phone: 607-253-3900
 Fax: 607-253-3943
<https://ahdc.vet.cornell.edu/>

Treatment: Aggressive fluid therapy is needed to correct dehydration, antibiotics to treat or prevent sepsis, antiemetic if vomiting, analgesia for abdominal pain, and nutritional support for hypoglycemia and anorexia. Leukopenia, thrombocytopenia, hypoalbuminemia, and hypokalemia are negative prognostic factors in domestic cats with panleukopenia.

Prevention and control:

Vaccination: Most cats produce a robust long lasting immunity following illness or vaccination. Vaccinated queens generally transfer protective levels of antibodies. The first vaccination is usually given at 6-9 weeks of age with booster vaccines given every 3-4 weeks with the last dose being administered when the kitten is > 16 weeks old, to ensure that interfering maternal antibodies do not inactivate the modified live virus or block vaccine response. A booster should be given 1 year later. Unvaccinated adults should be given a total of 2 doses of the vaccine 3-4 weeks apart. Vaccine titers suggest that triennial or longer booster intervals are effective after the initial series. Greater than 95% of domestic cats respond to primary vaccination series with protective titers that may last more than 7 years. A few non-domestic cats have been documented to be non-responders, so determination of titers is recommended.

Killed vaccines are often used in non-domestic cats due to rare cases of vaccine induced disease with modified live vaccines, though modified live vaccines are available. Pregnant, immunosuppressed, sick cats, or kittens < 4 weeks of age should not be vaccinated with a modified live product.

Fel-O-Vax (Boehringer Ingelheim) is a commonly used killed vaccine given as a 1 ml dose regardless of the size of the cat. A 0.5 ml dose Fel-O-Vax vaccine has also become commercially available recently.

Control: Virus sheds in all secretions in the acute phase and in feces for up to 6 weeks after recovery. Susceptible animals should not be with or in close proximity to positive animals until they have been vaccinated and/or protective antibody titers have been demonstrated. Transmission on fomites is common, thus stringent infectious disease control protocols are required. All surfaces should be disinfected with products labeled and proven effective against parvoviruses. It may also be necessary to bathe recovered animals especially if they are to be exposed to juveniles for whom vaccine protection cannot be assured.

Suggested disinfectant for housing facilities: Virus is very resistant to inactivation, survives for a long time in environment and is transmitted on fomites. Dilute household bleach, formaldehyde, glutaraldehyde or peroxygen disinfectants are effective.

Notification: None required

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Susceptible animals should not be introduced to infected animals until protective antibody titers have been demonstrated in the animals to be introduced. Viral shedding may occur for at least 6 weeks in infected animals and viral particles may remain infectious in the environment for more than a year.

Conditions for restoring disease-free status after an outbreak: Multiple swabs for PCR amplification and identification of FPV DNA should be collected from infected and exposed animals to assure that viral shedding has stopped.

Experts who may be consulted:

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FOOT AND MOUTH DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Artiodactylids (cloven-hooved animals), e.g. cattle, pigs, sheep, goats, cervids, African buffalo; also a few members of other orders (e.g., captive Asian elephants); Equids are not affected	Contact with affected animals (high concentrations of virus are present in FMD vesicles) or their bodily fluids (e.g., saliva, milk, semen), mechanical vectors (including people); ingestion (e.g., common source water or feed); insemination; aerosol (respiratory or oral); iatrogenic	Fever (2-3 days); vesicular lesions followed by erosions/ulcers on the tongue, lips, oral mucosa, teats and/or between the hooves; abundant stringy saliva; decreased appetite, lameness, abortion; myocarditis in young animals. Species with minimal signs (e.g., sheep) might not have characteristic vesiculation.	High morbidity (up to 100%) low mortality (except for young)	Depending on the phase and type of outbreak, infected animals and herds may be slaughtered. In a large outbreak, animals may be allowed to recover with palliative care	Importation bans (raw hides, trophies, unpreserved or uncooked animal products); surveillance test and slaughter, or quarantine until recovered; and disinfection of premises; strategic vaccination	Human cases seem to be very rare, with mild signs, and not of public health significance. Virus might also be carried mechanically in the nares for short periods

Fact Sheet compiled by: S. W. Jack; updated by James Roth and Gayle Brown

Sheet completed on: 31 March 2011; updated 28 April, 2018

Fact Sheet Reviewed by: Julie Napier

Susceptible animal groups: Artiodactylids (cloven-hooved animals), e.g., cattle, swine, cervids, antelope, buffalo, sheep, goats, giraffe, as well as a few members of other orders (e.g., Asian, but not African, elephants).

Causative organism: Foot and mouth disease virus (FMDV) *Aphthovirus* in family Picornaviridae. Multiple serotypes (O, A, C, SAT 1, SAT 2, SAT 3, Asia 1). Some strains primarily affect certain species (e.g., the pig O Cathay strain); Immunity to one serotype does not protect from other serotypes.

Zoonotic potential: Human infections seem to be very rare, with mild clinical signs and no public health significance. Many of these infections were reported in people exposed to large amounts of virus in vaccine plants, although cases also occurred after drinking FMDV-infected raw milk for several days.

Distribution: Endemic in parts of Asia, Africa, Middle East and South America

Incubation period: 2-14 days

Clinical signs: Fever (2-3 days); vesicles followed by erosions/ulcers on the tongue, lips, oral mucosa, teats and between the hooves; abundant stringy saliva if mouth is significantly affected; decreased appetite, lameness, abortion; sudden death from myocarditis in newborns; rare instances of sudden death in adults, especially in some severely affected wildlife species. The pattern of illness varies between species, and some species (e.g., sheep) can have minimal signs. Shedding may occur before the onset of clinical signs. Cattle may be persistently infected in the pharynx, but no evidence that they transmit infection. African buffalo can be long term shedders and transmit the virus.

Post mortem, gross, or histologic findings: Tongue/Oral: blanched foci to vesicles to complete ulceration with fibrin. Interdigital redness, vesicles or ulceration and similar on coronary bands are seen. Vesicles or erosions may also be found on udder, occasionally other sites. Myocardial pallor or streaking may be observed; young animals with myocardial lesions may not have vesicles. Lesions are species dependent, less severe in sheep and goats than cattle or swine. Location of lesions can also vary between species.

FOOT AND MOUTH DISEASE

Diagnosis: Grossly, it is indistinguishable from other vesicular diseases (vesicular stomatitis, swine vesicular disease, Seneca virus A, vesicular exanthema of swine). Other differential diagnoses include diseases with mouth and/or foot signs such as traumatic stomatitis, bovine virus diarrhea, bluetongue, malignant catarrhal fever, contagious ecthyma, and epizootic hemorrhagic disease of deer. Lab detection of FMDV is based upon virus isolation, antigen ELISA, and rRT-PCR. Serology tests for detecting exposure include virus neutralization and various ELISA assays.

Material required for laboratory analysis: Before collection of samples, proper authorities should be contacted and only send appropriate samples under secure conditions to authorized laboratories. Preferred sample is epithelium from un-ruptured or freshly ruptured vesicles, esophageal-pharyngeal (probang) samples. Other samples may include myocardium from heart failure deaths, milk and other secretions and excretions. For suspect carriers, esophageal-pharyngeal fluids should be submitted.

Relevant diagnostic laboratories: [National Animal Health Laboratory Network \(NAHLN\)](#), select FMD Laboratories for a list of the 45 labs approved for FMD diagnostics. FMD is a select agent and requires [BSL-3/BSL3 Ag](#). Limit access to building/lab, negative air pressure, HEPA filtered incoming air. Double HEPA-filtered air exit, all sewage treated, and work in specialized cabinets within lab.

Treatment: Depending on the phase and type of outbreak, infected animals and herds may be slaughtered. In a large outbreak, animals may be allowed to recover with palliative care.

Prevention and control: Avoidance of sources is most important. This approach can be via importation bans (raw hides, trophies, unpreserved/uncooked animal products); surveillance test and slaughter; or quarantine until recovered. Disinfection of premises is important as the virus could persist in environment possibly up to a few months especially under cold conditions. Virus is inactivated by acidification (pH <6) of muscle during rigor mortis, but can persist in other tissues (e.g., in bones, lymph nodes) if pH remains above 6.0. Vaccination has been applied in outbreaks.

Suggested disinfectant for housing facilities: Following removal of all organic debris (power-washer), most disinfectants will inactivate the FMD virus e.g. sodium hydroxide (2%), sodium carbonate (4%), citric acid (0.2%), acetic acid (2%), sodium hypochlorite (3%), potassium peroxydisulfate/sodium chloride (1%), and chlorine dioxide. Iodophors, quaternary ammonium compounds are less effective. [Use EPA-approved disinfectants for FMD.](#)

Notification: REPORTABLE DISEASE - Federal and State Animal Health Officials (AVIC and SAHO, respectively) must be notified. USDA-APHIS will contact the World Organization for Animal Health (OIE)

Measures required under the Animal Disease Surveillance Plan: Surveillance in zoos during an outbreak will be determined by the Responsible Regulatory Officials (Federal, State, or Tribal) depending on the epidemiology of the outbreak.

Contact USDA-APHIS:

Center for Epidemiology and Animal Health National Surveillance Unit

2150 Centre Avenue, Building B, Mailstop 2E6

Fort Collins, CO 80526-8117

national.surveillance.unit@aphis.usda.gov

<http://nsu.aphis.usda.gov/>

Measures required for introducing animals to infected animal: No animals should be introduced into the zoo until the disease is brought under control, or it is demonstrated that the zoo is free of infection and the animal to be introduced is also free of infection.

Contact USDA-APHIS:

Veterinary Services

APHIS, USDA

4700 River Road, Unit 41

Riverdale, MD 20737-1231

(301) 851-3595

FOOT AND MOUTH DISEASE

Conditions for restoring disease-free status after an outbreak: Surveillance to demonstrate absence of infection and absence of virus circulation, according to the [OIE Terrestrial Animal Health Code](#). Time to regain FMD free status varies with method of eradication and surveillance (e.g., use and type of vaccination). It may be possible for the zoo to be declared an FMD free compartment according to OIE Guidelines.

Experts who may be consulted: When FMD is suspected you must contact Federal and State Animal Health Officials (AVIC and SAHO, respectively); USDA-APHIS will contact the OIE.

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GIARDIASIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals	Contact with, or ingestion of contaminated food, water, soil, or animals.	Diarrhea, abdominal pain, bloating, dehydration, lethargy, anorexia	Generally mild	Fenbendazole; metronidazole	Personal and environmental hygiene	Yes

Fact Sheet compiled by: Gwen E. Myers

Sheet completed on: 1 February 2011; updated 15 August 2013

Fact Sheet Reviewed by: Joseph Camp; George D. Di Giovanni

Susceptible animal groups: Mammals; predominantly canids, felids, and humans.

Causative organism: *Giardia lamblia* (syn. *G. intestinalis*, *G. duodenalis*, etc.); currently, eight assemblages or genotype groups are identified and named A-H. Dogs are most commonly infected with Assemblages C and D, while cats are infected with Assemblage F. Humans are most commonly infected with Assemblages A and B with a few cases of E and F reported.

Zoonotic potential: Yes, although the taxonomic issues are under review to verify this conjecture as very few well-documented cases of zoonotic transmission have been published in the peer-reviewed literature.

Distribution: Worldwide

Incubation period: Time from exposure to clinical signs is generally 7-14 days although some infected animals show clinical signs as early as 5 days.

Clinical signs: Abdominal pain, diarrhea, gas or bloating, lethargy, dehydration, weight loss, anorexia, and vomiting are typical depending on severity of infection.

Post mortem, gross, or histologic findings: Histopathologic changes in the intestines vary from villous atrophy of the intestinal wall to hyperplasia of goblet cells and vacuolated epithelial cells.

Diagnosis: Two morphologic forms of *Giardia* exist: trophozoites and cysts. The trophozoite, a motile form, is binucleated, pear shaped, and flagellated. Diagnosis of *Giardia* infection can be difficult in that cysts are shed intermittently and are delicate, and artifacts (grass pollen, yeast, etc.) mimic to varying degrees the morphology of *Giardia* cysts. Microscopy of fresh feces may identify motile trophozoites that appear as a face with the two nuclei forming the eyes and median bodies forming the mouth. Mix a drop of fresh liquid feces with a drop of normal saline. Trophozoites are not often found in semi-formed or firm feces. Trophozoites have a concave ventral surface and a rapid "falling leaf" motion which may be the only motion visible may be the flagella. Duodenal fluid aspiration and examination of the sediment for motile trophozoites requires either endoscopy or exploratory laparotomy to obtain duodenal fluid, making this an impractical means of diagnosing *Giardia*.

Zinc sulfate fecal by centrifugation is better than zinc sulfate fecal flotation but due to intermittent shedding of cysts, the sensitivity is approximately 70%. Fecal ELISA tests identify *Giardia* specific antigens from trophozoites, avoiding the problem of intermittent cyst excretion in the feces. False negative ELISA results are not common; but a negative fecal ELISA does not eliminate the possibility of *Giardia* infection in an animal with appropriate clinical signs. Positive test results can occur in asymptomatic dogs and cats since some animals may harbor the organisms without having clinical signs.

IDEXX Laboratories has an in-house, quick SNAP *Giardia* test that is ELISA-based. Immunofluorescence

GIARDIASIS

(IFA) test identifies cysts. In one study, this test was the best single test for detecting subclinical giardiasis.
Material required for laboratory analysis: Feces
Relevant diagnostic laboratories: Any laboratory capable of diagnosing protozoal fecal parasites
Treatment: Fenbendazole and metronidazole have been used, although high doses have been associated with neurological dysfunction. Furazolidone (Furoxone® Suspension, SmithKline Beecham, 4 mg/kg BID for 7 days) is available as a suspension and is convenient to administer to cats and small dogs and has been shown to be effective in cats. Quinacrine (6.6 mg/kg BID For 5 days) has been shown to be 100% effective in dogs. Approximately half of the dogs treated developed minor and reversible anorexia, fever, or lethargy. Quinacrine has been shown to improve clinical signs in cats but not to eliminate infection; however, quinacrine is not currently available in the US.
Prevention and control: <i>Giardia</i> vaccine (Pfizer) is on the market but it is not intended to prevent infection in the vaccinated animal. Instead, the vaccine is licensed as an adjunct to treatment and is used to reduce the shedding of cysts by the vaccinated patient. Cysts are very resistant and can survive several months outside the host in wet, cold conditions, even water, but they are susceptible to desiccation in dry and hot conditions. Proper hygiene, especially to prevent human infection/zoonosis, is important.
Suggested disinfectant for housing facilities: Removal of organic matter prior to disinfection with bleach diluted to a 1:32 solution.
Notification: None required
Measures required under the Animal Disease Surveillance Plan: None
Measures required for introducing animals to infected animal: Recommended to keep infected or shedding animals isolated until clear of parasite.
Conditions for restoring disease-free status after an outbreak: Decontamination and disinfection of environment, and when possible, bathing animal prior to placing in cleaned environment.
Experts who may be consulted: Centers for Disease Control and Prevention Division of Foodborne, Waterborne, and Environmental Diseases Waterborne Disease Prevention Branch 1600 Clifton Rd Atlanta, GA 30333 800-CDC-INFO http://www.cdc.gov/ncezid/dfwed/index.html
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Animal group (s) affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primarily equids, also a risk to exotic felids, humans, dogs, cats, sheep, goats, camels, hamsters, mice, and guinea pigs	Injection, Ingestion, and inhalation of particles or direct contact between open skin or mucous membrane and infected tissue or secretions. Non-equid species often by ingestion of infected horse meat. Chronically infected asymptomatic horses are still highly infectious.	Loss of stamina, dyspnea, acute-coughing, high fever, nasal discharge and ulcers, epistaxis, fulminant septicemia Chronic cutaneous or generalized lymphadenopathy, and ulcerated skin nodules. Horses more commonly see chronic progressive form while acute sepsis is more common in donkeys and mules. Felids develop localized nodules on nasal mucosa and bloody nasal discharge within 8-14 days after consuming infected meat.	The course of infection is dependent on the route of exposure. Acute-aerosol/sepsis leads to death typically in 4-7 days to 3-4 weeks after onset of illness. Chronic form can last for years in horses with periodic relapses	Antibiotics may be used in endemic areas though will us. Need at least two given concurrently. Euthanasia required in non-endemic areas.	Strict entry requirements from endemic areas to non-endemic areas. CFT test and PCR used for diagnosis. Mallein ¹³ tests used commonly in endemic regions. Reportable in non-endemic areas to OIE and USDA as well as local veterinary authorities	Yes. Potential bio-terrorism weapon ⁷ . Tier 1 Select Agent

Fact Sheet compiled by: Annette Gendron

Sheet completed on: Updated 4 Sep 2018

Fact Sheet Reviewed by: Sarah Churgin

Susceptible animal groups

Equids are primarily affected with the chronic progressive form seen more often in horses while the acute form is more common in donkeys and mules. Other animals such as dogs, cats (including zoo & wild felids), sheep, goats, camels, wild cats, bears, wolves, hamsters, mice and guinea pigs are at risk. Cattle and pigs are resistant.

Causative organism

Burkholderia mallei (also previously designated *Pseudomonas mallei*, *Bacillus mallei*, *Pfeifferella mallei*, *Mycobacterium mallei*, *Loefferella mallei*, *Malleomyces mallei*, and *Actinobacillus mallei*), has been

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identified as the causative agent. It is a gram-negative, non-motile, non-spore forming, aerobic and facultative anaerobic bacillus as well as a facultative intracellular and host-adapted pathogen.

Zoonotic potential

Zoonotic transmission occurs through ingestion of contaminated meat, and injection, ingestion and inhalation of infectious particles or direct contact between open skin or mucous membranes and infected tissue or secretions.

Distribution

Re-emerging trans-boundary disease endemic with increasing outbreaks in parts of the Middle East, Asia, as well as Central and South America (also seen Africa). It has been eradicated from North America, Australia, Japan and Western Europe through surveillance and destruction of affected animals, and strict import restrictions.

Incubation period In natural infections - 4-7 days to several months/years.

Clinical signs

The organism is zoonotic with four basic forms in both horses and humans: cutaneous "farcy", upper respiratory, pulmonary and septicemic. In equids, the acute cutaneous form is more common in mules and donkeys, with death typically occurring in 4-7 days to 3- 4 weeks after onset of illness. The chronic cutaneous form of the disease is more common in horses and causes regional lymphadenopathy and skin nodules that ulcerate and drain, with induration, enlargement, and nodularity of regional lymphatics on the extremities and in other areas. The chronic form is characterized by flares and remissions over years. The acute respiratory form results in the highest mortality and may begin with ulceration of the nasal mucosa and nodules that secrete bloody discharge, often leading to sepsis. The stellate scars in the nasal mucosa from healed ulcers are considered characteristic of the disease. Nasal infections may spread to the lower respiratory system. The pulmonary form occurs in most clinical cases, often in combination with other forms of glanders and is characterized by nodular abscesses. The septic form of glanders results in coughing, a high fever and release of an infectious nasal discharge, often followed by fulminant septicemia and death within days. Multi-organ abscesses develop predominantly in the lung, liver and spleen and often lead to septic shock. Other lesions that can be seen are osteomyelitis, meningitis, orchitis or brain abscesses. Death may occur within 1-2 weeks or several months. Apparent survivors act as carriers and maintain the spread of the disease. Zoo and wild felids consuming infected meat will develop localized nodules on nasal mucosa and conjunctiva with bloody nasal discharge within 8-14 days after consuming contaminated meat.

Post mortem, gross, or histologic findings

Nodules, granulomas and ulcer formation seen in various tissues. Histopathologic lesions within the respiratory tract include vasculitis and thrombosis of vessels of the nasal mucosa with ulceration, suppuration and spread to the submucosa. In addition, glanders induces a neutrophilic leukocytosis and anemia caused by depressed erythropoietic activity in the bone marrow. Gram or Giemsa stains of lesion exudates may reveal the organisms.

Diagnosis

Clinical and bacteriological diagnosis of glanders is difficult in the early stages of the disease. Nearly 90% of infections exist as nonclinical or latent. Complement Fixation (CF) is the official test recommended by the OIE for international movement of equids. Unfortunately, in addition to false negative and false positive reactions, the test cannot differentiate *B. mallei* from *B. pseudomallei* or an infected from a "maleinized" (previously tested) animal. The "Mallein test" is the most commonly used test for glanders and uses a protein fraction of the glanders organism to test for a cell-mediated hypersensitivity response. It is injected intradermally i.e. intrapalpebral or is given topically by ocular drop. Palpebrae will swell markedly in 1-2 days in a positive reaction. The test is used more frequently in domesticated animals in endemic regions, but the sensitivity and specificity of the test depend largely on what protein fraction is used. The Mallein test may give a false positive by cross reaction with *Streptococcus equi*, *B. pseudomallei* or other *Burkholderia* spp. and may also leave the horse with a transient or permanent CF test for glanders and interfere with future serological testing. Culture of the organism is the gold standard. Due to its highly infectious nature, however, suspected isolates

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should only be sent to an accredited reference laboratory. Automated bacterial identification systems do not always correctly identify this organism, which can be a particular problem when the index of suspicion for *B. mallei* infection is low. Final differentiation of cultures of *B. mallei* from *B. pseudomallei* (melioidosis) can be done with rapid low risk DNA testing at a designated laboratory (with 16S rRNA sequencing and the use of a variety of molecular typing methods: *fliC* PCR, *flip* RT-PCR, et. A Western Blot test has been developed and an ELISA test using another immunoreactive protein is in development.

Material required for laboratory analysis: Serum and/or infected tissue

Relevant diagnostic laboratories

NVSL/USA – Complement Fixation (515) 337-7200

http://www.aphis.usda.gov/animalhealth/lab_info_services/about_nvsl.shtml

OIE: <https://www.fli.de/en/institutes/institute-of-bacterial-infections-and-zoonoses-ibiz/> (Germany) and

<http://www.cvrl.ae/contacts.php> (Dubai)

Treatment

No vaccine is available for animal or human use though several promising avenues are currently being pursued in rodent and on-human primate models. Information on antibiotic treatment is sparse and while gentamycin, azithromycin, doxycycline, ciprofloxacin and sulfonamides are thought to be effective for treatment in man and some laboratory animals, mortality would likely still be high and multiple antibiotics must be used concurrently. **As a rule, authorities forbid the treatment of glanders horses outside endemic areas. Animals diagnosed with glanders in non-endemic regions must be euthanized.**

Prevention and control

Any equids entering the US or other non-endemic countries must have a negative CF test for glanders.

Suggested disinfectant for housing facilities

Decontamination can be achieved with common disinfectants (solutions of benzalkonium chloride, 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, iodine, mercuric chloride in alcohol, and potassium permanganate), heat treatment to >72°C (130°F).

Notification

In suspected cases of glanders USDA-AVIC and state and local Veterinarians should be alerted. Internationally, cases should be reported to the Office International des Epizooties (OIE), the World Health Organization (WHO), and the state and local veterinary authority in each country.

Measures required under the Animal Disease Surveillance Plan Reportable disease

Measures required for introducing animals to infected animal

Infected animals would be isolated in endemic countries or euthanized in non-endemic countries.

Conditions for restoring disease-free status after an outbreak Disinfection and euthanasia recommended.

Isolation and testing of exposed animals if permitted.

Experts who may be consulted: NVSL and OIE personnel (see websites above)

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HANTAVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans, Rodentia, Chiroptera, Didelphimorphia, Soricomorpha	<p>Infection in rodents occurs horizontally, often associated with fighting.</p> <p>Humans are infected via inhalation of the virus in aerosolized urine, feces, or saliva; by direct contact with these materials; or by the bite of an infected rodent.</p>	<p>Early signs include fatigue, fever, myalgia, nausea, vomiting, and abdominal pain.</p> <p>Later signs include coughing, shortness of breath and tachycardia.</p> <p>Illness can progress rapidly to severe cardiorespiratory failure and shock.</p>	In humans, Sin Nombre Hantavirus has a 50% mortality rate.	No cure exists.	Avoid contact with wild and peridomestic rats and mice; rodent control; use appropriate personal protective equipment – especially respiratory - when infestations are severe.	Yes

Fact Sheet compiled by: Gerardo Suzán and A. Alonso Aguirre

Sheet completed on: February 21, 2018

Fact Sheet Reviewed by: Peter Black

Susceptible animal groups: Humans. Other mammal species may be infected through contact with rodents, but they are not known to have clinical signs or to transmit the virus to humans. Carrier rodents include cotton rat (*Sigmondon hispidus*), deer mouse (*Peromyscus maniculatus*), Rice rat (*Oryzomys palustris*), and white-footed mouse (*Peromyscus leucopus*).

Causative organism: Hantavirus (Sin Nombre) in the Americas causes a pulmonary syndrome while Old World hantaviruses in Eastern Asia cause hemorrhagic fever with renal syndrome and epidemic nephropathy in Europe.

Zoonotic potential: Yes, directly from rodents or their contaminated products.

Distribution: Hantavirus pulmonary syndrome is distributed in the Americas in rural areas in peridomestic settings (barns, outbuildings, and sheds). Old World hantaviruses that produce hemorrhagic fever with renal syndrome and epidemic nephropathy are reported in both rural and urban areas.

Incubation period: 1 to 5 weeks.

Clinical signs: In humans, early signs include fatigue, fever, myalgia (thighs, hips, back, and shoulders), nausea, vomiting, and abdominal pain. Later, up to 10 days post-infection, signs include coughing and shortness of breath, and tachycardia. Illness can progress rapidly to severe cardio-respiratory failure and shock.

Post mortem, gross, or histologic findings: Hantavirus pulmonary syndrome is characterized by a unique constellation of pulmonary, hematological, and reticuloendothelial pathological findings. Findings may include pleural effusions, alveolar edema and fibrin, and an interstitial mononuclear cell infiltrate. Immunoblast type cells in the lungs, blood, bone marrow, lymph nodes, liver, and spleen. Hematological findings include left-shifted neutrophilic leukocytosis, thrombocytopenia, hemoconcentration in severe cases, and circulating immunoblasts.

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Diagnosis: Detection of hantavirus-specific IgM antibodies or a 4-fold or greater increase in hantavirus-specific IgG antibody titer and detection of hantavirus antigen by immunohistochemistry in serum. Other tissues including lung, spleen, kidney, liver and heart can be used for Immunohistochemistry (IHC) and reverse transcriptase-PCR (RT-PCR) as post-mortem options.

Material required for laboratory analysis: Nobuto blood filter strips (Advantec Nobuto Blood Filter Strip, Cole-Palmer) is used with whole blood.

Relevant diagnostic laboratories:

Centers for Disease Control and Prevention, Viral Special Pathogens Branch
1600 Clifton Rd
Atlanta, GA 30333
Hotline (877) 232-3322
(404) 639-1510

Treatment: While no primary cure for hantavirus pulmonary syndrome, supportive treatment should include respiratory intensive care management and oxygen therapy. Ribavirin in treating hantavirus pulmonary syndrome has little effect.

Prevention and control: Avoid contact with wild and peridomestic rats and mice. Rodent control in and around houses, specially, if heavy rodent infestation is present. Ventilation helps to remove aerosolized virus inside structures prior to cleanup. While cleaning infested structures, use rubber boots or disposable shoe covers; rubber or latex gloves; protective goggles. Use appropriate respiratory protection when infestations are severe.

Suggested disinfectant for housing facilities: Two types of disinfecting solutions are recommended to clean up rodent materials.

1. General-Purpose Household Disinfectant --- Prepare according to the label, if not prediluted. Almost any agent commercially available in USA is sufficient as long as the label states that it is a disinfectant. Effective agents include those based on phenols, quaternary ammonium compounds, and hypochlorite.
2. Hypochlorite Solution (1:10 bleach solution) can be used in place of a commercial disinfectant. When using chlorine solution, avoid spilling the mixture on clothing or other items that might be damaged by bleach. Wear rubber, latex, vinyl, or nitrile gloves when preparing and using chlorine solutions. Chlorine solutions should be prepared fresh daily.

Notification: Request immediate notification of test results from the laboratory to the regional public health authority.

Measures required under the Animal Disease Surveillance Plan: Field researchers directly involved in disease ecology studies should follow the CDC guidelines for sampling small mammals for virologic testing (Mills et al., 1995).

Measures required for introducing animals to infected animal: Do not introduce infected animals to other places.

Conditions for restoring disease-free status after an outbreak: Thorough clean-up and disinfection and rodent control should be performed. Minimize contact of humans with rodents. Antibody and molecular surveillance in rodents and disease surveillance in humans.

Experts who may be consulted:

Centers for Disease Control and Prevention
Viral Special Pathogens Branch
1600 Clifton Rd
Atlanta, GA 30333
(877) 232-3322
(404) 639-1510

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Multiple taxa: primates; felids (primarily cheetahs, but lions, tigers and domestic cats, and other small felids have been reported); canids, rodents, ferret, sea otters. Disease is described best in humans, but induced and natural disease has been reported in multiple species.	Not well understood but probably through conspecific grooming and fecal-oral transmission.	Gastro-intestinal signs, primarily gastritis but hepatic and intestinal disease occurs in some species; signs range from asymptomatic to anorexia, vomiting, regurgitation, stomach ulceration, diarrhea with undigested food in feces, and weight loss.	Non-clinical or mild to severe; depending on immune status of animal and co-factors that are not well understood.	Multimodal symptomatic treatment to reduce <i>Helicobacter</i> spp. load can reduce gastric irritation and clinical signs, but reinfection/recrudescence is likely.	Difficult but iatrogenic exposure can be prevented through appropriate cleaning of endoscopy equipment.	Possibly

Fact Sheet compiled by: Copper Aitken-Palmer

Sheet completed on: 20 February 2018

Fact Sheet Reviewed by: Lily Parkinson

Susceptible animal groups: Humans are the most broadly susceptible group. Within the veterinary field, felids (in particular cheetah), ferrets, non-human primates and rodents are susceptible. Gastritis associated with *Helicobacter*-like organisms is a profound cause of morbidity and mortality in the cheetah (S. African cheetah, 40% of the mortalities; Cheetah Research Council indicated that 86% of cheetah study population is affected). A few reports of *Helicobacter*-like organisms have been reported in association with gastritis in other species including felids (bobcat, *Felis rufus*; Pallas cat, *F. manul*; Canada lynx, *F. lynx canadensis*; fishing cats, *F. viverrina*; margays, *F. wiedii*; sand cats, *F. margarita*; African lion, *Panthera leo*; snow leopards, *P. uncia*; Siberian tiger, *P. tigris altaica*; jaguar, *P. onca*), domestic dogs, southern sea otter (*Enhydra lutris nereis*) and non-human primates (cynomolgus monkeys). Laboratory induced infections to study *Helicobacter* spp. primarily have involved domestic ferrets, macaques, pigs, guinea pigs, hamsters and mice.

Causative organism: The genus *Helicobacter* was created in 1989 with approximately 20 species currently described across all taxa. The essential property of almost all *Helicobacter* spp. is the presence of sheathed flagella, and in most species, possession of strong ureolytic (urease producing) ability, particularly those associated with gastric mucosa. Considerable diversity in cell morphology is present with respect to cell length, number and location of flagella, and presence of periplasmic fibrils. *H. pylori* has a global distribution and infects human gastric mucosa (predominately the gastric cardia) with evidence for infection in cats. The most commonly described pathogenic species of *Helicobacter* include: *H. pylori* (human), *H. heilmannii* (cat, dog), *H. felis* (mouse model), *Helicobacter acinonychis* (formerly *H. acinonyx*; persists in the gastric fundus in cheetah), *H. mustelae* (domestic ferrets) and *H. enhydrae* (southern sea otter). *H. acinonychis* lacks the *cag*

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pathogenicity island (PAI), but is otherwise the most closely related *Helicobacter* to *H. pylori*. The PAI is the characteristic component causing the human neutrophilic inflammatory response, but has not been associated with *Helicobacter* spp. infecting cheetah. Multiple strains of *H. acinonychis* have been reported, but the demographic of these strains within North America and other populations of felids is poorly understood.

The urease produced by *Helicobacter* and the flagella allow the organism to survive in the gastric environment over a wide spectrum of pH, penetrate into gastric mucous layer, and reach the gastric epithelium where it can then attach to cells. Both cellular immune response and humoral response to *H. pylori* are believed to contribute to disease pathogenesis.

In cheetah, gastritis is associated with single species or multi-species infections of *Helicobacter* spp. (*H. pylori*-like, *H. heilmannii*, *H. felis*, or *H. acinonychis* (formerly *H. acinonyx*)). *Helicobacter*-associated gastritis causes morbidity and mortality in captive cheetah, but this reaction to *Helicobacter* spp. is not seen in free-ranging cheetahs when infected with the same *Helicobacter* spp. It has been hypothesized that immunomodulation caused by chronic stress (elevated glucocorticoids) or other factors may play a role in the pathogenesis of cheetah gastritis. Pet cats are frequently colonized by *H. heilmannii* without substantial correlation between infection and degree of gastritis. Differences in the pathogenicity of *Helicobacter* spp. across taxa are apparent, making understanding the pathogenesis, epidemiology and treatment difficult.

An occurrence of natural infection with *H. pylori* in a group of cynomolgus monkeys was associated with chronic active gastritis and gastric erosions. *H. pylori* were isolated from these monkeys in different countries within Asia with multiple strains isolated.

Zoonotic potential: The exact route of transmission of *H. pylori* among people is unknown. Several routes of transmission of *H. pylori* have been proposed including fecal-oral, oral-oral, gastro-oral, and via respiratory droplets. In humans, familial associated spread from person-to-person is suspected. Under controlled laboratory conditions, human sourced *H. pylori* has been shown to infect non-human primates. However, *H. pylori* occurring naturally in monkeys (or other species) are unlikely to represent a major route of transmission to humans, since close contact between nonhuman primates and humans is typically limited. *H. pylori* has been cultured from feline salivary and gastric sections, and *H. pylori* DNA has been found in in feline feces and dental plaque raising the possibility that *H. pylori* could be transmitted from cats to humans via saliva, vomit, or feces. *H. pylori* in humans can be excreted through several routes, with concentrations highest in vomitus. In developing countries, it is suspected that *H. pylori* may have an environmental reservoir (e.g. untreated water or contaminated food). Transmission of *Helicobacter* and subsequent clinical disease between humans and animals is poorly studied, but veterinarians should be careful and take personal protective precautions for potential exposure. In humans, *H. pylori* is associated with gastric cancer and is a known carcinogen of the stomach. Human medical endoscopists and endoscopy nurses have significantly higher rates of *H. pylori* than other medical professionals. Because of this, appropriate precautions using proper personal protective equipment (gloves, masks) should be used by veterinary staff conducting endoscopy, performing dental procedures, handling saliva or fecal material.

Distribution: *H. pylori* is the most common bacterial infection in the world affecting people, with estimates that it infects half of the people worldwide, but causes clinical disease in only a small percentage of those infected. The discrepancy between infection and clinical disease is a problem for physicians; it is difficult to discern when to treat patients. To help with this challenge, standardized human medical guidelines recommend only treating people suffering from peptic ulcer disease or mucosally associated lymphoma.

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The distribution of *Helicobacter* spp. in animals is poorly understood and under studied. Hand raised cheetah have been found to be *Helicobacter* negative until introduced to other cheetah (personal comm. S. Citino). But it can be assumed that most cheetah (free-ranging and captive) have been exposed to various *Helicobacter* spp. of varying strains.

Incubation period: Unknown

Clinical signs: Clinical signs range across taxa, but most are consistent with gastrointestinal signs. Cheetah with *Helicobacter*-associated gastritis display partial or full anorexia as the most common clinical sign leading to vomiting, regurgitation, diarrhea with undigested meat in feces, gastroesophageal reflux disease (GERD), acquired lower esophageal sphincter dysfunction, acquired hiatal hernia, and weight loss.

Clinical pathological, gross, and histopathological findings: *Helicobacter*-associated gastritis cannot be identified by gross evaluation of the stomach by endoscopy. Gastric ulcers can be identified via ante-mortem endoscopy evaluation or post-mortem gross evaluation, but further testing is needed to identify *Helicobacter*. As a spiral shaped bacterium, cytology can be helpful when diagnosing *Helicobacter*-associated gastritis. Histopathologic and immunological findings in cheetah with *Helicobacter*-associated gastritis are described as florid lymphocyte and plasma cell infiltrates within the gastric lamina propria and glandular epithelium, parietal cell apoptosis, leading to gland hyperplasia, goblet cell metaplasia, fibrosis and atrophy of the glandular fundus. Cheetahs with severe gastritis have larger numbers of active B cells and plasma cells.

Diagnosis: Rapid urease test, C-13-urea breath test (UBT), serology, gastric biopsy with histopathology (rec. minimum 5 biopsies for submission for gastritis & helicobacter evaluation), and touch cytology are all highly accurate invasive diagnostic tests for gastric *Helicobacter* organisms, whereas culture and polymerase chain reaction are the only means to identify *Helicobacter* to the species level.

Material required for laboratory analysis: Stomach (multiple fundic biopsies recommended for cheetah, ferret, dogs, and cats) biopsies (full thickness with mucosa) for histopathology, once initial diagnosis and grading of gastritis has been performed, non-invasive C-13-urea breath test (UBT) can offer an alternative to repeated biopsies for therapeutic monitoring.

Relevant diagnostic laboratories:

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Treatment: Triple therapy with a proton pump inhibitor (PPI), in combination with amoxicillin and clarithromycin is the established treatment for *H. pylori*. Metronidazole is used in the place of amoxicillin as part of the triple therapy for penicillin hypersensitive patients. Metronidazole is an important treatment for *Helicobacter*, but resistance among strains of *H. acinonychis* and *H. pylori* have been reported. For human cases of *H. pylori*, resistance to metronidazole has been reported in up to 80%, and resistance to clarithromycin in 2-10% of strains cultured. Resistance to one antibiotic, when triple therapy is attempted reduces the efficacy of therapy up to 50%. For *H. pylori*, quadruple therapy incorporating a bismuth compound with a PPI, tetracycline and metronidazole has been a choice for rescue therapy if triple medication course is not successful. Ranitidine-bismuth citrate has been shown to over-come metronidazole and clarithromycin resistance, and can be used in place of a PPI for rescue therapy as studied in humans. PPI triple therapy has been shown to provide

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the most consistent and durable therapy in humans. The exact mechanism by which PPI exert their effect on *H. pylori* eradication is not clear, but it is suspected that the potent acid suppression creates an optimal pH for bacterial growth and cell division allowing the key antibiotics amoxicillin and clarithromycin to act more effectively on the bacterium. *H. pylori* resistance to amoxicillin is not often reported, but amoxicillin is less effective when used alone on *H. pylori* than clarithromycin or metronidazole.

Because treatment of *Helicobacter* requires the use of several medications, compliance is a significant challenge to success. Resistance of *H. pylori* toward levofloxacin is rising worldwide, due to a point mutation reducing quinolone susceptibility. Because the quinolones are used for second line therapy when triple or quadruple courses are ineffective, a major concern for human medicine exists. Resistance to amoxicillin and tetracycline is low due to the need for multiple simultaneous mutations in genes. The comparison of drug resistance across different *Helicobacter* species is poorly studied, but *H. acinonychis* is used to model *Helicobacter* drug resistance.

In cheetah, optimal treatments are described as lansoprazole/clarithromycin/amoxicillin treatment group which produced a short-term decrease in inflammation when compared to controls. Lansoprazole has been shown to have direct bacteriocidal activity against *Helicobacter* spp. Prednisone should not be used because it has no effect on gastric inflammation and does not reduce *Helicobacter* load. Further treatment protocols recommend omeprazole/clarithromycin/amoxicillin or tetracycline/metronidazole/Pepto-Bismol for 28 days to achieve short-term *Helicobacter* eradication in cheetahs. Alternative treatments for delayed gastric emptying in cheetah associated with bacterial gastritis have been described using both Y-U pyloroplasty and incisional gastropexy. This procedure was combined with *Helicobacter* multi-therapy for tetracycline, metronidazole, and bismuth subsalicylate for one week.

Prevention and control: Personal protective equipment such as wearing barrier gloves and hand washing is recommended to prevent exposure. Proper cleaning of endoscopy equipment requires use of a detergent (enzymatic cleaner) and brush (mechanical cleaning over manual cleaning preferred) to remove blood, mucus, and tissue from the endoscope channels prior to disinfection. The World Congresses of Gastroenterology recommends that endoscopes be soaked in 2% activated glutaraldehyde for at least 10 minutes after cleaning to prevent transfer of *Helicobacter* between patients. Sterilization of biopsy forceps, or the use of disposable biopsy forceps is preferred to prevent transfer of *Helicobacter*. Typically, as biopsy forceps penetrate the gastric mucosa, they are difficult to clean and pose a significant risk for cross transfer among patients.

Suggested disinfectant for housing facilities: None

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: None

Conditions for restoring disease-free status after an outbreak: Because transmission is poorly understood, it is suspected there cannot be a disease-free status for susceptible species. *Helicobacter*-associated disease does not present as an “outbreak”. It is believed that secondary factors are necessary to result in clinical disease (i.e., gastritis) associated with *Helicobacter* across all taxa.

Experts who may be consulted:

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HELMINTHS OF UNGULATES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, mostly ungulates	Fecal/oral mainly from grazing on contaminated pastures.	Weight loss, progressive weakness, anemia, diarrhea, failure to thrive, ventral edema.	Large range with some cases mild, but infection can be fatal in animals with concurrent debilitating conditions	Anthelmintic treatment based on parasite susceptibility, pasture rotation, use of mixed species exhibits	Routine fecal examination and deworming based on these findings, promote good general health of the animals	Yes, but, with proper precautions, risk is low.

Fact Sheet compiled by: Rebecca Bloch

Sheet completed on: 9 June 2011; updated 30 October 2012

Fact Sheet Reviewed by: Thomas Craig; Holly Haefele

Susceptible animal groups: Ungulates, other mammals

Causative organism: Trematodes, cestodes, nematodes, acanthocephalans

Zoonotic potential: A risk of contracting *Trichinella* spp., *Spirometra* spp., or *Taenia* spp. is present from consumption of undercooked pork or beef, or eating watercress with *Fasciola* species attached. The public health significance is low and can be avoided with proper food safety.

Distribution: Worldwide, though the particular parasite of concern in a given area will vary by location, temperature, and moisture conditions.

Incubation period: Varies by parasite and environmental conditions and often larvae become dormant during unfavorable conditions both in the host and environment.

Clinical signs: These presentations depend on the type of infection, and the age, previous experience with the parasite, and health status of the animal, and may be absent in an otherwise healthy animal. In more severely affected animals, clinical signs may include weight loss, progressive weakness, anemia, diarrhea, and hypoproteinemia with development of subcutaneous edema especially in the intermandibular space and ventral abdomen.

Post mortem, gross, or histologic findings: Thin body condition with depletion of internal fat stores. Adult parasite presence in the organ it inhabits with possible associated inflammation of this tissue. Anemia and fluid in body cavities may also be seen.

Diagnosis: Sample 5-10% of animals in a herd situation, and more may be necessary based on housing and predisposition to being affected. Fecal egg counts can be performed quantitatively with tests like the McMaster's test for animals housed in larger groups or can be performed qualitatively with a simple float test for small numbers of animals. Quantitative fecal exams performed before and after deworming for a comparative fecal egg count reduction, fecal larval cultures, larval culture sensitivities, and pasture larval counts are recommended in areas facing large amounts of parasite resistance to anthelmintic medications.

Material required for laboratory analysis: Fresh fecal samples are optimal, if they can be analyzed within 1-2 hours, but otherwise refrigerate at 4°C. Samples kept in anoxic conditions do not develop and are useful for prolonged periods of time if cool. Refrigerated samples can be shipped over a 24-48h period to an outside lab packed with ice or other coolant, but do not freeze samples.

Relevant diagnostic laboratories: Most parasitology laboratories are capable of running larval cultures to speciate the parasite.

HELMINTHS OF UNGULATES

Treatment: Supportive care for animals that are debilitated by this infection. Anthelmintic administration based on parasite level and susceptibility is recommended. Anthelmintic resistance is a problem in some areas, an example being *Haemonchus contortus* in Texas. Drug alternatives such as copper oxide wire particles and bioactive condensed tannins can be used. The best time to make use of routine deworming (i.e., not clinically affected animals) is during the “off-season” when the parasites are in the host and not on the ground. Off season timing is determined by the specific parasites being targeted.

Prevention and control: Options for prevention include: pasture rotation; housing dead end hosts with definitive hosts (i.e., equids housed with ruminants); timely removal of feces to prevent eggs from developing into infective third stage larvae; use of elevated feeding stations or feed troughs to remove food sources from the ground; and reduction of numbers or elimination of intermediate hosts.

Routine monitoring of fecal parasite levels through fecal exams during peak larval parasite times of spring and summer, comparative fecal egg count reduction, fecal larval cultures, larval culture sensitivities, and pasture larval counts are recommended in problem situations. Characterization of the abundance and type of parasites present at post-mortem examination should be performed. Additional monitoring and treatment for neonates, lactating females, and other animals under higher stress conditions should be considered.

More recent avenues of control include the following. Creation of refugia by allowing for survival of some parasites through treatment of only the most affected animals to create a pool of parasites that are not resistant to the commonly used anthelmintics. These parasites can dilute the genetics from anthelmintic resistant parasites. Use of a nematode-trapping fungus, *Duddingtonia flagrans*, administered orally to reduce developing larvae numbers once they are deposited in feces. Work is being undertaken to create vaccines for specific parasites to reduce the impact of infection but these are not commercially available.

Suggested disinfectant for housing facilities: Remove fecal material promptly from enclosures. Appropriate sanitation and disinfection should be performed.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: To prevent introduction of a novel or resistant parasite to the resident population, quarantine with repeat fecal examinations is recommended. If possible the new animal should be housed on a dry lot or other surface that can be completely cleaned to prevent reinfection following anthelmintic treatment. Repeat fecal examination is recommended 7 days following treatment with at least two negative samples before the animal is introduced to pasture.

Conditions for restoring disease-free status after an outbreak: Re-establish a parasite control plan based on culture and parasite load. Remove as much fecal material from the environment as possible.

Experts who may be consulted:

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**HEMOTROPIC MYCOPLASMA
(HEMOBARTONELLOSIS, FELINE INFECTIOUS ANEMIA OR
HEMOTROPIC MYCOPLASMOSIS)**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Felids Canids Ursids Mice Cattle Swine	Blood sucking arthropods: fleas and ticks. Vertical: <i>in utero</i> , during parturition, or lactation. Horizontal: bite wounds. Iatrogenic: blood transfusion or infected needles.	Fever Anorexia Weight loss Anemia Tachycardia Splenomegaly Vomiting	Can vary from mild to severe and can lead to death in some animals.	Antibiotics; blood transfusion; steroids if an immune mediated component is suspected	Eliminate and prevent arthropods	Yes

Fact Sheet compiled by: Gretchen A. Cole

Sheet completed on: 21 June 2011; updated 5 March 2013; updated 9 January 2018

Fact Sheet Reviewed by: Sathya Chinnadurai

Susceptible animal groups: Felids, canids, ursids

Causative organism: Specialized mycoplasma bacteria that lack a cell wall and are small (diameter 0.1-1.0µm). They can be circular or bar shape, which aggregate and form pinion teeth on the surface of red blood cells.

Mycoplasma haemofelis (formerly *Hemobartonella felis* and *Eperythrozoon felis*). *M. haemocanis*, *M. haemomuris*, *M. wenyonii*, *M. haemosuis*

Zoonotic potential : Yes, one report in an immunodeficiency virus-infected human co-infected with *Mycoplasma haemofelis* and *Bartonella henselae* in Brazil

Distribution: Worldwide

Incubation period: In the domestic cat, it takes 2-17 days from infection until parasites are seen in blood. Peak parasitemia occurs over 1-5 days. Clinical signs generally begin 1 month after infection.

Clinical signs: Commonly, fever, anorexia, and weight loss are observed. Additionally, tachycardia, anemia, decreased hemoglobin, slight to moderate icterus, vomiting, and splenomegaly may be seen.

Post mortem, gross, or histologic finding: No pathognomonic postmortem findings are associated with this disease. Emaciation, splenomegaly (2-5 x normal size), friable spleen, icterus, and bone marrow hyperplasia may be observed

Diagnosis: Mycoplasmas cannot grow in culture media. PCR is the most reliable diagnostic test. Blood smear should be examined by direct microscopy before starting treatment. Organisms may be found in fresh, uncoagulated blood smear. These smears should be examined daily for 5-7 days since parasitemia is cyclic. Parasites are found on the surface of the erythrocyte (extracellular) or free in the smear. It is recommended to use Giemsa, Wright-Giemsa, May-Gruenwald-Giemsa, or Wright-Leishman stains to be able to differentiate this organism from stain precipitate, refractile artifacts, and *Cytauxzoon*; the latter is intracellular, normally found in the center and occurring singly. Direct Coombs' test may be positive in some species during the acute phase.

Material required for laboratory analysis: Microscope, blood smear slide, and stain. Blood in EDTA and standard blood shipment supplies to submit for PCR.

Relevant diagnostic laboratories: Most commercial veterinary laboratories can examine blood smears and submit a sample for PCR testing.

HEMOTROPIC MYCOPLASMA
(HEMOBARTONELLOSIS, FELINE INFECTIOUS ANEMIA OR
HEMOTROPIC MYCOPLASMOSIS)

<p>Treatment: Common treatments in domestic cat include tetracycline, doxycycline, or enrofloxacin. If severe anemia is present, consider glucocorticoid treatment such as prednisolone.</p>
<p>Prevention and control: Prevent and eliminate arthropod vectors (flea and tick control). Blood for transfusion should be PCR tested.</p>
<p>Suggested disinfectant for housing facilities: Standard cleaning and disinfection of areas to remove blood and control of ectoparasites should eliminate the organism from housing facilities</p>
<p>Notification: Currently none</p>
<p>Measures required under the Animal Disease Surveillance Plan: Currently none</p>
<p>Measures required for introducing animals to infected animal: Eliminate fleas and ticks. In nondomestic cats, negative animals have been housed with positive animals without evidence of horizontal transfer. However, since carrier state may occur, the possibility of transmission in animals with direct contact or close enough to share ectoparasites should be considered.</p>
<p>Conditions for restoring disease-free status after an outbreak: Treat affected animals, eliminate ectoparasites, and prevent exposure to new ectoparasites. Due to carrier state, may not be able to consider a population disease-free.</p>
<p>Experts who may be consulted: Joanne Messick DVM, DACVP Associate Professor Comparative Pathobiology Purdue University 625 Harrison Street West Lafayette, IN 47907 765-496-1748 jmessic@purdue.edu</p>
<p>References:</p> <ol style="list-style-type: none">1. André MR, Adania CH, Allegretti SM, Machado RZ. Hemoplasmas in wild canids and felids in Brazil. <i>J Zoo Wildl Med.</i> 2011;42:342-347.2. Dos Santos AP, dos Santos RP, Biondo AW, Dora JM, Goldani LZ, de Oliveira ST, Guimarães AMS, Timenetsky J, de Moraes HA, González FHD, Messick JB. Hemoplasma infection in HIV-positive patient. Brazil. <i>Emerg Infect Dis.</i> 2008;14:1922-1924.3. Haefner M, Burke TJ, Kitchell BE, Lamont LA, Schaeffer DJ, Behr M, Messick JB. Identification of <i>Haemobartonella felis</i> (<i>Mycoplasma Haemofelis</i>) in captive nondomestic cats. <i>J Zoo Wildl Med.</i> 2003;34:139-143.4. Harvey JW. Hemotropic mycoplasmosis (Hemobartonellosis). In: Greene, C.E. (ed.) <i>Infectious diseases of the dog and cat</i> 3rd ed. St. Louis (MO): Saunders Elsevier; 2006. p. 252-260.5. Harvey JW. Hemotropic Mycoplasmosis (Haemobartonellosis). In: Tilley L.P. and Smith F.W.K. (eds.). <i>Blackwell's Five Minute Veterinary Consult Canine and Feline</i>, 4th ed. Ames (IA); Blackwell Publishing; 2007. p. 591.6. Iso T, Suzuki J, Sasaoka F, Sashida H, Watanabe Y, Fujihara M, Nagai K, Harasawa R. Hemotropic mycoplasma infection in wild black bears (<i>Ursus thibetanus japonicus</i>). <i>Vet Microbiol.</i> 2013;16:184-189.7. Fard RMN, Vahedi S, Mohammadkhan F. Haemotropic mycoplasmas (haemoplasmas): a review. <i>Int J Adv Biol Biom Res.</i> 2014;2(5):1484-1503.8. Messick JB. Hemotropic Mycoplasmas (Hemoplasmas). MerckVetManual.com [cited 2018 Jan 9] Available from http://www.merckvetmanual.com/circulatory-system/blood-parasites/hemotropic-mycoplasmas

HEMOGREGARINES OF REPTILES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Reptiles, including tuatara	Vector-borne; vector varies by species or genera of parasite (e.g., leeches for <i>Haemogregarina</i> of aquatic turtles, ticks for <i>Hemolivia</i> , and a wide range of invertebrates for <i>Hepatozoon</i> in terrestrial reptiles)	Usually none.	Usually non-clinical. Mild disease may be observed in unnatural hosts.	No effective treatment known for reptiles. A decrease in parasitemias, but not clearance, has been noted with atovaquone-proguanil in one study.	Avoid contact with potential vectors. Captive animals should have effective and safe acaricides applied.	No.

Fact Sheet compiled by: Michael J. Yabsley

Sheet completed on: 7 August 2013, updated 2018.

Fact Sheet Reviewed by: Ellis C. Greiner; Guilherme G. Verocai

Susceptible animal groups: Reptiles. Certain genera are generally detected in certain groups but recent genetic data suggests that many of these parasite species have broad host ranges. For example, the genus *Haemogregarina* is most commonly reported from aquatic turtles whereas *Hepatozoon* has a wide range of reptilian hosts including snakes, lizards, tuatara, and tortoises.

Causative organism: There are currently four genera of haemogregarines (Apicomplexa: Adeleiorina) reported from reptiles.

- *Haemogregarina* are intraerythrocytic parasites that are most commonly reported from aquatic turtles. Leeches are the only known vectors for aquatic turtle parasites. Numerous other hosts, such as alligators, snakes, and tortoises have reported *Haemogregarina* spp. infections. However, these reports are based on morphologic data from only intraerythrocyte stages, which is insufficient to distinguish the genera. The absence of morphologic data for other life stages and vectors is a common problem among all genera and “species” of hemogregarines of reptiles that has hindered appropriate classification.
- *Hepatozoon* are intraleukocytic parasites that infect a wide range of reptiles - as well as mammals and birds. Many species infecting terrestrial reptiles previously classified within *Haemogregarina* were transferred to *Hepatozoon*, and so this change should be considered for a correct and up-to-date diagnosis.
- *Karyolysus* are intraerythrocytic parasites of lizards in the genera *Lacerta* and *Podarcis*. These parasites are transmitted by mites.
- *Hemolivia* are intraerythrocytic parasites of tortoises and lizards - and a few amphibians. Parasites with known life cycles utilize ticks in the genera *Amblyomma* or *Hyalomma* as vectors.

Zoonotic potential: None.

Distribution: Worldwide depending on range of appropriate hosts and vectors.

Incubation period: Highly variable. It is also unknown for most species as most species have only been detected in naturally infected hosts that have unknown histories. Generally, in experimental trials, parasites are not observed in the blood for several weeks.

Clinical signs: Generally no clinical signs are noted in natural hosts, although the parasites are often observed in blood smears during routine examination. Unnatural hosts (e.g., experimental studies or captive exotic animals) may exhibit lethargy and anorexia.

Clinical pathological, gross, and histopathological findings: Animals with very high parasitemias may develop mild anemia. Unnatural hosts may develop leukocytosis and elevated AST. Lesions are generally mild and microscopic. Intracellular stages can be observed in liver, lung, or spleen of vertebrate hosts. Granulomas are sometimes observed surrounding haemogregarine stages. Histopathologic lesions (e.g., necrosis and severe

HEMOGREGARINES OF REPTILES

inflammatory infiltrates surrounding parasite stages in liver and lungs) may be more severe in unnatural hosts.
Diagnosis: Examination of stained thin blood smears. Meronts can be observed by histologic examination of liver and other organs from tissues collected at post mortem examinations.
Material required for laboratory analysis: Thin blood smears fixed and stained for detection of intracellular parasite stages. Formalin fixed tissues for histologic evaluation for meronts.
Relevant diagnostic laboratories: Many diagnostic laboratories can examine blood smears and tissue sections for parasites.
Treatment: No effective treatment known for reptiles. A decrease in parasitemias, but not clearance, has been noted with atovaquone-proguanil in one study.
Prevention and control: Because the haemogregarines are vector-borne, limiting exposure of reptiles to ectoparasites is necessary to prevent transmission. Four acaricides (chlorpyrifos, cyfluthrin, lindane, and permethrin) proved efficacy against tick infestation in leopard tortoises.
Suggested disinfectant for housing facilities: It is not a matter of disinfection but rather prevention of exposure to ectoparasites and limiting environmental contamination with tick life-stages.
Notification: None.
Measures required under the Animal Disease Surveillance Plan: None.
Measures required for introducing animals to infected animal: These parasites are vector-borne so direct contact between animals is not a risk factor for infection. However, ectoparasite prevention should be implemented.
Conditions for restoring disease-free status after an outbreak: n/a
<p>Experts who may be consulted: Michael J. Yabsley Associate Professor College of Veterinary Medicine University of Georgia Athens, Georgia 30602 (706) 542-1741 myabsley@uga.edu</p>
<p>References:</p> <ol style="list-style-type: none"> Burridge MJ, Trevor F, Allan SA, Mahan SM. Evaluation of safety and efficacy of acaricides for control of the African tortoise tick (<i>Amblyomma marmoratum</i>) on leopard tortoises (<i>Geochelone pardalis</i>). <i>J Zoo Wildl Med.</i> 2002;33:52-57. Foronda P, Santana-Morales MA, Orós J, Abreu-Acosta N, Ortega-Rivas A, Lorenzo-Morales J, Valladares B. Clinical efficacy of antiparasite treatments against intestinal helminths and haematic protozoa in <i>Gallotia caesaris</i> (lizards). <i>Exp Parasitol.</i> 2007;116: 361-365. Krampitz HE, Haberkorn A. Experimental treatment of <i>Hepatozoon</i> infections with the anticoccidial agent toltrazuril. <i>Zentralbl Veterinarmed B.</i> 1988;35:131-137. Siddall ME. Phylogeny of adeleid blood parasites with partial systematic revision of the haemogregarine complex. <i>J Euk Microbiol.</i> 1995;42:116-125. Telford SR Jr. Hemoparasites of the Reptilia: Color Atlas and Text. Boca Raton (FL): CRC Press; 2008. 376 p. Wozniak EJ, Kazacos KR, Telford, Jr. SR, McLaughlin GL. Characterization of the clinical and anatomical pathological changes associated with <i>Hepatozoon mocassini</i> infections in unnatural reptilian hosts. <i>Int J Parasitol.</i> 1996;26:141-146. Wozniak EJ, Telford, Jr. SR, DeNardo DF, McLaughlin GL, Butler JF. Granulomatous hepatitis associated with <i>Hepatozoon</i> sp. meronts in a southern water snake (<i>Nerodia fasciata pictiventris</i>). <i>J Zoo Wildl. Med.</i> 1998;29:68-71.

HEPATITIS A

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans, non-human primates	Fecal-oral route	Occasionally mild illness (anorexia, lethargy, vomiting, fever, and diarrhea); liver enzyme elevation; jaundice common in humans	Rarely fatal; most recover acute infections without permanent liver damage	Rarely indicated, supportive care	Vaccination or immune globulin potentially	Yes

Fact Sheet compiled by: Zoltan S. Gyimesi

Sheet completed on: 4 February 2011, 21 August 2013; updated 30 January 2018

Fact Sheet Reviewed by: Ed Ramsay

Susceptible animal groups: Humans, non-human primates (chimpanzees, Old World monkeys, New World monkeys).

Causative organism: Hepatitis A virus (HAV); *Hepatovirus* genus, *Picornaviridae* family. Both human and simian strains.

Zoonotic potential: Yes

Distribution: Worldwide

Incubation period: 15-50 days, fecal-oral transmission. Following exposure and infection, virus can be shed in feces prior to seroconversion or clinical signs.

Clinical signs: Virus rarely causes clinical disease in non-human primates. Infected individuals can be viremic for up to 30 days prior to the onset of clinical signs. Seroconversion may be associated with transient liver enzyme elevation (AST, ALT, total bilirubin). Nonspecific illness (anorexia, lethargy, fever) or gastrointestinal disease is possible. Duration of viremia and fecal shedding can be 2 months or more.

Post mortem, gross, or histologic findings: Hepatocellular degeneration and necrosis, Kupffer cell proliferation, and lymphocytic periportal hepatitis can be observed. Histologic changes that may be present are similar to liver lesions caused by infection with this virus in humans.

Diagnosis: Blood testing for antibody/antigen. Liver biopsy/histopathology. Fecal PCR.

Material required for laboratory analysis: Whole blood, serum/plasma. Feces or liver potentially.

Relevant diagnostic laboratories:

VRL Laboratories
7540 Louis Pasteur Road, Suite 200
San Antonio, Texas 78229
877-615-7275
fax 210-615-7771
Anthony.Cooke@vrl.net

Zoologix Inc.
9811 Owensmouth Avenue, Suite 4
Chatsworth, California 91311
818-717-8880
818-717-8881 fax
info@zoologix.com

Treatment: This is not typically indicated. Supportive care can be provided.

Prevention and control: In humans, immune globulin (containing sufficient anti-HAV concentrations to be protective) or inactivated vaccine (typically for people at higher risk) can be administered. Vaccines can be given post-exposure during outbreaks per WHO.

Suggested disinfectant for housing facilities: Sodium hypochlorite or 2% glutaraldehyde.

HEPATITIS A

Notification: Public health officials may need to be notified if zoonotic (primate to human) transmission occurs.

Measures required under the Animal Disease Surveillance Plan: None currently

Measures required for introducing animals to infected animal: HAV can remain infective in stored feces for at least 30 days. If concerned about introduction of animals to a known infected animal, animals that are seronegative (i.e. not immune), the seronegative animal could be immunized with the human HAV vaccine prior to introduction. Similarly, a seropositive animal with negative stool samples (by PCR) is probably recovered and no longer infectious to others.

Conditions for restoring disease-free status after an outbreak: Clinically significant outbreaks are uncommon. Disease-free status should be obtainable via appropriate environmental disinfection, and making sure primates are either seronegative or seropositive but no longer shedding HAV.

Experts who may be consulted:

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References:

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HEPATITIS B VIRUS (HBV)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primates, esp. gibbons, woolly monkeys, apes, humans; also rodents, birds, bats.	Transmitted vertically (perinatal) or horizontal (percutaneous or mucosal exposure to infected body fluids, i.e. blood, saliva, sexual fluids, wound exudate)	Weight loss, lethargy, anorexia, icterus, abdominal discomfort, nausea, vomiting, pyrexia, joint pain	Often asymptomatic in non-human primates but can cause severe disease in gibbons and woolly monkeys; increased prevalence of hepatocellular carcinoma in chronic infections in woodchucks, humans.	Supportive care; antivirals or α -interferon can be attempted but to date unsuccessful in animal cases.	Human recombinant vaccine should be considered for non-exposed primates	Assumed but unproven

Fact Sheet compiled by: Ellen Bronson, med. vet., DACZM

Sheet completed on: 31 January 2011; updated 12 March 2013 and 14 January 2018

Fact Sheet Reviewed by: Sam Sander

Susceptible animal groups: Hepadnaviruses are divided into two genera: Orthohepadnavirus in mammals; Avihepadnavirus in birds. Orthohepadnavirus infect humans, apes, and rodents. Human Hepatitis B virus consists of at least 10 genotypes (A through J) with several sub-genotypes. Non-human primate hepatitis B viruses are species-specific and infect chimpanzees, orangutans, gorillas, gibbons, and woolly monkeys. Rarely reported or experimental in other primates (macaques, baboons, spider monkey, vervet monkey, and ruffed lemurs). Species-specific rodent hepadnaviruses also infect woodchucks, ground squirrels, and arctic squirrels. Most recently, hepadnaviruses have been identified in multiple species of bats. Avihepadnaviruses infect birds, including ducks, geese, herons, storks, cranes. Other hepadnaviruses have recently been identified in fish (African cichlid, white sucker, bluegill) and amphibians (Tibetan frog). Woodchucks and ducks are used as experimental models for hepatitis B in humans. Chimpanzees were historically used as a surrogate model for human HBV, but federal regulation in US no longer permits their use for invasive research.

Causative organism:

Orthohepadnaviruses (Mammals)

- Human hepatitis B virus (at least 10 genotypes most with several sub-genotypes)
- Chimpanzee hepatitis B virus (ChHBV)
- Orangutan hepatitis B virus (OuHBV)
- Gorilla hepatitis B virus (GoHBV)
- Gibbon hepatitis B virus (GiHBV)
- Woolly monkey hepatitis B virus (WMHBV)
- Woodchuck hepatitis virus (WHV)
- Ground squirrel hepatitis virus (GSHV)
- Arctic ground squirrel hepatitis virus (ASHV)
- Bat hepatitis virus

Avihepadnaviruses (Birds)

- Duck hepatitis B virus
- Heron hepatitis B virus
- Stork hepatitis B virus
- Crane hepatitis B virus
- Ross' goose hepatitis B virus

HEPATITIS B VIRUS (HBV)

- Snow goose hepatitis B virus
- Parrot hepatitis B virus

Zoonotic potential: Transmission of nonhuman primate hepatitis B viruses to humans is in theory possible although yet unproven; transmission of human HBV infection to non-human primates is well documented. Risk analysis should be performed for primate and veterinary staff in zoos and rehabilitation centers to assess need for vaccination against HBV.

Distribution: Multiple species and subspecies-specific and regional variants exist, but many are thought to cross-infect other species, although further epidemiologic and molecular studies are ongoing and needed. Recombination between ape variants has been proven. Infection has been shown in free-ranging chimpanzee, gorilla, orangutan, and gibbon populations.

Incubation period: 30 - 180 days (average 75 - 90 days)

Clinical signs: Infection can result in:

1. Acute transient or fulminant hepatitis, with fever, anorexia, lethargy, nausea, vomiting, icterus, abdominal discomfort, ascites. Increases in alanine transferase (ALT) and aspartate aminotransferase (AST) documented in several species.
2. Asymptomatic infection or mild disease and clearance of the virus with lifelong immunity.
3. Chronic hepatitis leading to liver failure or hepatocellular carcinoma. Increases in ALT and AST possible.

Post mortem, gross, or histologic findings: Hepatitis, hepatic necrosis, hepatic fibrosis is seen in humans, gibbons, and wooly monkeys, but rarely in other primates. Chronic infections can lead to hepatic cirrhosis and hepatocellular carcinoma in humans as well as in woodchucks, to a lesser degree in ground squirrels and ducks, but has not been reported in non-human primates. More cases with histologic and clinical disease may become evident as non-human primates diagnosed only in the past few decades age and develop chronic disease.

Diagnosis: Increased ALT and AST on biochemical analysis. Since the genome of human and non-human primate hepatitis B viruses are similar, human Hepatitis B testing is applicable in non-human primates as follows:

HBsAg+ and HBsAB- indicates active, acute or chronic infection;

HBsAg- and HBsAB+ indicates exposure but clearance of virus and natural immunity (or vaccination);

HBcAg+ indicates acute infection (< 6 mo);

HBcAB+ indicates acute or chronic infection; indicates previous exposure or chronic infected carrier status;

HBeAg+ indicates active virus production and infectivity;

HBeAg+ and HBeAB- indicates active virus production and high infectivity;

HBeAg- and HBeAB+ indicates low or no viral shedding and typically a predictor of long-term clearance of virus, but still potentially infectious;

PCR testing also available and indicates infectivity if positive.

Material required for laboratory analysis: Serum for liver enzyme analysis and serology testing; serum or whole blood EDTA or ACD for PCR testing.

Relevant diagnostic laboratories:

1. VRL Labs, P.O. Box 40100, 7540 Louis Pasteur, Suite 200, San Antonio, Texas 78229, Tel. 877-615-7275; www.vrl.net
HBsAg, HBsAB, HBcAB: 0.5-1.0 ml serum for each test required
Hepatitis B PCR: 2 ml fresh EDTA whole blood
2. Zoologix, Inc., 9811 Owensmouth Ave, Suite 4, Chatsworth, CA 91311; Tel. 818-717-8880.
<http://www.zoologix.com/primate/Datasheets/HepatitisB.htm>
Hepatitis B total antibody testing (ELISA): 0.5 ml EDTA whole blood or spun serum/plasma
Qualitative real time PCR: 0.2 ml EDTA or ACD whole blood, 0.2 ml plasma or serum, 0.2 ml fresh/frozen/fixed liver tissue

HEPATITIS B VIRUS (HBV)

Treatment: Supportive care, no specific treatment proven in non-human primates. In humans, tenofovir or entecavir and other nucleoside analog antivirals or α -interferon are given if high HBeAg+ and DNA+ and increased ALT (chronic active hepatitis). In humans, antivirals suppress the virus but do not provide a cure, while α -interferon cures a low percent of those treated for 1 year. Lamivudine and α -interferon have been attempted in limited cases in chimpanzees and woodchucks, respectively, without signs of improvement.

Prevention and control: Screen colony once and new animals at preshipment or quarantine examination with HBsAg or PCR. Avoid adding positive breeding animals to negative and unvaccinated groups. Vaccination can be used to protect negative (HBsAB- or PCR-) animals if exposed to positive animals. Two single antigen recombinant vaccines are currently available in the US in humans (Engerix B and Recombivax HB) and given at 0, 1, and 6 months with life-long immunity in humans.

Suggested disinfectant for housing facilities: 1:10 bleach; The virus can survive up to 7 days even on surfaces contaminated by dried blood/bodily fluids.

Notification: None

Measures required under the Animal Disease Surveillance Plan: N/A

Measures required for introducing animals to infected animal: Animal to be introduced should be tested, if (HBsAg- or PCR- and HBsAB-), animal should be vaccinated before introduced to positive (HBsAg+ or PCR+) animal.

Conditions for restoring disease-free status after an outbreak: Area should be completely cleaned and disinfected. All animals should be tested with HBsAg or PCR and HBsAB to determine status. Animals that are HBsAg- or PCR- and HBsAB- should be immunized.

Experts who may be consulted:

Robert E. Lanford, Ph.D.
 Southwest National Primate Research Center
 Department of Virology and Immunology
 Texas Biomedical Research Institute
 7620 NW Loop 410
 San Antonio, TX 78227
 210 258 9445
rlanford@txbiomed.org

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DUCK VIRAL ENTERITIS (DUCK PLAGUE)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ducks, geese, swan of all ages	Bird to bird contact or via environment; water is important for transmission. Spontaneous viral shedding by duck plague carriers, particularly during spring	Diarrhea, blood stained vent, cyanotic bill, inability to fly, convulsions, polydipsia, hypersensitivity to light	Moderate to severe	No effective treatment	Minimize exposure; vaccine for commercial flocks	No

Fact Sheet compiled by: Gwen E. Myers

Sheet completed on: 21 February 2011; updated 15 August 2013

Fact Sheet Reviewed by: Simone Stoute; Gary Riggs

Susceptible animal groups: Ducks, geese, swan - susceptibility varies greatly among waterfowl species (blue-winged teal > Canada goose > mallard, Muscovy > pintail); other aquatic birds do not become infected, with exception of two coots in Spain during an epizootic. All ages are susceptible. Juveniles may be more susceptible than adults, but in commercial waterfowl, adult breeders' mortality may be higher than young ducks. Sometimes higher mortality reported in females than in males. Carriers can produce infected offspring, which also may shed virus.

Causative organism: Herpesvirus, Anatid herpesvirus 1

Zoonotic potential: No

Distribution: North America, Europe, Asia, Africa

Incubation period: Bird to bird contact or via environment. Water appears important for transmission. Incubation period (exposure to death) is 3-7 days in domestic ducks, as long as 14 days in wild populations.

Clinical signs: Hypersensitivity to light with birds seeking cover and darkened areas, extreme thirst, droopiness, decreased egg production, bloody discharge from vent or bill, inappetence, ataxia, inability to fly, convulsions, and phallus prolapse. Birds can also have a characteristic 'cold sore' lesion under tongue especially during the carrier state.

Shedding: oral, cloacal, fecal, egg & from tissues and body fluids of carcasses. Spontaneous virus shedding by duck plague carriers, particularly during spring - may be related to physiological stresses of daylight duration change and onset of breeding.

Post mortem, gross, or histologic findings:

Gross:

-Buccal cavity: whitish plaques in pharynx occasionally.

-Esophagus: petechial to ecchymotic hemorrhages, necrotic/diphtheritic/cheesy membranous lesions along longitudinal folds on mucosal surface if slightly longer course, particularly caudal esophagus and common in swans.

-Proventriculus: focal mucosal hemorrhage and/or necrosis.

-Intestines: Hemorrhagic enteritis. Variable extent, from petechiation and small ulcers (e.g. in jejunum) to hemorrhagic/necrotic annular rings (ducks) or discs ('button ulcers') (geese, swans) in intestines (related to lymphoid tissue distribution) in ileum.

-Cloaca: mucosal hemorrhages, later necrotic/diphtheritic/caseous membranous lesions as in esophagus.

-Cardiovascular system: petechiae to paintbrush hemorrhages on surface, particularly at base and in coronary grooves (common) or in myocardium. May be particularly visible on pericardial fat.

DUCK VIRAL ENTERITIS (DUCK PLAGUE)

- Liver: Pinpoint hemorrhages (petechiae) and/or focal necrosis. May be swollen, friable, pale (copper colored).
- Thymus and bursa of Fabricius (young birds): hemorrhages, surrounding tissues edematous.

Histologic:

- Focal hemorrhages in most organs.
- Liver: Necrosis of hepatocytes, with hemorrhage and limited heterophil infiltration. Occasional areas of caseous necrosis with surrounding coagulation necrosis are observed.
- Gastrointestinal tract: Necrosis of epithelial cells sloughed into lumen that have been raised from surface by hemorrhage.
- Large eosinophilic intranuclear inclusion bodies may be found in: hepatocytes, bile duct epithelial cells, epithelial cells of esophagus, intestine, bursa of Fabricius, pancreatic cells and Hassall's corpuscles.

Diagnosis: Generally a post-mortem diagnosis. Viral isolation, mortality and lesions following animal sub-inoculation, serum neutralization, ID of a herpesvirus using EM, microscopic confirmation of viral intranuclear inclusion bodies in tissue cells and PCR.

Material required for laboratory analysis: Tissue samples; liver, lung, spleen, kidney, cloacal swabs.

Relevant diagnostic laboratories:

State Animal Disease Diagnostic laboratories

Texas Veterinary Medical Diagnostic Lab
College Station Laboratory
P.O. Box Drawer 3040
College Station, Texas 77841-3040
(979) 845-3414

Treatment: No successful treatment.

Prevention and control: Prevention aimed at minimizing exposure of the population-at-risk; depopulation, removal of birds from the infected environment, sanitation, and disinfection. Avirulent, live-virus vaccine developed for domestic white Pekin ducks but it is not reliable in protecting other species.

Suggested disinfectant for housing facilities: Virus is hardy, survives for weeks in ideal environmental conditions. Phenolic based disinfectants, Chlorine bleach; water may be decontaminated by chlorination (3ppm). Scrub concrete ponds with hypochlorite (5.25% solution).

Notification: Reportable to State Veterinarian and USDA-APHIS-VS involvement

Measures required under the Animal Disease Surveillance Plan: No known requirements as this is reportable for tracking/surveillance.

Measures required for introducing animals to infected animal: Not recommended - infected animals should be isolated or culled.

Conditions for restoring disease-free status after an outbreak: Quarantine, depopulate, clean and disinfect environment for captive flocks.

Experts who may be consulted:

Simone Stoute, DVM, PhD, DACPV
Director, Cornell University Duck Research Laboratory
Department of Population Medicine and Diagnostic Sciences
College of Veterinary Medicine
Cornell University
P.O. Box 217
Eastport, NY 11941
631-325-0600
sts66@cornell.edu

DUCK VIRAL ENTERITIS (DUCK PLAGUE)

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American Association of Zoo Veterinarians Infectious Disease Manual
ENDOTHELIOTROPIC ELEPHANT HERPESVIRUS (EEHV)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Asian elephants, especially ages 1-8 years. Rarely, African elephants.	Direct transmission between animals via trunk secretions and saliva or other body fluids.	Signs may be very mild. Edema of head, neck, trunk, legs. Cyanotic, swollen tongue. Lethargy, anorexia, mild colic, diarrhea, or constipation Lameness. Alterations in sleep patterns. Monocytopenia, thrombocytopenia.	Can be fatal in young elephants, if not – and even when – promptly treated. It may cause ulcers or vesicles in mouth and on vaginal mucosa. Milder clinical or sub-clinical forms exist. In African elephants, carrier state also exists in lymphoid lung nodules, and possibly reactivated in skin nodules.	Antiviral medications: Famciclovir orally or rectally. Ganciclovir intravenously, acyclovir orally, rectally or intravenously. Supportive care: intravenous fluid support, plasma transfusions. see eehvinfo.org for more	It is recommended to run PCR on whole blood of Asian elephants 1-8 years of age weekly to detect early viremia before clinical signs.	No.

Fact Sheet compiled by: Lauren Howard

Sheet completed on: 25 January 2011; updated 3 January 2013, January 3, 2018

Fact Sheet Reviewed by: Gretchen Cole

Susceptible animal groups: Infant and juvenile Asian (and very occasionally African) elephants are more likely to die from EEHV Hemorrhagic disease than older animals although there are reported cases of fatalities in elephants up to 40 years old. Adult elephants of both species may be subclinical carriers or may display a milder form of the disease with intermittent oral and vaginal/vestibular lesions.

Causative organism: Elephant Endotheliotropic Herpesviruses (EEHV) = Novel genus names Probosciviruses. Several types and multiple strains have been isolated and identified from fatal cases and clinically ill elephants.

Zoonotic potential: None known.

Distribution: Seven related species/types of EEHV have been identified in captive Asian or African elephants throughout the world. Multiple cases of the same hemorrhagic disease have been identified in wild Asian calves in Asia. Most captive and all wild elephants likely carry several EEHV types in a latent state. EEHV 1, 3/4 (test used does not distinguish between 3 and 4) and 5 detected in trunk washes of clinically normal camp elephants in India.

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ENDOTHELIO TROPIC ELEPHANT HERPESVIRUS (EEHV)

EEHV Strain	Species	Clinical Picture
1A	Asian	Hemorrhagic disease
1B	Asian	Hemorrhagic disease
2	African	Hemorrhagic disease; lung nodules, skin nodules
3	Asian/African	Hemorrhagic disease; lung nodules, skin nodules
4	Asian	Hemorrhagic disease
5	Asian	Hemorrhagic disease
6	African	Lung nodules
7	African	Lung nodules, skin nodules

Incubation period: In retrospective analysis of clinical cases, EEHV viremia has been detectable on quantitative PCR of whole blood 1 to 2 weeks prior to clinical signs. It is suspected that EEHV infections usually remain latent with sporadic subclinical reactivation (i.e., shedding in trunk washes or other secretions) throughout the lifetime of an infected elephant.

Clinical signs: Changes in hemogram (monocytopenia, thrombocytopenia) may occur before clinical signs of illness. Initial signs may be mild or vague: lethargy, decreased food or water intake, mild colic, diarrhea, lameness or stiffness, oral mucosal lesions, ocular lesions, and alterations in sleep patterns may occur. More severe signs include edema of the head, neck, trunk, and thoracic limbs, and lingual cyanosis.

Post mortem, gross, or histologic findings: Gross necropsy findings may include pericardial effusion with diffuse petechial hemorrhages throughout the heart, tongue, and visceral surfaces. Lingual cyanosis and hepatomegaly may also be seen. Ulcerations of the oral cavity, larynx and large intestine have been seen. Histologic findings may include extensive microhemorrhages and edema in the heart and tongue, with lymphocytic, monocytic, and neutrophilic infiltration of the myocardium. Capillary endothelial cells of the myocardium, tongue, and hepatic sinusoids may contain amphophilic to basophilic viral inclusion bodies. These herpesviral particles are usually intranuclear, and occasionally intracytoplasmic, but have not been seen outside of cells.

Diagnosis: PCR on whole blood detects viremia. . Viremia may be low and remain low/subclinical or may increase and lead to EEHV Hemorrhagic Disease, serial testing is recommended in any viremic elephant. Close evaluation of the hemogram can identify early monocytopenia and thrombocytopenia. Virus can also be detected in serum of severely affected animals. Post-mortem PCR analysis can be done on heart, liver, tongue, intestines, and any other hemorrhagic tissues. PCR on lung nodules can be performed; it is important to note that several EEHV types have been found by PCR in lung nodules of asymptomatic carrier Africans, but not known yet in asymptomatic Asians. Trunk wash or saliva swab PCR may demonstrate EEHV shedding, which occurs sporadically in most elephants at one point or another.

Material required for laboratory analysis: Whole blood in EDTA tube, frozen (PCR).
 Frozen serum (PCR, future antibody testing – no test available as of 2018).
 Transfer all liquid samples to plastic tubes before shipping
 Heart, liver, spleen, lymph nodes, intestines, skin or mucosal nodules, lung nodules, frozen (PCR).
 Cell pellet from centrifuged trunk wash (real-time PCR), or saliva swab in DNA preservative. Fresh, unfrozen samples of serum, positive trunk washes and lesions needed for attempts at cell culture.

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ENDOTHELIO TROPIC ELEPHANT HERPESVIRUS (EEHV)

Relevant diagnostic laboratories

Smithsonian, National Zoological Park
Department of Pathology
Attn: Erin Latimer/Laura Richman
3001 Connecticut Ave NW
Washington, DC 20008
(202) 633-4252
(703) 855-9611
latimere@si.edu
Please call or/email before sending samples

Baylor College of Medicine, Department of Virology and Microbiology
Performs quantitative real time PCR on whole blood or trunk wash samples.
For details, contact: Dr. Paul Ling: pling@bcm.edu
Lab phone: (713) 798 8475
Cell phone: (281) 460 1696

Treatment: Famciclovir (8-15 mg/kg orally or rectally TID) for Asian elephants has been reported (Brock *et al* 2012). Ganciclovir has also been used but must be given intravenously. Acyclovir has been used in several Asian countries.

Prevention and control: Weekly blood collection for whole blood PCR is recommended for Asian elephants 1 to 8 years of age, to detect viremia early on in disease process and allow for early treatment. Weekly CBCs may also help detect early viremia. Bank frozen whole blood, serum and trunk wash on all clinical elephants and herd mates for potential future study. Once prevalence is known from ongoing investigations, informed decisions can be made in regards to movement of individual elephants between populations.

Suggested disinfectant for housing facilities: Bleach diluted to 1:10 solution in water is often used to disinfect surfaces contaminated with most herpesviruses, although it has not been proven to inactivate EEHV.

Notification: No special notification process required.

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: Calves should not be isolated from their dams unless necessary to facilitate treatment.

Conditions for restoring disease-free status after an outbreak: No cure is available for latent herpesviral infection. It is assumed to be endemic in both Asians and Africans.

Experts who may be consulted:

Website with information on detection, treatment, etc: www.eehvinfo.org

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Smithsonian's National Zoological
Department of Pathology
3001 Connecticut Ave. NW
Washington, DC 20008
202-633-4252
(703) 855-9611
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Dennis Schmitt DVM (treatment advice)
203 Karls Hall
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ENDOTHELIO TROPIC ELEPHANT HERPESVIRUS (EEHV)

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American Association of Zoo Veterinarians Infectious Disease Manual
**EPSTEIN-BARR VIRUS (*HUMAN HERPESVIRUS 4*), EBV-RELATED
 LYMPHOCRYPTOVIRUSES**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p>EBV affects primarily humans</p> <p>EBV-related viruses affect a wide range of NHP (Old World monkeys, apes, some New World species)</p> <p>Lymphocryptovirus has been associated with carcinomas of sea lions.</p>	Direct contact through saliva	<p>EBV in man mostly asymptomatic, but can manifest as Infectious Mononucleosis, Burkitt's lymphoma, nasopharyngeal carcinoma, hairy leukoplakia, immunodeficiency-associated lymphoproliferative disease. In other primates, lymphoproliferative disorders are presented.</p> <p>EBV-related viruses: usually asymptomatic, may cause lymphoproliferative disorders.</p>	Fatal in cases of malignant tumors or lymphomas.	None reported.	Ubiquitous.	Yes, possible zoono-ponotic disease.

Fact Sheet compiled by: Sam Rivera; updated by Jan Ramer

Sheet completed on: 1 June 2011; updated 23 July 2013

Fact Sheet Reviewed by: Sam Rivera; Kevin Brunner

Susceptible animal groups:

EBV: Pongidae

EBV-related simian viruses: Old and New World NHP.

Causative organism: Epstein-Barr virus (*Human herpesvirus 4*), EBV-related lymphocryptoviruses (LCV): *Papiine herpesvirus 1*, *Cercopithecine herpesvirus 14*, *Macacine herpesvirus 4*, *Panine herpesvirus 1*, *Pongine herpesvirus 2*, *Gorilline herpesvirus 1*, *Callitrichine herpesvirus 3*.

Zoonotic potential: Yes

Distribution:

EBV in man Worldwide

EBV-related LCV found in most old world NHP and some new world NHP.

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**EPSTEIN-BARR VIRUS (*HUMAN HERPESVIRUS 4*), EBV-RELATED
 LYMPHOCRYPTOVIRUSES**

Incubation period: Variable
Clinical signs: These diseases are presented mainly as asymptomatic infections. EBV in man can cause infectious mononucleosis, nasopharyngeal carcinomas, Burkitt's lymphoma, non-Hodgkin's lymphoma; in great apes can possibly cause malignant lymphomas; in macaques axillary/inguinal lymphadenopathy, similar to EBV induced infectious mononucleosis in humans without pharyngitis and splenomegaly, B-cell lymphoma, epithelial hyperkeratotic lesions on oral cavity, esophagus, chest, hands, and genitalia; in baboons malignant lymphoma; in orangutan leukemia; in gorilla B-cell lymphoma; in common marmoset B-cell lymphoma.
Post mortem, gross, or histologic findings: Lymphoproliferative disorders.
Diagnosis: Virus isolation, serology, PCR, Histopathology, Immunohistochemistry.
Material required for laboratory analysis: Serum, whole blood, lymph nodes, spleen, bone marrow.
<p>Relevant diagnostic laboratories:</p> <p>Pathogen Detection Laboratory, California National Primate Research Center, Road 98 & Hutchison, University of California Davis, California 95616 (530) 752-8242 Fax: (530) 752-4816 PDL@primate.ucdavis.edu http://pdl.primare.ucdavis.edu</p> <p>BioReliance, Serology/PCR Laboratories 14920 Broschart Rd. Rockville, Maryland 20850 (301) 610-2227 (310) 610-2587 ahs@bioreliance.com</p> <p>Virus Reference Laboratories, Inc. 7540 Louis Pasteur Road San Antonio, Texas 78229 (887) 615-7275 Fax: (210) 615-7771</p> <p>Zoologix Inc. 9811 Owensmouth Avenue, Suite 4 Chatsworth, California 91311-3800 818-717-8880 Fax: 818-717-8881 info@zoologix.com</p>
Treatment: None reported.
Prevention and control: Ubiquitous in captive collections.
Suggested disinfectant for housing facilities: Lipid solvents, soap, UV-light, heat.
Notification: None at this time.
Measures required under the Animal Disease Surveillance Plan: None at this time.
Measures required for introducing animals to infected animal: Ubiquitous in captive collections

American Association of Zoo Veterinarians Infectious Disease Manual
**EPSTEIN-BARR VIRUS (*HUMAN HERPESVIRUS 4*), EBV-RELATED
LYMPHOCRYPTOVIRUSES**

Conditions for restoring disease-free status after an outbreak: Latent infections preclude establishment precludes a disease-free status after an outbreak.

Experts who may be consulted:

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EQUINE HERPESVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals – equids primarily; recombinant EHV1/9 strains have been shown to infect ursids, artiodactylids, camelids, rhinoceros, and rodents.	EHV1: aerosol droplets, contact, fomite Infected foals, etal membranes and aborted fetuses are highly contagious	EHV1: abortion in mares and mild respiratory disease in horses <2 years; neurologic form more common in older animals, signs range from ataxia to paralysis and death	EHV1: mild to severe possible, outbreaks if uncontrolled EHV3: generally mild, with lesions resolving within two weeks.	EHV1: supportive care for encephalomyelitis. EHV3: topical antiseptics to prevent infection and reduce discomfort	EHV1: vaccination all pregnant mares, isolation of known cases EHV3: no vaccine available, isolation of cases. EHV4: vaccination of horses <5 years old EHV9: no vaccine available	Not reported.
	EHV3: sexually transmitted, flies feeding on vaginal discharge of infected mares, fomites	EHV3: ulcers along cutaneous mucous membranes, especially genital tract.	EHV4: mild infections, secondary bacterial infections can increase severity	EHV4: supportive care EHV9: supportive care, seizure control		
	EHV4: aerosol droplets, contact, fomite	EHV4: respiratory disease in horses < 2 years old. Depression, nasal discharge, fever. Rarely causes abortion in pregnant mares.	EHV9: ranges from mild illness to severe disease, with progression in a short time period			
	EHV9: unknown, fomite transmission suspected	EHV9: neurologic signs in affected aberrant hosts, including ataxia, seizures, and progressive disease				

EQUINE HERPESVIRUS

Sheet completed on: 1 August 2013
Fact Sheet Reviewed by: Ray Wack; John Vacek
Susceptible animal groups: Equids [EHV 1, 3, 4]; exotic/zoo cases of infection with a recombinant EHV1/EHV9 virus have been published in onager (<i>Equus hemionus</i>), polar bear (<i>Ursus maritimus</i>), Grevy's zebra (<i>E. grevyi</i>), plains zebra (<i>E. quagga</i>), blackbuck (<i>Antilope cervicapra</i>), Thomson's gazelle (<i>Eudorcas thomsonii</i>), reticulated giraffe (<i>Giraffa camelopardalis reticulata</i>), llama (<i>Lama glama</i>), alpaca (<i>Vicugna pacos</i>), black bear (<i>Ursus americanus</i>), guinea pig (<i>Cavia porcellus</i>), and Bactrian camel (<i>Camelus bactrianus</i>). Experimental infection has been demonstrated in Syrian hamsters (<i>Mesocricetus auratus</i>), domestic dogs (<i>Canis lupus familiaris</i>), and domestic pigs (<i>Sus scrofa</i>).
Causative organism: Equine viral abortion (Equine Herpesvirus 1 [EHV1]); Equine herpes myeloencephalopathy (EHV1); Equine coital exanthema (EHV3); Equine rhinopneumonitis (EHV4); Gazelle herpesvirus 1 (EHV9)
Zoonotic potential: No evidence for potential zoonosis is associated with any EHV strain.
Distribution: EHV strains are endemic worldwide, with no specific distribution pattern. EHV1/9 can be carried by exotic equids with no clinical signs.
Incubation period: <u>EHV1:</u> Abortion in pregnant mares 2-4 weeks following exposure. Lifelong infection, with potential for recrudescence during stress or treatment with steroids. Neurologic form incubation averages 3-8 days but up to 14 days <u>EHV3:</u> As short as 2 days. <u>EHV4:</u> 2-10 days following exposure. <u>EHV1/9:</u> recombinant: unknown
Clinical signs: <u>EHV1</u> abortion: Sporadic or abortion "storm" can be observed. Spontaneous abortion of fetus within amniotic membranes in pregnant mares with no premonitory signs in the last trimester of gestation. Foals that are born alive are extremely weak and die within days. <u>EHV1</u> encephalomyelopathy: Encephalomyelitis varies in severity. Mild cases are noted with slight ataxia, urinary incontinence, flaccid tail, decreased anal tone, limb edema and pyrexia. Severe cases result in paralysis, seizures, blindness, and ultimately death. Paresis and paralysis are often noted with an ascending pattern from the hindlimbs. Colic, ocular lesions, anorexia, and pyrexia are also reported. Mild cases may resolve uneventfully. <u>EHV3:</u> Vesicular and ulcerative lesions are noted on the superficial mucosa of the external reproductive organs. Lesions are transient and heal in several weeks, leaving spots of depigmented skin. Stallions may be reluctant to breed. Affected horses may become life-long carriers, with flare-ups possible. <u>EHV1/4</u> respiratory disease: Most common in foals older than 2 months, when maternal immunity is waning. Increased rectal temperature, serous to mucopurulent nasal discharge, anorexia, and depression, with recovery by 3 weeks. Clinical signs are uncommon in horses over 2 years of age. Abortion in pregnant mares may occur rarely. <u>EHV1/9</u> recombinant: Range of clinical signs, usually results in neurological disease in affected animals. Polar bears and black bears have been reported with tremors, excessive blinking, ptialism, opisthotonos, seizures and progressive neurologic disease. A giraffe was euthanized due to ataxia, incoordination, abdominal pain, and a progressively deteriorating condition. Thomson's gazelles have been reported with recumbency, seizures, and progressive neurologic disease. EHV related abortion has been reported in a Asian rhino. Guinea pigs housed in the same building as affected Thomson's gazelles were reported with abortion, hindlimb paralysis, and ataxia.

EQUINE HERPESVIRUS

Post mortem, gross, or histologic findings:

EHV1 abortion: splenomegaly, grey necrotic foci in liver and pleural/peritoneal edema in aborted fetuses. Herpesviral intranuclear inclusion bodies in affected tissue.

EHV1 encephalomyelopathy: Cases are noted with areas of hemorrhage throughout the CNS, and vasculitis and thrombosis of neural endothelial cells, with ischemic necrosis histologically.

EHV3: ulcers and vesicles on the vaginal, vestibular, vulvar, preputial, or penial mucosa of affected horses. Similar lesions may also be noted on oral mucosa or teats.

EHV1/4 respiratory disease: Focal areas of necrosis in liver, spleen, and lungs with intranuclear inclusion bodies. Bronchointerstitial pneumonia may be noted when infected with secondary bacterial infections.

EHV1/9 recombinant: Nonsuppurative encephalitis, with or without lymphohistocytic cuffing, multifocal gliosis, and vasculitis have been reported in a variety of affected species. Intranuclear inclusion bodies are sporadically reported.

Diagnosis:

EHV1 abortion/encephalomyelopathy:

- Pathology: Based on gross and histologic pathology in aborted foals, increased likelihood if intranuclear inclusion bodies are noted. Vasculitis in CNS tissue of encephalomyelitis cases.
- IHC: demonstrates viral presence in affected tissues
- CSF analysis: positive EHM horses typically have xanthochromia with increased protein. A monocytic pleocytosis is variably present. CSF samples are not accurate for PCR or ELISA testing.
- Viral isolation: (gold standard) Growth in horse and rabbit cell cultures, allows differentiation from EHV4 which only grows on equine cell cultures. Isolation from nasal swabs or blood samples of neurological horses, best results when taken during initial pyrexia. High viral burdens are more likely to have rapid turnaround time.
- PCR: can detect viral presence in collected tissues, including nasal swabs or uncoagulated (EDTA) blood, at low levels. Non-quantitative is run more routinely, but quantitative real-time is available.
- Paired serology: fourfold or greater increase in virus neutralizing antibody titers, or a single titer of 1:256 or higher, are consistent with positive diagnosis. However, this approach cannot distinguish between EHV1 and EHV4.
- ELISA: test pregnant mare serum when fetal tissues are not available to diagnose.

EHV3:

- Clinical: based on physical exam findings
- Paired serology: comparison of acute and convalescent serum samples for a rise in antibody titers.
- Electron microscopy: investigation of clinical samples, including scrapings from the affected mucosa
- Virus isolation: growth in equine cell cultures

EHV4:

- Clinical signs
- Virus isolation: growth only on equine origin cell lines
- ELISA: can distinguish EHV4 from EHV1
- Paired serology: comparison of acute and convalescent serum samples for a rise in antibody titers.

EHV1/9 recombinant:

- PCR: analysis of collected tissue samples
- Western Blot: detection of viral proteins in neurologic tissue

Material required for laboratory analysis:

EHV1: Serum for ELISA testing, fetal tissue (lung, thymus, spleen) for histologic diagnosis, nasal swabs or blood (EDTA) for virus isolation or PCR analysis.

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EHV3: serum, scrapings of mucosa from affected areas.

EHV4: nasal swabs, whole blood, serum for various testing modalities.

EHV1/9: recombinant: nasal swabs, serum, CNS tissue

Relevant diagnostic laboratories: Real-time PCR analysis offered by:

http://www.aphis.usda.gov/vs/nahss/equine/ehv/ehv_ehm_recommendations_051611.pdf

http://www.vetmed.ucdavis.edu/ceh/ehv1_diagnostic.cfm

Treatment:

EHV1 encephalomyelopathy: Strict isolation, supportive care for encephalomyelitis. Urinary bladder decompression and rectal evacuation for incontinent patients and sling support if recumbent. Corticosteroids given IV once to twice daily for 3-5 days, followed by a tapered regimen, to decrease CNS inflammation. Treatment with antiviral medications has not been investigated, although good *in vitro* efficacy has been demonstrated.

EHV3: Antiseptic lotions and ointments to prevent secondary infection or discomfort. Discontinue breeding until all lesions are healed.

EHV1/4: respiratory disease: Supportive care.

EHV1/9 recombinant: Supportive care.

Prevention and control:

EHV1 abortion: Inactivated vaccines have been used to prevent abortion, with dosing at 5, 7, and 9 months of pregnancy. Literature does not currently indicate a protective effect of vaccination, but vaccines are successful at producing a high antibody response and limit nasal shedding. In cases of outbreaks, prophylactic vaccination of all horses is controversial. Isolation of pregnant mares and maintenance of closed groups is recommended to prevent further outbreaks in cases of infection. Any horse with respiratory signs also should be isolated. Horses will become infected life-long, with possible recrudescence during times of stress.

EHV1 myeloencephalopathy: quarantine exposed horses. No vaccine has been shown to be protective; however, it is recommended to vaccinate with inactivated vaccines to increase antibody titers and decrease shedding. Concerns have been noted that horses that have been vaccinated frequently are more likely to develop myeloencephalopathy.

EHV3: No vaccines are available. Isolation of affected horses. Horses will become infected life-long, with possible recrudescence during times of stress.

EHV1/4 respiratory disease: Immunity after natural infection is short lived. Modified-live vaccines available for pneumonia, inactivated vaccines are also capable of inducing a high antibody response. Vaccine will decrease severity/incidence but still not prevent the disease. Horses <5 years old should have the first vaccination at 3-4 months of age, with boosters every 6 months, or as determined by the product. Horses will become infected life-long, with possible recrudescence during times of stress.

EHV1/9 recombinant: No vaccine available, maintain separation of potential host species from aberrant hosts that have demonstrated susceptibility.

Suggested disinfectant for housing facilities: Being an enveloped virus, EHV is susceptible to most disinfectants and detergents.

Notification: No special notification requirements for any viral strain.

Measures required under the Animal Disease Surveillance Plan: None currently for any viral strain.

Measures required for introducing animals to infected animal: It is recommended that an isolation period of 21-28 days be placed on any animal that has tested positive or exhibited clinical signs of any form of the disease. Affected animals will remain latently infected following the quarantine period, and may continue to shed virus during times of stress.

Conditions for restoring disease-free status after an outbreak: EHV infected animals will remain latently infected for the duration of their lives. They should remain isolated from healthy individuals.

EQUINE HERPESVIRUS

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FELINE RHINOTRACHEITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Felidae	Droplets, fomites	Ocular nasal discharge, anorexia, depression	High morbidity, low mortality	Famciclovir, lysine, supportive care	Vaccination	No

Fact Sheet compiled by: Ray Wack; updated by Christine Molter

Sheet completed on: 1 March 2011; updated 3 November 2012; updated 2 January 2018

Fact Sheet Reviewed by: James Evermann, Lynelle Johnson, Ray Wack

Susceptible animal groups: Felidae

Causative organism: Feline herpesvirus type-1

Zoonotic potential: None

Distribution: Worldwide

Incubation period: 2 – 6 days (recrudescence ~ 7 days after stressful event)

Clinical signs: Fever, sneezing, keratoconjunctivitis, ulcerative keratitis, salivation, facial dermatitis, Initially serous then mucopurulent ocular and nasal discharge, anorexia, and depression are observed typically. Chronic cases may develop ulcerative keratitis. Disease generally has high morbidity and low mortality except in kittens, immunocompromised, or geriatric cats. Co-infection with other respiratory viruses (especially calicivirus) and secondary bacterial infections are common. In cheetah, proliferative skin lesions at mucocutaneous interfaces have been observed. Clinical signs may persist up to 6 weeks.

Post-mortem, gross, or histologic findings: Erythematous swollen nasal mucus membranes and conjunctiva, hyperemic larynx and trachea, serous or purulent discharge in nares or eyes, early in the disease acidophilic intranuclear inclusions may be seen in affected epithelial cells.

Diagnosis: Clinical signs tend to be more upper respiratory and ocular than with calicivirus infections, but generally are challenging to differentiate. PCR and viral isolation performed on oronasal swabs can indicate presence of the infectious organism, but do not confirm FHV-1 as the causative agent of disease. PCR performed on facial dermatitis lesions and some ocular lesions (including corneal sequestra) is highly correlated with FHV-1 as the causative agent of disease. Cytology identification of acidophilic intranuclear inclusions affected epithelium is diagnostic. In latently infected cats, PCR and virus isolation is usually negative due to sequestered viral DNA in neurons.

Material required for laboratory analysis: Oronasal swabs, conjunctival scraping, respiratory epithelium

Relevant diagnostic laboratories:

Washington Animal Disease Diagnostic Lab
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 Pullman WA 99164-7034
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 waddl@vetmed.wsu.edu
<http://waddl.vetmed.wsu.edu/>

Animal Health Diagnostic Center,
 College of Veterinary Medicine, Cornell University
 PO Box 5786
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 Ithaca, NY 14852-5786
 Phone : 607-253-3900
 Fax: 607-253-3943
<https://ahdc.vet.cornell.edu/>

FELINE RHINOTRACHEITIS

Treatment: General supportive treatment, including broad spectrum antibiotics for secondary bacterial infections; famciclovir can inhibit viral replication and lessen clinical signs. Lysine supplementation may be used. Oxygen or nebulization can be required in severe cases with respiratory distress or hard secretions. Nutritional support and fluid therapy are often required due to anorexia. Proliferative skin lesions may require wide excision or cryosurgery in cheetahs.

Prevention and control: Inactive and modified live vaccines, either injectable or intranasal formulations, usually in combination with other felid viruses are available. Vaccination does not prevent infection or shedding, but can reduce severity of signs and decrease the amount of shedding. Generally, only inactivated vaccines are used in non-domestic felids. Primary vaccination consists of 1 ml of vaccine (Fel-O-Vax, Boehringer Ingelheim) given every 2 – 3 weeks from 6 weeks through 18 weeks of age or a minimum of 3 vaccines in an unvaccinated adult cat. Response to vaccination should be documented with serum neutralization (SN) titer 2 – 3 weeks after the last vaccine. A SN titer of > or equal to 1:16 is considered protective. Antibody titers frequently decline rapidly in exotics and may not accurately reflect susceptibility. Cellular and mucosal immunity are important in moderating or preventing disease. Triennial booster vaccinations are recommended, but more frequent vaccination may be required if there is high exposure risk, due to the rapid antibody decline in some species.

In cheetahs, predictors of FHV infection included a dam receiving a pre-parturition modified live vaccine, being from a small litter, being born to a primiparous dam, and male sex.

Suggested disinfectant for housing facilities: Virus susceptible to most disinfectants, including dilute household bleach, quaternary ammonium disinfectants, peroxygen disinfectants.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Felids should be vaccinated, with response to vaccine documented prior to exposure to known positive cats.

Conditions for restoring disease-free status after an outbreak: Virus is shed intermittently potentially for remaining life of infected animal, but does not survive long in dry environments.

Experts who may be consulted:

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Sea turtles, especially green sea turtles.	Unknown; viral etiology, water-bourne, direct contact and <i>Ozo-branchus</i> leech are suspected. Horizontal transmission experimentally proven	Masses on the skin and viscera	Depends on location of nodules and immune function. Morbidity can reach 92%. Mortalities can reach 88%.	Supportive care; surgical debridement/debulking; euthanasia.	None; in captivity, quarantine affected individuals.	No

Fact Sheet compiled by: Catherine Hadfield

Sheet completed on: 8 April 2011; updated: 5 July 2013

Fact Sheet Reviewed by: Leigh Ann Clayton, Lawrence Herbst, Craig Harms

Susceptible animal groups: Predominantly green turtles (*Chelonia mydas*) are affected. However, Kemp's and olive Ridley (*Lepidochelys kempii* and *L. olivacea*) and loggerhead sea turtles (*Caretta caretta*) also may be. Rarely hawksbill turtles (*Eretmochelys imbricata*) have been affected. The problem has not been reported in leatherback sea turtles (*Dermochelys coriacea*).

Causative organism: An alphaherpesvirus (chelonid fibropapilloma-associated herpesvirus, CFPHV) consistently is detected and lesions can be transmitted using cell-free tumor extracts, but the virus has not been isolated in culture. Virus types appear to cluster based on geographic origin, rather than host species: western Atlantic (Florida, Barbados, recently reported from Texas); Atlantic (Puerto Rico, recently reported from Gulf of Guinea); midwest-Pacific (Hawaii, Australia, Indonesia); and eastern Pacific (Costa Rica, California). It is possible that other viruses such as tornovirus, retroviruses, and reoviruses are involved. Changes in the environment, co-infections, or ecological factors affecting disease expression, or virus transmission, are likely causes for the recent emergence of FP epizootics at multiple locations around the world.

Zoonotic potential: None.

Distribution: Worldwide, but primarily circumtropical. Prevalence of disease varies with location (0 – 92%). It may be associated with eutrophic coastal ecosystems with high human population densities and agricultural run-off. The issue was first documented in the 1930s. Reported increase in prevalence in the late 1950's, especially in specific areas such as the Florida Keys and Indian River Lagoon, Florida, and Hawaii. Prevalence seems to be decreasing in Hawaii while increasing in other regions.

Incubation period: Clinically apparent FP developed 15 – 43 weeks after experimental inoculation. Initiation of tumor growth was positively correlated with water temperature. Inoculated turtles developed antibodies to CFPHV in < 1 year if they developed tumors. Turtles that did not develop tumors, did not seroconvert.

Clinical signs: White/grey/black nodules, 0.1 to >30 cm diameter, focal or multifocal, often involving the head, neck, and limbs, develop as fibropapillomas. Internal nodules (fibromas) are less common. Many fibropapillomatous lesions will resolve spontaneously. Number and severity may increase with curved carapace length (CCL) then decrease as CCL increases further. When tumors are numerous or large in size, they may impinge on function of affected structures which leads to progressive debilitation and death. Larger or ulcerated masses often have secondary infections.

Postmortem, gross, or histologic findings: Fibropapillomas are raised, sessile or polypoid masses with

FIBROPAPILLOMATOSIS

verrucous or smooth surfaces. Internal tumors can be found on the heart, lungs, liver, gall bladder, kidneys, skeletal muscle, and gastrointestinal tract, and are generally described as fibromas, myxofibromas, and fibrosarcomas of low-grade malignancy. Common histologic descriptions include vacuolation of the cytoplasm, balloon degeneration of epidermal cells, and benign papillary epidermal hyperplasia (especially in the stratum spinosum) occurring on thick stalks of proliferating fibrovascular stroma characterized by disorganized collagen fibers. Perivascular mononuclear cell inflammation is often observed in the deeper layers of the dermis.

Diagnosis: Clinical diagnosis is usually based on presence of skin or oral masses consistent in appearance with fibropapillomas. Endoscopy, laparoscopy, radiography, ultrasonography, MRI, and coeliotomy can be useful diagnostic modalities for identifying visceral tumors.

Definitive diagnosis requires compatible histopathology findings. Further support of a diagnosis occurs if intralosomal DNA of CFPHV are detected by polymerase chain reaction (PCR) from tissue obtained from tumors. *In situ* hybridization (ISH) can be used to detect CFPHV in nuclei of infected epithelial cells.

Material required for laboratory analysis Formalin-fixed tissue for histology and frozen tissue for PCR.

Relevant diagnostic laboratories: All histopathology laboratories can assess tissues for compatible lesions. Herpesviral PCR and sequencing is available at the University of Florida and Hubbs-SeaWorld Research Institute.

Treatment: Supportive care (appropriate temperature, good water quality, low stocking density, fluid therapy, adequate nutrition, and, as needed, systemic antibiotics). The lesions may be excised surgically; laser surgery followed by second intention healing is usually recommended. Controlled studies on improved outcomes are lacking. Acyclovir has been found anecdotally to be useful. Tumors on and around the eyes are most important to remove. Turtles with internal tumors may require euthanasia.

Prevention and control: Currently no prevention and control measures for wild populations are available. In captivity, fibropapillomatous turtles should be quarantined from unaffected turtles, including by a separate water system. Some turtle rehabilitation centers will not accept turtles affected by fibropapillomas.

Suggested disinfectant for housing facilities: Standard disinfectants effective for herpesviruses should be effective against CFPHV.

Notification: None.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Unaffected individuals should not be introduced to affected turtles.

Conditions for restoring disease-free status after an outbreak: None known. It is probable that affected turtles are CFPHV carriers for life. Experienced marine turtle rehabilitation facilities consider release of animals if they remain tumor-free for one year after surgical removal, although sufficient long-term housing is rarely available in large stranding events to accommodate this approach.

Experts who may be consulted:

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 Institute for Animal Studies
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UF Diagnostic Lab

Tissue PCR for herpesvirus – notify laboratory before shipping for submission forms and parameters; use ice packs or dry ice via FedEx, UPS, or DHL

Costs (July 2013): \$100.00/test/sample. Turnaround time is 2-3 weeks.

<http://labs.vetmed.ufl.edu/sample-requirements/microbiology-parasitology-serology/zoo-med-infections/>

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HERPESVIRUS B

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Macaques; other non-human primates; humans	Bites, scratches, fomites, saliva or bodily fluids, mucosal absorption possible, laboratory transmission (i.e., needle puncture)	Macaques: generally intermittent mucosal pustules. Non macaque species: Sometimes are herpes-like mucosal lesions are associated with the point of infection. Most cases present with fever, and encephalitis manifesting neurologic signs, including: dysphagia, ataxia, confusion, paresthesia and paralysis	Non macaque species: Without treatment, it is very fatal (70 - 80%). Some patients continue to have neurologic sequelae after recovery	Anti-virals, such as: ganciclovir, valacyclovir, and famciclovir. Many patients opt to remain on drugs for years after initial infection	Personal protective equipment, including gowns or coveralls, gloves, goggles and a mask or respirator.	Yes

Fact Sheet compiled by: Melinda Rostal

Sheet updated on: 3 May 2011; updated 30 October 2012

Fact Sheet Reviewed by: Richard Eberle; Jan Ramer

Susceptible animal groups: The disease occurs naturally in all macaques. Humans and other nonhuman primates are susceptible.

Causative organism: Macacine herpesvirus 1, also called herpes B virus or B virus

Zoonotic potential: Yes

Distribution: All reported cases in humans have been people that work with captive macaques or have been exposed in the field. Macaques are usually asymptomatic and may have been seronegative at the last screening or even at the time of the exposure prior to seroconversion. One report of human infection from Vietnam has been documented otherwise no cases from individuals has been reported in Asia in areas that macaques inhabit.

Incubation period: < 2 days to 2-3 weeks, usually 5-8 days. Two cases were reported where the patient had no previous exposure to macaques or exposure had occurred over 10 years previously.

Clinical signs:

Macaques: 80-100% of sexually mature macaques, especially if they have contact with other macaques, are seropositive for B virus. They rarely have clinical signs indicating infection or recrudescence; however, they will occasionally have herpes-like skin mucosal pustules. Research on shedding frequency is inconclusive, although it increases during periods of stress, including the breeding season.

Humans: Sometimes there are herpes-like skin/mucosal lesions associated with the point of infection accompanied by pain and itching and erythema. Most cases present with fever, and encephalitis manifesting in neurologic signs including: dysphagia, ataxia, confusion, paresthesia and paralysis. Latency is established

HERPESVIRUS B

and reactivation can occur.

Post mortem, gross, or histologic findings:

Macaques: Most often, histological evidence of acute infection is present without gross pathology. The virus often remains latent in the trigeminal or sacral nerve ganglia, from which culture or PCR may be used to detect the virus. If oral or genital lesions are present there may be vesicle formation with leukocytic invasion of the area; when keratinized cells overlying the vesicle slough, a plaque of necrotic fibrinous material remains overlying the base of the ulcer. Intranuclear inclusions can be seen in tissues showing recent signs of degeneration.

Humans: Few reports of histological findings have been documented. Inclusion bodies are not typically found. Reported findings include severe inflammatory and degenerative changes in the spinal cord, particularly in the cervical cord and brainstem; the thalamus and hypothalamus may also be infected.

Diagnosis: B virus antibody ELISA or B virus recombinant ELISA assays are used. A negative antibody titer does not indicate the animal is not infected, only that it is not currently producing antibodies. Rising titers may be associated with viral shedding period. Diagnosis is often based on Western blot or virus neutralizing antibodies as well as virus isolation. PCR has been developed as well.

Material required for laboratory analysis:

For serology: 0.5-2.0 ml serum in plastic tube. Store and ship at -20°C or with dry ice.

For virology: Swabs of vesicle or other lesions. Place swab in 1-2 ml viral transport media, store at -80°C. CSF, autopsy samples of brainstem, biopsies from the site of inoculation: place in plastic storage/shipment tubes. Ship samples on dry ice according to US Department of Transportation regulations.

Relevant diagnostic laboratories: B -virus is a BSL 4 agent.

B Virus Research and Resource Laboratory
Dr. Julia Hilliard
Georgia State University, Viral Immunology Center
161 Jesse Hill Jr Dr
Atlanta, GA 30302-4118
jhilliard@gsu.edu
For emergency: (404) 358-8168

Enteric, Respiratory, and Neurological Virus Laboratory
Dr. David Brown
Central Public Health Laboratory
61 ColindaleA ve. London NW9 5HT, England
dbrown@phls.org.uk

Virus Reference Laboratory (non-human primates only)
VRL – San Antonio
7540 Louis Pasteur Dr., Ste. 200
San Antonio, Texas 78229
(210) 614-7350
Anthony.Cooke@vrl.net

Treatment: Non macaques: Anti-virals are used to attempt to control or prevent encephalitis. Recommended medications include: ganciclovir, valacyclovir, and famciclovir. Many patients must remain on anti-virals for years after exposure. FEAU (2'-fluoro-5-ethyl-Ara-U) is a new anti-viral that appears effective in cell culture, but has not been used in a human case.

Prevention and control: All macaques need to be treated as B virus-infected. Due to the severity of

HERPESVIRUS B

infection with B virus in humans the US Centers for Disease Control and Prevention (CDC) developed guidelines to prevent B virus in workers handling macaques (<http://www.cdc.gov/mmwr/preview/mmwrhtml/00015936.htm>.) The most recently updated version of these recommendations was given in 2002 by the B Virus Working Group. The recommendations are briefly outlined below.

- Personnel must wear appropriate personal protective equipment including glasses and faceshields, masks, long sleeve protecting clothing, and nitrile or latex gloves. If the animal is not sedated, leather gloves extending to the shoulder should be used.
- Personnel must be trained in the associated risks of infection and appropriate response protocols.
- Upon possible exposure, the person should immediately wash the wound or lavage the mucous membrane exposed for 15 minutes.
- Collect baseline serum and culture samples from the person and the macaque.
- Starting prophylaxis with an anti-viral within 24 hours is recommended if the case meets one of the following criteria:
 - Exposure of mucosa or injured skin to an ill or immunocompromised or shedding macaque.
 - Exposure of mucosa or injured skin that is not adequately cleaned.
 - Laceration is of the head, neck or torso.
 - Deep puncture bite or a needle puncture associated with macaque CSF fluid, herpes-like lesions, eyelids or mucosa.
 - A post-cleaning culture of wound is positive for B virus.
 - A laceration is caused by an object contaminated by macaque mucosal, genital or saliva secretions.
- Prophylaxis with an anti-viral should be considered if the case meets one of the following criteria:
 - Exposure of mucosa or injured skin that has been adequately cleaned.
 - A needles puncture was associated with blood from an ill or immunocompromised macaque.
 - Skin that was recently exposed to contaminated macaque body fluid or cell culture has been lacerated.

Suggested disinfectant for housing facilities: Macaque housing should be cleaned with hot water and detergent by staff utilizing appropriate PPE such as masks and face protection while cleaning. Currently efforts are underway to create specific pathogen free (SPF) colonies of macaques and some groups has successfully maintained populations of macaques that are 99.3% free of B virus for longer than 7 years. This process involves initially keeping the macaques isolated and culling seropositive animals. No current recommendations for disinfecting housing facilities are available since in seropositive populations the virus tends to be ubiquitous and in SPF populations positive animals should be culled. All macaques should be treated as if positive for B virus. Research is also underway to create a B virus vaccine for macaques.

Notification: B virus has been a CDC Select Agent; however, as of 4 December 2012, it is no longer a select agent. B virus infections are not reportable on a national level, although states may vary in their reporting requirements.

Measures required under the Animal Disease Surveillance Plan: This virus is not listed under Annex A or B.

Measures required for introducing animals to an infected animal(s): If a seronegative animal is introduced to a seropositive animal it should be expected that the animal will seroconvert eventually, especially after the onset of sexual maturity. SPF colonies must cull seropositive animals and closely monitor cagemates to prevent the virus from becoming established in the colony.

Conditions for restoring disease-free status after an outbreak: No specific standards exist at this time; however, it is recommended to test the animals for antibodies one month apart and again following a time

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period of greater than 6 months, but less than 12 months.

Experts who may be consulted:

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References:

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HERPESVIRUS HOMINIS TYPES 1 AND 2

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Predominantly affects humans but can cause disease in NHP: Aotidae, Callitrichidae, Cebidae, Hylobatidae, Lemuridae, Pongidae, Scandentia.	Direct contact or airborne	Mostly asymptomatic, but can result in recurrent rhinitis, labial herpes, lingual plaques/ulcers, rhinitis, nasal discharge, conjunctivitis, salivation, ataxia, ulcerative dermatitis, death	Fatal disease in Cebidae, Callitrichidae, and tree shrews. Rarely fatal in Pongidae	Acyclovir, valacyclovir	Avoid contact with humans with active herpes lesions	Zoonothroponotic disease

Fact Sheet compiled by: Sam Rivera; updated by Jan Ramer

Sheet completed on: 1 June 2011; updated 23 July 2013

Fact Sheet Reviewed by: Sam Rivera; Kevin Brunner

Susceptible animal groups: Aotidae, Callitrichidae, Cebidae, Hylobatidae, Lemuridae, Pongidae, Scandentia.

Causative organism: Herpesvirus hominis types 1 and 2.

Zoonotic potential: Zoonothroponotic disease

Distribution: Worldwide

Incubation period: In NHP: 2-14 days after experimental infection.

Clinical signs: In great apes, oral and pharyngeal vesicles and ulcers, vesicles on the lips and nose, conjunctival lesions, pustules, vesicles and/or ulcerated lesions of the genitalia, listlessness, anorexia. Infant great apes: diarrhea, vomiting, dyspnea, vesicles, death. Other NHP: conjunctival, oral and cutaneous lesions (vesicles to ulcers), rhinitis, keratitis, weakness, depression, anorexia, excessive salivation, nasal discharge, myoclonus, ataxia, seizures. Peracute death has been seen in callitrichids.

Post mortem, gross, or histologic findings: Multifocal vesicular and necrotizing dermatitis (face, arms, chest, legs), gingivitis and stomatitis, hepatomegaly with mottling, congestion or necrotic foci, splenomegaly with congestion, pulmonary edema, lymphadenopathy, ocular lesions (conjunctivitis, blepharitis, ulcers), adrenal enlargement, necrosis or hemorrhage, CNS lesions (edema, hemorrhage, focal softening and necrosis, congested meninges), multifocal meningoencephalitis (in gibbons) with multifocal nonsuppurative perivascular cuffing, necrosis, and gliosis and Cowdry type A intranuclear inclusions in spleen and liver.

Diagnosis: Serology, virus isolation, PCR, in-situ hybridization, histopatology.

Material required for laboratory analysis: Material from vesicles or other lesion, serum or whole blood.

Relevant diagnostic laboratories:

Pathogen Detection Laboratory

California National Primate Research Center, Road 98 & Hutchison

University of California

Davis, California 95616

HERPESVIRUS HOMINIS TYPES 1 AND 2

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BioReliance
Serology/PCR Laboratories
14920 Broschart Rd.
Rockville, Maryland 20850
(301) 610-2227
(310) 610-2587
ahs@bioreliance.com

Virus Reference Laboratories, Inc.
7540 Louis Pasteur Road
San Antonio, Texas 78229
(877) 615-7275
Fax: (210) 615-7771

Zoologix Inc.
9811 Owensmouth Avenue, Suite 4
Chatsworth, California 91311-3800
818-717-8880
818-717-8881
info@zoologix.com.

Treatment: Acyclovir, valacyclovir.

Prevention and control: Avoid contact with humans with active herpes lesions. In owl monkeys, a modified live vaccine has been shown to be protective.

Suggested disinfectant for housing facilities: Lipid solvents, soap, UV-light, heat.

Notification: None at this time.

Measures required under the Animal Disease Surveillance Plan: None at this time.

Measures required for introducing animals to infected animal: Keep susceptible species away from known positive NHP.

Conditions for restoring disease-free status after an outbreak: Many NHP antibody-positive, latent infections possible, so exposure and disease free status is difficult.

Experts who may be consulted:

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American Association of Zoo Veterinarians Infectious Disease Manual
**SIMIAN VARICELLA VIRUS (*CEROPITHECINE HERPESVIRUS 6, 7, 9*)
AND VARICELLA-ZOSTER VIRUS (*HUMAN HERPESVIRUS 3*)**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Varicella-zoster virus (VZV) affects man and Pongidea Simian Varicella Virus (SVV) affects African and Asian NHP	Direct contact, inhalation of aerosolized virus	VZV: chickenpox or zoster in NHP SVV: generalized vesicular eruptions, hyperthermia	Rarely a fatal disease. Severity depends on age and immune-competency.	Acyclovir, Vidarabine mono-phosphate, Foscarnet, and other anti-virals.	Avoid contact between humans with chickenpox/ Zoster and non-human primates. Isolate affected NHP	Not reported as a zoonotic disease

Fact Sheet compiled by: Sam Rivera; updated by Jan Ramer

Sheet completed on: 1 June 2011; updated 23 July 2013

Fact Sheet Reviewed by: Sam Rivera; Kevin Brunner

Susceptible animal groups: VZV: Pongidae; SVV: Old world NHP, apes, and humans.

Causative organism: Simian varicella virus (Ceropithecine herpesvirus 6, 7 and 9), Varicella-zoster virus (Human herpesvirus 3).

Zoonotic potential: Not reported

Distribution: Simian Varicella Viruses occur naturally in Africa and Asia and are found worldwide in captive populations. Human Varicella-Zoster Virus is found worldwide.

Incubation period: VZV: 7-14 days; SVV: 10-15 days

Clinical signs:

VZV: In Pongidae, pruritic, pustulovesicular rash, conjunctivitis, fever, anorexia, lethargy, lymphadenopathy, coughing, and sneezing can be observed. Zoster with severe axillary and thoracic cutaneous ulceration was reported in an elderly gorilla with concurrent immunosuppressive disease.

SVV: generalized vesicular rash, mild fever, anorexia, lethargy, vesiculoulcerative dermatitis is observed. Although rarely a fatal disease, high fatality (within 48 hours) has been reported in natural outbreaks.

Post mortem, gross, or histologic findings: VZV in Pongidae occasionally chickenpox-like disease or oral/perioral vesicles. SVV: vesiculoulcerative lesions of the skin, and oral mucous membranes, hemorrhages and necrosis in internal organs.

Diagnosis: Serology, virus isolation, PCR, EM

Material required for laboratory analysis: Vesicular material, whole blood or serum.

Relevant diagnostic laboratories:

Pathogen Detection Laboratory
California National Primate Research Center, Road 98 & Hutchison
University of California
Davis, California 95616
(530) 752-8242
Fax: (530) 752-4816
PDL@primate.ucdavis.edu

American Association of Zoo Veterinarians Infectious Disease Manual
**SIMIAN VARICELLA VIRUS (*CEROPITHECINE HERPESVIRUS 6, 7, 9*)
AND VARICELLA-ZOSTER VIRUS (*HUMAN HERPESVIRUS 3*)**

<http://pdl.primate.ucdavis.edu>

BioReliance
Serology/PCR Laboratories
14920 Broschart Rd.
Rockville, Maryland 20850
(301) 610-2227
(310) 610-2587
ahs@bioreliance.com

Virus Reference Laboratories, Inc.
7540 Louis Pasteur Road
San Antonio, Texas 78229
(877) 615-7275
Fax: (210) 615-7771

Zoologix Inc.
9811 Owensmouth Avenue, Suite 4
Chatsworth, California 91311-3800
818-717-8880
818-717-8881
info@zoologix.com

Treatment: Varicella-Zoster immunoglobulins, acyclovir, famcyclovir, valacyclovir, trifluridine, vidarabine monophosphate, foscarnet.

Prevention and control: Avoid contact between humans with chickenpox or zoster and NHP.

Suggested disinfectant for housing facilities: Organic solvents, detergents, proteases, UV-light, heat.

Notification: None at this time.

Measures required under the Animal Disease Surveillance Plan: None at this time.

Measures required for introducing animals to infected animal: Self-limiting disease, animals can be introduced once skin lesions heal.

Conditions for restoring disease-free status after an outbreak: Self-limiting disease

Experts who may be consulted:

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References:

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Zoster/Chickenpox_Varicella/Chickenpox_Varicella.htm#Control Accessed 6 August 2013.

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INFECTIOUS BOVINE RHINOTRACHEITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ruminants – cattle, goats, sheep, swine, red deer, American and Malaysian buffalo, and Brazilian tapirs.	Direct contact or aerosolization of viral particles from an infected animal. It also is transmitted sexually and via artificial insemination. Infection may become latent and can reoccur with stress, resulting in viral shedding.	Decreased milk production. Upper respiratory disease; conjunctivitis, corneal disease, and panophthalmitis; and reproductive, neurologic, and gastro-intestinal signs. Encephalitis in calves.	Mild to severe, depending on secondary bacterial invasion.	No treatment for the virus itself but supportive care should be provided. Antibiotics in the feed and water are used to treat the secondary bacterial infections.	Vaccination; isolation or removal of affected individuals and young until fully vaccinated; decrease stress. Use of a “marker” vaccine is helpful for screening. IBR is eradicated in 6 countries & is possible in others.	No.

Fact Sheet compiled by: Christie Hicks

Sheet completed on: updated 5 February 2018.

Fact Sheet Reviewed by: AJ Marlar

Susceptible animal groups: Ruminants especially cattle, goats, swine, red deer, Malaysian buffalo, and Brazilian tapirs. Young animals at weaning age and those in crowded conditions are especially susceptible. However, the disease does occur in adult animals, especially non-vaccinated pregnant ruminants.

Causative organism: Bovine Herpesvirus 1 (an alphaherpesvirus)

Zoonotic potential: None.

Distribution: Worldwide distribution is considered present. Eradication is being attempted in several western European countries.

Incubation period: 2 to 6 days. Outbreaks reach a maximum intensity by the 2nd to 3rd week with mostly all recovered by the 4th to 6th week. Uncomplicated BHV-1 (IBR) can resolve in 4 to 5 days if no secondary infections are present.

Clinical signs: An early sign is a decrease in milk production. Respiratory signs include coughing, serous to mucopurulent nasal discharge. Ophthalmic signs include conjunctivitis with an ocular discharge and corneal opacity, or panophthalmitis. “Red Nose” (muzzle hyperemia), respiratory distress due to discharges, salivation, anorexia, and pyrexia may also be seen. Secondary infections are possible and can lead to a bronchopneumonia. Neonates may present with generalized infection similar to a septicemia, enteritis, and/or encephalitis. IBR has been associated with a high mortality rate in calves (< 1 month of age) with no preceding signs. Mid- to late-term abortions can occur up to 100 days post exposure with infection of the dam and genital tract infections occurring. Infertility and birth defects have also been seen. Subclinical infections can occur.

Post mortem, gross, or histologic findings: Within the upper respiratory tract and trachea, petechial to ecchymotic hemorrhages are observed in the mucous membranes of the nasal cavity and paranasal sinuses. Focal areas of necrosis are present in the nose, pharynx, larynx, and trachea which may join together to form plaques. The sinuses can be filled with a serous to a serofibrinous exudate that may extend into the pharynx. The pharyngeal and pulmonary lymph nodes may become swollen and hemorrhagic. If the tracheitis extends into the bronchi and bronchioles, the epithelium can be sloughed into the airways. Nasal lesions consist of clusters of gray necrotic foci on the mucous membranes of the septal mucosa and/or with pseudo-diphtheritic

INFECTIOUS BOVINE RHINOTRACHEITIS

yellow plaques. Aborted fetuses have multifocal non-raised white lesions throughout the liver. Placentitis is occasionally seen.

Diagnosis: For acute cases, PCR may be performed. A serum neutralization test can be used with paired serum antibody titers at least 2 weeks apart, except in abortion cases as the titer is already at its highest level. ELISA for an antibody titer with a concurrent rise is available but also indirect hemagglutination and complement fixation serology as well. Virus isolation via nasal fluids at the early onset of disease is possible. Gross lesions at necropsy and histopathology. Immunoperoxidase, virus isolation, and fluorescent antibody staining on fetal tissues can also be performed in abortion cases. Patients with corneal lesions would require ruling out malignant catarrhal fever, infectious bovine keratoconjunctivitis (*Moraxella bovis*), and squamous cell carcinoma.

Material required for laboratory analysis: Nasal fluids, serum, plasma, milk, placenta, and/or tissues, for example the liver.

Relevant diagnostic laboratories: Any state laboratory can perform the testing.

Treatment: While no treatment for the virus itself exists, one may treat for secondary bacterial infections with antibiotics and supportive care. Most cases recover in 4 to 5 days if secondary infections are not present.

Prevention and control: Vaccination with a modified live vaccine (MLV) given parenterally (SC or IM) or IN is possible. MLV given IM during pregnancy may cause abortions, especially in the third to eighth months of gestation, therefore a MLV is best administered twice before breeding with the second administration occurring 30 days prior to prebreeding and the next vaccination within 12 months afterwards. The MLV is also not safe for nursing calves unless the dam has been vaccinated within the last year and at prebreeding with the same vaccine. Vaccinating with an inactivated multivalent vaccine given SC or IM will protect against abortions if given prior to breeding. The use of the IN vaccine may be helpful for: a local rapid immune response; in those that are already pregnant; and may prevent new cases in an outbreak as long as the individuals vaccinated are not showing any clinical symptoms at the time of IN vaccination. For control: it is important to vaccinate at 6 to 8 months of age or 2 to 3 weeks before weaning, before introduction into the herd, prior to breeding, and annually thereafter. Quarantine all new individuals 4 weeks after arriving, with testing for IBR before arrival and before entrance into the herd. Eradication can be attempted by screening all individuals that are at least 1 year of age and removing any seropositive reactors, this process should continue annually. The best way to differentiate between the natural virus versus a vaccine titer is to use ELISA to test for Glycoprotein E (gE). gE is present in natural infections, however, newer “marker” vaccines have deleted gE from their make-up. Caution must be used as conventional vaccines still contain gE and can cause a false seropositive result. Currently, IBR is eradicated in Austria, Denmark, Finland, Sweden, Switzerland, and Norway.

Suggested disinfectant for housing facilities: As an enveloped virus, it can be managed by lipid solvents, bleach, and hydrogen peroxides. This virus is also inactivated by UV light and heat.

Notification: No.

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: A period of 2-3 weeks after the illness starts should be waited before introducing any new individuals into the herd. Viral particles can still be seen in nasal secretions throughout this time. All new arrivals into the herd must be vaccinated prior to entry and then revaccinated in 3 months and again at 6 months.

Conditions for restoring disease-free status after an outbreak: Serologic testing to detect any seropositive individuals. Removal of these individuals and/or creating 2 separate herds. The animals that fully recover from this disease will have long-term immunity to future outbreaks.

Experts who may be consulted: Any state veterinarian.

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INFECTIOUS BOVINE RHINOTRACHEITIS

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KOI HERPESVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Fishes, specifically members of the genus <i>Cyprinus</i> .	Horizontal and via fomites and possibly arthropod vectors.	Signs may be non-specific but can include, enophthalmia, branchitis, hemorrhagic gills, branchial necrosis, areas of skin depigmentation, and blisters.	Mortality may reach 100% and frequently is over 80%.	None.	Strict biosecurity and quarantine protocols should be followed based on information available through the OIE and USDA.	No.

Fact Sheet compiled by: Gregory A. Lewbart

Sheet completed on: 4 September 2013

Fact Sheet Reviewed by: Denise Petty; Thomas Waltzek; Ruth Francis-Floyd

Susceptible animal groups: Carp and koi (*Cyprinus carpio*) with evidence that goldfish (*Carassius auratus*) and other cyprinids can be non-clinical carriers of the virus.

Causative organism: Cyprinid herpesvirus-3 (Koi Herpesvirus or KHV).

Zoonotic potential: None

Distribution: Global, especially in temperate geographical areas, except for Australia. The disease was first identified in England, 1996.

Incubation period: Incubation period varies depending on water temperature; most cases are detected at 22°-25.5°C. Latent infections can likely persist for months or even years. Arthropods such as the fish louse (*Argulus* sp.) are likely vectors.

Clinical signs: Clinical signs include, but are not limited to, lethargy, enophthalmia, depigmented areas and blisters of the body surface, branchitis, branchial hemorrhage, branchial necrosis, and high mortality.

Post mortem, gross, or histologic findings: At necropsy, affected fish may have generalized – possibly sanguineous – edema, organ hemorrhage, intestinal inflammation, branchial hemorrhage, branchial necrosis, mottled organs, and excessive abdominal adhesions.

Diagnosis: Diagnosis can be made directly with viral isolation from spleen or caudal kidney on a susceptible cell line such as Koi Fin (KF); this technique usually requires sacrificial euthanasia. Non-lethal direct methods utilizing polymerase chain reaction (PCR) that can be performed on blood, gill tissue biopsies, and feces. Non-lethal indirect methods include enzyme-linked immunosorbent assay (ELISA) and virus neutralization (VN) on blood, but currently test is not available.. Positive indirect method samples only indicate that a fish has produced antibodies to the virus and may not, or ever have been, infected with KHV although this can vary by testing specificity.

Material required for laboratory analysis: Live, moribund fish are the best specimens for an accurate diagnosis. Virus isolation from appropriate tissues is superior to PCR and the indirect methods abovementioned.

Relevant diagnostic laboratories: Testing is available at various approved state and federal laboratories. http://www.aphis.usda.gov/animal_health/lab_info_services/downloads/ApprovedLabs_Aquaculture.pdf, including PCR at <http://www.vetdna.com/archive/koiherpes>.

Treatment None are effective or recommended.

KOI HERPESVIRUS

Prevention and control: Facilities holding and importing high risk cyprinid fishes should be diligent in following standard quarantine protocols and adhere to appropriate and periodic testing/screening as prescribed by the OIE and USDA. A modified live vaccine called (Cavoy[®], Novartis Inc.) was approved for use on koi by the FDA in 2012, but the company is no longer distributing the vaccine.

Suggested disinfectant for housing facilities: Disinfection protocol depends on the size, type and nature of the materials and sites to be disinfected. When an active outbreak of KHV is confirmed, the infected stocks should be depopulated and all areas that held the infected fish must be disinfected. The virus can survive in the environment for about 3 days but can be inactivated by sodium hypochlorite (200 ppm for 1 hour), quaternary ammonium (500 ppm for 1 hour), formalin, ozone, organic iodophors, gamma and ultraviolet radiation, pH extremes of < 4.0 or > 10.00, and heating at 60⁰ C for 15 minutes. All equipment and tanks, raceways and ponds should be disinfected. USDA APHIS also recommends that incoming water to the farms be treated with sand filtration and UV.

Notification: All suspect cases should be necropsied and USDA contacted for proper routing of diagnostic samples. Confirmed cases must be reported to the USDA.

Measures required under the Animal Disease Surveillance Plan: Once an infection is reported, a facility has to follow the recommendations described in the International Aquatic Animal Health Code and the Diagnostic Manual for Aquatic Animal Diseases by OIE to be declared free of KHV. In the US, USDA recommendations must be followed.

Measures required for introducing animals to infected animal: Not applicable.

Conditions for restoring disease-free status after an outbreak: See the OIE and USDA web sites for current information. Periodic testing with negative results may be required. A complete summary of the disease and diagnostic procedures may be found on the Office International des Epizooties (OIE) web site.

Experts who may be consulted:

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MACROPOD HERPESVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Macropods, potoroids; possibly other marsupials	Direct contact	<u>MaHV-1</u> : death, pyrexia, respiratory signs, conjunctivitis, anogenital vesicles. <u>MaHV-2</u> : death, conjunctivitis, oral and anogenital lesions. <u>MaHV-3</u> : possibly anogenital ulcerations.	Severe fatal clinical disease reported with MaHV-1 and -2. Mild lesions in immune-compromised individuals with MaHV-3.	None known. Supportive care. Efficacy of antiviral drugs unknown.	Isolate positive individuals from negative individuals.	Not reported; unlikely

Fact Sheet compiled by: Joseph A. Smith

Sheet completed on: 1 February 2011; updated 15 July 2013

Fact Sheet Reviewed by: James Wellehan, Roman Pogradichniy

Susceptible animal groups: This disease generally affects macropods and potoroids. MaHV-1 was first described in parma wallabies (*Macropus parma*). MaHV-2 was first described in grey dorcopsis (*Dorcopsis muelleri luctuosa*) and quokkas (*Setonix brachyurus*). MaHV-3 was first described in eastern grey kangaroos (*M. giganteus*). Non-specified herpesvirus infections have also been reported in tammar wallabies (*M. eugenii*), western grey kangaroos (*M. fuliginosus*), brush-tailed rat kangaroos (*Bettongia penicillata*) and rufous rat kangaroos (*Aepyprymnus rufescens*). MaHV-1 has been experimentally induced in brush-tailed possums (*Trichosurus vulpecula*).

Causative organism: Alphaherpesviruses Macropodid herpesvirus 1 (MaHV-1) and Macropodid herpesvirus 2 (MaHV-2); Gammaherpesvirus Macropodid herpesvirus 3 (MaHV-3)

Zoonotic potential: Not reported; unlikely.

Distribution: Clinical disease has only been reported in captive animals. One study reported seropositive rates of 23% of wild marsupials and 41% of captive marsupials in Australia, although specificity of the serologic testing is unknown. MaHV-1 and MaHV-2 have only been reported in animals in Australia. MaHV-3 has been reported in captive eastern grey kangaroos in the US as well as in wild eastern grey kangaroos.

Incubation period: Not known.

Clinical signs:

MaHV-1: Fatal systemic infections resulting in severe clinical signs including pyrexia, respiratory signs, conjunctivitis, and anogenital vesicles.

MaHV-2: Fatal systemic disease characterized by conjunctivitis and lesions on the oral and anogenital mucous membranes.

MaHV-3: Typically a subclinical systemic disease. Mild to moderate ulcerative cloacitis found in immunocompromised individuals may be associated with this virus.

Post mortem, gross, or histologic findings:

MACROPOD HERPESVIRUS

MaHV-1: Gross-Vesicles and ulcers of skin, lips, eyelids, and anogenital mucosa; rhinitis; mild to severe keratitis. Histologic-necrotic epithelium and inflammatory debris; numerous large basophilic or eosinophilic intranuclear inclusions.

MaHV-2: Gross-purulent conjunctivitis; red edematous lung lesions; pinpoint (1mm) yellow foci in the liver; erythematous mucosal lesions in the gastrointestinal tract; mesenteric lymphadenopathy; erythematous mucous membranes; yellow plaques on reproductive tract. Histologic-disseminated focal necrosis; intranuclear acidophilic or basophilic inclusions.

MaHV-3: Gross-Ulcerative cloacitis was found in MaHV-3 positive individuals. A definitive association with the virus has not been proven. Histologic-No inclusions were identified in PCR-positive tissues.

Diagnosis: Virus isolation, PCR, or serology. Serology is not available in North America.

Material required for laboratory analysis: Swab or tissue sample of affected tissue.

Relevant diagnostic laboratories:

Herpesvirus consensus PCR: University of Florida Zoological Medicine Laboratory.

<http://labs.vetmed.ufl.edu/sample-requirements/microbiology-parasitology-serology/zoo-med-infections/>

Serologic tests are not commercially available in North America.

Treatment: No successful treatment is reported. Clinical signs should be treated with supportive care. The use of antiviral drugs with these viruses has not been reported.

Prevention and control: Specific guidelines have not been reported or investigated. Generally, positive individuals should be kept physically separated from negative individuals to prevent direct transmission. Care should also be taken to prevent indirect transmission through fomites. The role of vectors in the transmission of these viruses is unknown.

Suggested disinfectant for housing facilities: No studies of disinfectant efficacy have been reported to date. However, as enveloped viruses, macropod herpesviruses are presumed to be unstable in the environment and should be susceptible to the most common disinfectant strategies.

Notification: Not a reportable disease.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Animals should be assumed to be infected for life with the possibility of transmitting virus to non-infected animals.

Conditions for restoring disease-free status after an outbreak: Due to life-long infections, removal of positive animals from the group is the only known method for obtaining a disease-free status.

Experts who may be consulted:

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MACROPOD HERPESVIRUS

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MALIGNANT CATARRHAL FEVER (MCF)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p>Many species of <i>Artiodactyla</i>, including bovids, cervids, giraffids, suids</p> <p>Blue and black wildebeest are common maintenance hosts.</p>	<p>Direct contact with infected individuals and bodily fluids (nasal and ocular secretions).</p> <p>Fomites Water Rangeland Feed</p> <p>Aerosol transmission also important for some of the viruses.</p> <p>Transmitted only between carriers and clinically susceptible animals. Affected animals do not transmit MCF to their conspecifics.</p>	<p>Mucous membrane ulceration and inflammation, high fever, oral and nasal exudates, corneal opacities, and lymph-adenopathy. Additional signs may include: neurologic signs, diarrhea, arthritis, and skin lesions.</p>	<p>Typically fatal in susceptible species</p> <p>Up to 25% of cattle may develop chronic disease with a waxing and waning course. Up to 5% may clinically recover, but most eventually die.</p>	<p>None, usually ineffective</p>	<p>Separate clinically susceptible species from known carrier species such as sheep, goats, and wildebeest in known infected areas to prevent direct transmission</p> <p>Prevent fomite transmission</p> <p>Use precautions to prevent aerosol transmission</p>	<p>No</p>

Fact Sheet compiled by: Danelle M. Okeson and Enrique Yarto

Sheet completed on: updated December 2017

Fact Sheet Reviewed by: Gretchen Cole

Susceptible animal groups: Cervidae, Bovidae, Giraffidae, Suidae

Causative organism: Herpesviruses classified in the genus *Macavirus*. At least 10 viruses have been identified that are categorized within the MCF virus group. Some have been associated with MCF in clinically susceptible species.

Alcelaphine herpesvirus 1 (AIHV-1; classic African MCF/wildebeest-associated), carried by wildebeest; susceptible species = Cervidae and Bovidae

Ovine herpesvirus 2 (OvHV-2; sheep-associated), carried by domestic and wild bighorn sheep, considered endemic in domestic sheep; susceptible species = ruminant species and swine. Most MCF cases in domestic cattle and bison in the US are due to OvHV-2. European breeds of cattle (*Bos taurus*), are relatively resistant, but Bali cattle, bison, and some cervid species such as Pere David's deer are highly susceptible.

Caprine herpesvirus 2 (CpHV-2), carried by domestic and exotic goats, considered endemic in domestic goats; clinically susceptible species = white-tailed deer, Sika deer, moose and pudu.

A herpesvirus referred to as "malignant catarrhal fever virus-white tailed deer" (MCFV-WTD), carrier unknown; susceptible species = white-tailed deer.

Ibex-MCFV, carried by Nubian ibex (*Capra nubiana*); prior to a case in a captive bongo (*Tragelaphus euryceros*) the virus was not considered pathogenic.

Alcelaphine herpesvirus 2 (AIHV-2), identified in but non-pathogenic in Jackson's hartebeest; clinically

MALIGNANT CATARRHAL FEVER (MCF)

susceptible species = Barbary red deer (*Cervus elaphus barbarus*)

Other herpesviruses categorized in the same group as the pathogenic MCF viruses have been identified in aoudad, roan antelope, musk ox, gemsbok, but do not yet appear to cause disease under natural conditions.

Zoonotic potential: No

Distribution: Disease may occur worldwide in situations in which clinically susceptible species are in contact with carrier species.

Incubation period: It varies depending on several factors such as amount of virus transmitted and host. In field outbreaks, the incubation period for bison is about 40 to 70 days. Cattle have become ill in as few as 9 days, while other evidence suggests that some cattle may be subclinically infected for 20 months or more before developing the disease. The latter case could be due to a long period of subclinical infection followed by viral reactivation leading to clinical disease. Cattle have become infected 11-73 days after the administration of blood from OvHV-2 infected sick cattle.

Clinical signs: These vary with susceptibility of affected species. Highly susceptible species may have a peracute course with few to no clinical signs or sudden death after non-specific signs such as depression, weakness, and diarrhea. Acute disease may involve high fever and a loss of appetite. Clinical signs may include mucous membrane ulceration and inflammation, high fever, oral and nasal exudates, corneal opacities (common in domestic cattle), and lymphadenopathy. Additional signs may include: neurologic signs, diarrhea, arthritis, and skin lesions may also develop.

Domestic sheep: Systemic necrotizing vasculitis or “polyarteritis nodosa” has been found to be associated with OvHV-2.

Free-ranging bighorn sheep: muscle atrophy, marked weight loss, and bilaterally symmetric alopecia with hyperpigmentation and crusting over the face, medial surfaces of the pinnae, dorsal trunk, distal limbs, perineal area, and tail was found in a free-ranging bighorn sheep affected by OvHV-2.

Moose: lymphocytic vasculitis in the brain and panuveitis were seen in a captive moose which died of CPHV-2.

Carrier species (wildebeest) do not typically develop clinical signs.

Post mortem, gross, or histologic findings: These may vary with disease severity and course, but often include “inflammation and epithelial necrosis in the gastrointestinal, respiratory, and urinary tracts, with lymphoproliferation, infiltration of nonlymphoid tissues (particularly the renal cortex and periportal areas of the liver) by lymphoid cells, and vasculitis”.

Diagnosis: PCR (polymerase chain reaction) is the method of choice for viral detection.

Serological tests for antibodies include competitive inhibition ELISA (cELISA), immunoperoxidase test (IPT), neutralization test (NT) and others. Detection of antibodies indicates infection, not necessarily disease.

In susceptible species such as cattle, bison and deer, detection of MCF antibodies indicates infection, but is not diagnostic of disease; lack of antibodies when performing cELISA usually is an indicative of a lack of infection, with the exception of very early stages of infection <1 week before antibodies can be produced.

Material required for laboratory analysis: Antibody testing by cELISA - serum or plasma.

Antemortem detection of viral DNA by PCR - whole blood in EDTA.

Postmortem detection of viral DNA by PCR – preferred fixed tissue samples - lymph node or spleen; but lung, brain, kidney, and intestine among others are also acceptable.

Relevant diagnostic laboratories:

Washington Animal Disease Diagnostic Laboratory (WADDL), Pullman, Washington.

<http://www.vetmed.wsu.edu/mcf/>

National Veterinary Services Laboratories (NVSL) in Ames, Iowa.

NSW Government-Department of Primary Industries.

Treatment: No treatment is available or usually ineffective. Supportive care may be administered, but

MALIGNANT CATARRHAL FEVER (MCF)

disease is often acute and fatal in highly susceptible species. Some animals may die without clinical signs. Occasional reports of recovery in treated cattle exist, but some cattle may also recover without treatment.

Prevention and control: Separate clinically susceptible species from carrier species such as wildebeest, domestic and exotic sheep, and domestic and exotic goat species. Bovids, particularly bison and water buffalo, are highly susceptible to MCF. Exotic members of the bovidae family such as bongo antelope have died from MCF traced back to an exotic goat species. Wildebeest-associated MCF has occurred in domestic cattle in the U.S. when the two species were housed together.

Cervids should not be mixed with sheep, goats, or wildebeest.

Prevent direct contact and fomite transmission. Transmission of the wildebeest-associated form (AIHV-1) and the sheep-associated form (OvHV-2) is believed to occur primarily from either direct contact with infected body fluids or secretions, or via fomites such as water sources, feeders, caretakers, and birds. Calving is considered a high risk period for transmission.

Use precautions to prevent aerosol transmission. Transmission of the disease over relatively short distances has occurred, indicating that direct contact is not absolutely necessary. Aerosol transmission is a significant mode of transmission of OvHV-2 in domestic sheep.

Suggested disinfectant for housing facilities:

Herpesviruses causing MCF are typically “fragile and quickly inactivated in harsh environments”, so common disinfectants are likely effective. However, if heavy organic debris is present the OIE recommends 3% sodium hypochlorite.

Notification: The wildebeest-associated and sheep-associated forms are reportable diseases under USDA-APHIS-VS National Animal Health Reporting System. MCF clinical signs may appear similar to foreign animal diseases such as rinderpest and foot and mouth disease.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Clinically susceptible species should not be introduced to carrier species. Clinically susceptible species should be physically separated from carrier species. In addition, separate keeper staff and equipment should be used to prevent fomite transmission.

Conditions for restoring disease-free status after an outbreak: See prevention and control measures.

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American Association of Zoo Veterinarians Infectious Disease Manual
PSITTACID HERPESVIRUS 1/PACHECO'S DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Psittacines, rarely passerines	Ingestion of contaminated material from oral secretions or feces. Aerosol route is possible but not proven.	Death with few premonitory signs. Rarely nonspecific signs, including lethargy and the presence of bile pigments in urine. Three of four PcHV-1 genotypes have been associated with oral and cloacal mucosal papillomas.	Dependent on viral genotype and species of birds, death can range from single birds to flock majority. Virtually all birds showing signs of the acute form will die unless treated. Mucosal papillomas cause considerable morbidity but are rarely fatal.	Acyclovir is very effective at stopping outbreaks when the entire flock is treated.	Closed flocks, isolating or culling subclinical carriers, and testing new arrivals at quarantine may assist in disease prevention.	No

Fact Sheet compiled by: Nadia Stegeman

Sheet completed on: 3 August 2011; updated 20 March 2013

Fact Sheet Reviewed by: David N. Phalen; Lauren V. Powers

Susceptible animal groups: Psittacines; and less commonly passerines although it has been reported in birds such as finches, canaries, and barbets (family Lybiidae).

Causative organism: Psittacid herpesvirus 1 (PsHV-1), formerly "Pacheco's disease (PD) virus", has 4 genotypes corresponding to 3 serotypes, and is an alphaherpesvirus. The pathogenicity of genotype varies significantly although all four genotypes have been shown to cause PD. Recently, in African grey parrots, psittacid herpesvirus 2 (PsHV-2) was identified from cloacal mucosa.

Zoonotic potential: None reported.

Distribution: Presumably worldwide due to bird trade, but it is most prevalent in densely populated captive psittacine collections. Case reports have documented confirmed disease in North America, Europe, Africa, Australia/New Zealand, the Middle East and Asia. A recent study suggests a 7% prevalence of PsHV-1 infection in the general US population of parrots. It is suspected that these viruses have evolved with Central and South American parrots.

Incubation period: Experimentally, 5-10 days to establish infection. Papillomas develop within a year of infection.

Clinical signs:

Acute: Death with no premonitory signs, aside from possible depression, anorexia, diarrhea, and yellow urates (biliverdinuria). In antemortem clinical chemistries, marked AST elevation can be found.

Chronic: Oral/cloacal papillomas that produce tenesmus and can be associated with frank blood from the

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cloaca. In extreme cases, and when bile duct and pancreatic duct carcinomas develop, chronic wasting can occur.

Post mortem, gross, or histologic findings:

Due to rapid death, acutely affected birds may show no gross lesions. However, when abnormalities are present, they may be in the liver, spleen, kidneys and intestines. Histopathological findings include multi-organ (e.g., spleen, intestines, pancreas, trachea, air sacs) necrotizing lesions, hemorrhage and congestion of the liver, spleen, and kidneys and hepatomegaly or splenomegaly may be seen. Intranuclear inclusion bodies (Cowdry Type A) are most common in the liver, but have been demonstrated in the kidneys, spleen, pancreas and small intestines.

In the chronic form of the disease, mucosal papillomas may be seen, most commonly in the oral and cloacal mucosa or upper gastrointestinal tract. These lesions are found in the disease complex termed internal papillomatosis of parrots (IPP). A high prevalence of carcinomas in the bile duct and pancreatic duct has been observed in aviaries where IPP had been noted in birds infected with PsHV-1 genotype 3. These tumors can be, but not always, detected with coelomic ultrasound and are associated with a rise in serum GGT. Many ventricular and cloacal carcinomas appear to be caused by PsHVs. Cloacal carcinomas have a grave prognosis due to the reportedly high metastatic rate. One case report discusses chronic active pancreatitis (with diabetes mellitus, weight loss, PU/PD, glucosuria, and hyperglycemia) associated with PsHV-1 infection in a cockatiel (*Nymphicus hollandicus*).

Diagnosis:

Inapparently infected birds: Gross identification of mucosal papillomas. PCR or real-time PCR on cloacal, oral mucosal swabs and whole blood. It is important to note that the virus is shed intermittently, leading to the possibility of false negative results by PCR. However, the majority of birds remain PCR positive at all time; the sensitivity of mucosal swabs is higher than that of whole blood. Serology has practical merit. Birds that are serologically positive are likely latently infected.

Post-mortem specimens: Characteristic histologic findings, electron microscopy, cell culture, immunofluorescent antibody staining and PCR.

Material required for laboratory analysis: It is important to note that the virus is shed intermittently, leading to the possibility of false negatives. However, the majority of birds remain PCR positive at all times. The sensitivity of mucosal swabs is higher than that of whole blood.

Pacheco's Disease: Whole blood, tissues (frozen liver/spleen or swabs), frozen liver/spleen for culture, choanal/cloacal swabs, histopathology of liver, spleen, pancreas, intestine, crop

Subclinically infected birds: Choanal/cloaca swabs, serum, whole blood

Relevant diagnostic laboratories:

Veterinary Molecular Diagnostics

5989 Meijer Dr. Suite 5

Milford, Ohio 45150

513-576-1808

(PCR based DNA probe; can detect all PsHV-1 variants)

Treatment: Acyclovir (80-100mg/kg three times a day for 10 days; Zovivax, GlaxoSmithKline) has been shown to reduce the sickness and death of PsHV-1 affected birds and generally, after a few days of treatment, all deaths cease. Acylovir has been associated with kidney damage in some species but this problem is uncommon or rare.

Mucosal papillomas typically wax and wane and only require surgical intervention in extreme cases.

Surgical resection of papillomas is a palliative treatment.

Prevention and control: Screening and isolation of infected individuals is critical. PCR positive birds

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should be housed separately from other parrots. Macaws, conures and Amazon parrots and should be carefully examined prior to acquisition. That being said, not all PcHV-1 genotypes are serologically cross reactive, meaning that infection with one variant of the virus does not protect from infection from another. Control methods in the midst of an outbreak are debated. While some support catching and moving individual birds, others advocate minimal disturbances until the outbreak is over. Immediate treatment of exposed birds with acyclovir at 1mg/ml drinking water and 400mg/kg of soft mash) is indicated. Gavage feeding at 70-100mg/kg BID has also been suggested.

Commercial monovalent vaccine (killed virus) for PsHV-1 is derived from a single, unreported serotype. It is not known how much protection this vaccination provides against variants other than genotype 1. Complications from vaccine include injection granulomas and acute death. Cockatoos appear to be overrepresented in populations experiencing complications. Additionally the product, Psittimmune PDV (Biomune in Lenexa, Kansas) no longer appears available. One case report suggests autogenous, formalin-inactivated vaccine with aluminum hydroxide gel adjuvant may stop virus spread, decreasing morbidity and mortality.

Individuals with this disease can continue to be used as breeders. However, all eggs must be artificially incubated and hand-raised until vertical transmission impacts are better established.

Suggested disinfectant for housing facilities: As PsHV-1 is an enveloped virus, it is readily inactivated by commonly used disinfectants. EPA approved disinfectant (virucidal, fungicidal, bacteriocidal) or sodium hypochlorite (bleach) solution (800ppm) is effective for most herpesviruses. It can also be inactivated by heating to 56°C for 10 minutes or by exposing it to pH <5.

Notification: None required, although notification to institutions that received birds previously exposed to chronic shedders is recommended.

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Isolating infected and exposed individuals, testing exposed individuals after clinical signs in the aviary subside.

Experts who may be consulted:

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PSEUDORABIES (AUJESZKY'S DISEASE)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Swine are the only natural hosts of the virus. However, it affects a wide range of mammalian hosts, excluding humans and some non-human primates.	Primarily through venereal route or horizontal transmission via oral, nasal, digestive or reproductive mucosa. Other potential methods of transmission include mechanical (via fomites or vehicles) and viral aerosolization.	Pyrexia, depression, anorexia, tremors, incoordination, vomiting, ptyalism (foaming), blindness, convulsions, coma, and death.	High and low virulence strains are known. Disease is highly virulent and often 100% fatal in susceptible non-suids.	No treatment is available. Surviving animals are infected for life.	Vaccination is available with regulatory permission and is effective at reducing clinical signs. Infected operations are quarantined and infected animals removed. Feral swine may be monitored to identify high risk areas.	No.

Fact Sheet compiled by: Kerri Pedersen and Yvonne Nadler

Sheet completed on: 9 September 2013

Fact Sheet Reviewed by: Tom Deliberto; Troy Bigelow; Evan Sorley; Lowell Anderson; Mark Schoenbaum

Susceptible animal groups: Domestic and feral swine are the primary hosts but disease can be transmitted to other mammalian species. The virus is known to infect deer, foxes, raccoons, skunks, bears, rats, coyotes, mink and panthers. Cattle, goats, dogs, and cats also are susceptible to the disease. Experimental infection has been seen in rhesus monkeys, marmosets and several bird species.

Causative organism: Pseudorabies also referred to as Aujeszky's disease is caused by Suid herpesvirus 1 *Varicellovirus* in family *Herpesviridae*.

Zoonotic potential: None

Distribution: Pseudorabies occurs worldwide, but has virtually disappeared from domestic pigs in several parts of Europe, Great Britain, and New Zealand. US implemented an eradication program and as of 2004, all commercial swine were considered pseudorabies-free. However, pseudorabies is considered endemic in US feral swine.

Incubation period: Typically 2-6 days and suckling pigs have shorter incubation period of 48 hours.

Clinical signs: Clinical signs are variable and morbidity and mortality decreases with increasing age in swine. Pregnant sows may abort or have stillborn young, whereas newborn piglets may present with neurologic disease or high mortality rates especially in piglets from herds with no prior exposure. Weaned pigs present respiratory illness with fever, anorexia, and weight loss. Sneezing, rubbing of the nose and coughing may occur with or without trembling and incoordination. Adult swine can exhibit mild respiratory distress, fever during acute

PSEUDORABIES (AUJESZKY'S DISEASE)

infection. Surviving individuals become lifelong carriers of the virus while exhibiting minimal to no further clinical signs. In swine, the virus may become latent in cranial nerve ganglia, may recrudesce and shed live virus months later.

Pseudorabies is virulent in susceptible animal species, which often experience intense pruritus or “mad itch” which causes them to scratch and bite themselves. Other clinical signs include respiratory problems, general neurologic signs, weakness, convulsions, and fever.

Clinical pathological, gross, and histopathological findings: Gross lesions are minimal or absent, and none are pathognomonic. Serous or fibrinonecrotic rhinitis may be found. Tonsillar inflammation may be observed as fibrinous exudate or an erosive fibrinonecrotic lesion. Small (<1 mm), pale foci in liver and/or spleen appear as slightly irregular or with vague edges, and not a crisp, well-demarcated appearance; in young piglets, liver lesions are more common than in adults but occur only occasionally. Reddened foci may be scattered on the pleura of the lungs and with or without pulmonary edema, congestion, or consolidation. Non-suppurative meningoencephalitis is noted upon examination of white and gray matter; mononuclear perivascular cuffing; neuronal necrosis; thickened meninges.

In non-suid species, edema, congestion and hemorrhage in the spinal cord have been noted. These lesions are usually found in the portion of the spinal cord that innervates the area of pruritus. Microscopically, cellular infiltration and neuronal degeneration is seen. CNS lesions are similar to those found in pigs, but milder in severity.

Diagnosis: Serologic tests for virus or antibody detection are available and include serum neutralization (SN), latex agglutination (LA), and enzyme-linked immunosorbent assay (ELISA). A fluorescent antibody test on tissue sections, immunohistochemistry on formalin-fixed tissues, or virus isolation may be used to identify virus in the brain, tonsils, and spleen. A polymerase chain reaction (PCR) test has been described but is not in common use.

Material required for laboratory analysis: Serum or tissue with brain, spleen and lung are preferred tissues for diagnosis in suids. Diagnostic samples should be kept cold for virus isolation submission.

Nasal swabs, or samples of oropharyngeal fluid or tonsil tissue from suspected porcine can be used for virus isolation. The virus may also be found in the lung, spleen, liver, kidney, or lymph nodes. In other species, the virus may be isolated from the pruritic area of the skin, and from the spinal cord area innervating the pruritic area.

Relevant diagnostic laboratories: Most diagnostic laboratories can test for pseudorabies.

Treatment: No treatment is available.

Prevention and control: Vaccination can be effective at preventing and controlling outbreaks in domestic swine; permission must be obtained from state animal health official for vaccine usage. State and Federal regulations prevent movement of infected pigs and monitoring to identify newly infected animals. Since feral swine are a known reservoir, measures are taken to prevent contact between feral and domestic swine. Infected domestic herds are placed under quarantine. Infected animals movements are controlled and regulated. In severe cases depopulation is a method of control.

The risk to zoological animals exists from biosecurity breaches allowing infected feral swine (reservoirs) or other wildlife from coming into contact with zoological animals. Sound biosecurity measures are highly effective in preventing introduction into zoological facilities.

Suggested disinfectant for housing facilities: PRV is susceptible to inactivation by sodium hydroxide, bleach, iodine-based products, phenolic disinfectants, quaternary ammonium compounds, formaldehyde, and chlorhexadine. These disinfectants are not effective unless contaminated objects have been thoroughly cleaned before the disinfectants are applied. PRV is also susceptible to thermal inactivation.

Notification: Pseudorabies is a notifiable disease only when found in commercial production swine.

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PSEUDORABIES (AUJESZKY'S DISEASE)

Measures required under the Animal Disease Surveillance Plan: http://www.aphis.usda.gov/vs/nahss/swine/prv/prv_surveillance_plan_final_draft_04_16_08.pdf for current National Surveillance Plan. Proposed changes Veterinary Services National Surveillance Plan under review in 2013 http://www.aphis.usda.gov/animal_health/animal_dis_spec/swine/downloads/manage_swine_bruc_n_pseu_virus_10-086-1_concept.pdf .

Measures required for introducing animals to infected animal: Do not introduce non-infected animals to infected animals; animals should be tested prior to moving them and prior to introducing to known disease-free animals.

Conditions for restoring disease-free status after an outbreak: In commercial swine herds, quarantine, animal testing and removal from herd will be under the direction of an Accredited veterinarian. Premises should be disinfected and left vacated for at least 30 days following removal of infected animal.

Experts who may be consulted: Federal and state veterinary authority (AVIC and state veterinarian, respectively).

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Saimiriine Herpesvirus 1 (SaHV1)

Animal group(s) affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Natural host species: squirrel monkeys, spider monkeys, capuchins and possibly woolly monkeys; Susceptible species: owl monkeys, titi monkeys, spider monkeys, all marmosets and tamarins	Fecal-oral route, aerosols, fomites, sexual transmission	Natural hosts: oral lesions (rare) or asymptomatic; owl monkeys, titi monkeys, marmosets and tamarins: anorexia, oral lesions, pruritis, sneezing, nasal discharge, diarrhea, swollen eyelids, 76-100% mortality (2-3d after onset of clinical signs)	Natural hosts: asymptomatic to oral lesions; owl monkeys, titi monkeys, marmosets, and tamarins have a 76-100% mortality rate	None; May try herpes antiviral therapy (famcyclovir, gancyclovir) although no data is available on efficacy.	Do not mix squirrel monkeys, spider monkeys, and capuchins with owl monkeys, titi monkeys, marmosets, or tamarins. Do not share cleaning utensils, enrichment or perching materials, etc. due to risk of fomite transmission. Serological screening of natural hosts.	Possible (but only one report of non-fatal encephalitis in a human secondary to a squirrel monkey bite).

Fact Sheet compiled by: Elizabeth E. Hammond

Sheet completed on: 14 September 2018

Fact Sheet Reviewed by: Genny Dumonceaux

Susceptible animal groups Natural hosts: squirrel monkeys (*Saimiri sciureus*), spider monkeys (*Ateles* sp), capuchins (*Cebus* sp), possibly woolly monkeys (*Lagothrix* sp). Aberrant hosts: Owl monkeys (*Aotus* sp), marmosets (*Callithrix* sp), tamarins (*Saguinus* sp), titi monkeys (*Callicebus* sp)

Causative organism: Saimiriine herpesvirus (Alphaherpesvirus); aka, Herpesvirus tamarinus, Herpesvirus T, Herpesvirus platyrrhinae

Zoonotic potential: slight – one case report of a lab worker with non-fatal encephalitis secondary to a squirrel monkey bite

Distribution: South and Central America, worldwide in captivity for natural hosts

Incubation period: 7-10 days

Clinical symptoms: Squirrel monkeys, spider monkeys, capuchins: oral lesions (rare) or asymptomatic; owl monkeys, titi monkeys, marmosets, tamarins: anorexia, oral lesions, pruritus, sneezing, nasal discharge, diarrhea, swollen eyelids, 76-100% mortality (2-3d after onset of clinical signs); once infected, always a carrier (latent infections with intermittent shedding); resembles herpes simplex infection

Post mortem, gross, or histologic findings: Gross: ulcerative dermatitis, mucosal ulceration
Histopathology: hepatic necrosis with multinucleated syncytial cells and intranuclear inclusion bodies. Also necrosis in spleen, kidney, lung, intestines, and adrenal gland; necrosis of the epidermis with multinucleated

Saimariine Herpesvirus 1 (SaHV1)

giant cells with intranuclear viral inclusions. Inflammatory response may be minimal in acute disease. Lesions may be minimal if encephalitis is present.
Diagnosis: virus isolation, molecular techniques, serology, histopathology, clinical signs and history of contact with natural host
Material required for laboratory analysis: serum, whole blood, skin, oral mucosa, liver
Relevant diagnostic laboratories: VRL Laboratories, PO Box 40100, 7540 Louis Pasteur, Suite 200, San Antonio, TX 78229, USA, 1-877-615-7275, www.vrl.net ; Zoologix, 9811 Owensmouth Ave, Ste 4, Chatsworth, CA 91311-3800, USA, 818-717-8880, www.zoologix.com
Treatment: none; supportive care
Prevention and control: Prevent contact between squirrel monkeys, spider monkeys, and capuchins, with owl monkeys, titi monkeys, marmosets, or tamarins. Prevent cross contamination by excluding the use of shared equipment and enrichment devices. A live vaccine has been effective in owl monkeys; however, vaccine-induced disease has been observed.
Suggested disinfectant for housing facilities: most common disinfectants will kill the herpesvirus, including chlorine bleach and quaternary ammonium
Notification: None
Measures required under the Animal Disease Surveillance Plan: None
Measures required for introducing animals to infected animal: Do not introduce natural host species to susceptible species; once infected, animals will remain carriers and sporadically shed virus.
Conditions for restoring disease-free status after an outbreak: Disinfect environment, cleaning tools, furniture, etc. Avoid mixing natural hosts with susceptible species.
Experts who may be consulted:
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SA-8 (CERCOPITHECINE HERPESVIRUS 2)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Cercopithecidae (predominantly African green monkey and baboon)	Direct contact	Mostly asymptomatic, transient vesicular stomatitis in young animals. Small vesicles and pustules can be found on genital and oral mucous membranes.	Mild. Oral and genital lesions. Experimental infection has resulted in pneumonia.	No treatment needed due to nature of infection. Symptomatic treatment for severe lesions would be indicated.	Disease rare despite high virus prevalence.	Not reported.

Fact Sheet compiled by: Sam Rivera; updated by Jan Ramer

Sheet completed on: 1 June 2011; updated 23 July 2013

Fact Sheet Reviewed by: Sam Rivera; Kevin Brunner

Susceptible animal groups: Cercopithecidae

Causative organism: SA-8 (Cercopithecine herpesvirus 2).

Zoonotic potential: None

Distribution: African green monkey natural host, but is found in captivity worldwide. Common asymptomatic infection of baboons.

Incubation period: Unknown

Clinical signs: Most infections are clinically silent, transient vesicular stomatitis in young animals. Oral and genital vesicles and pustules are possible. Occasional severe genital lesions have been noted with inguinal lymphadenopathy.

Post mortem, gross, or histologic findings: Vesicular stomatitis.

Diagnosis: Serology, virus isolation.

Material required for laboratory analysis: Material from the lesions, whole blood or serum.

Relevant diagnostic laboratories:

Pathogen Detection Laboratory
 California National Primate Research Center
 Road 98 & Hutchison
 University of California
 Davis, California 95616
 (530) 752-8242
 Fax: (530) 752-4816
 PDL@primate.ucdavis.edu
 http://pdl.primite.ucdavis.edu

BioReliance, Serology/PCR Laboratories
 14920 Broschart Rd.

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Rockville, Maryland 20850
Phone: (301) 610-2227
Fax: (310) 610-2587
ahs@bioreliance.com

Virus Reference Laboratories, Inc.
7540 Louis Pasteur Road
San Antonio, Texas 78229
(877) 615-7275
Fax: (210) 615-7771

Zoologix Inc.
9811 Owensmouth Avenue, Suite 4
Chatsworth, California 91311-3800
818-717-8880
Fax: 818-717-8881
info@zoologix.com

Treatment: Symptomatic treatment for severe lesions would be indicated.

Prevention and control: Disease rare despite high virus prevalence.

Suggested disinfectant for housing facilities: Lipid solvents, soap, UV-light, heat.

Notification: None at this time.

Measures required under the Animal Disease Surveillance Plan: None at this time.

Measures required for introducing animals to infected animal: None at this time.

Conditions for restoring disease-free status after an outbreak: Latent infection, possible life-long infection.

Experts who may be consulted:

Chih-Ling Zao, PhD
Chief Scientific Officer
VRL Laboratories
P.O. Box 40100
7540 Louis Pasteur, Suite 200
San Antonio, Texas 78229
Phone: 877-615-7275
Fax: 210-615-7771
chih-ling.zao@vrl.net

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TESTUDINID HERPESVIRUSES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All tortoises are considered susceptible.	<p>Experimental work has shown that intranasal and intramuscular inoculation is followed by development of the disease.</p> <p>Close contact is considered one of the most relevant events for natural transmission to occur.</p> <p>It is not clear if aerosolization plays a significant role.</p>	<p>Classic, but not necessarily always occurring, clinical signs include diphtheronecrotic stomatitis and glossitis, nasal, ocular and oral discharge, conjunctivitis, lethargy, anorexia, respiratory and neurological signs.</p>	<p>Depends of the species affected and on the viral genotype involved. TeHV1, -2, and -3 have been associated with clinical disease. More recently also TeHV4 was detected in a clinically ill tortoise coinfecting with <i>Mycoplasma</i> sp. Close to 100% morbidity and mortality have been reported for naïve Hermann's tortoises while Greek tortoises are considered more resistant.</p>	<p>Antivirals (e.g., acyclovir and ganciclovir) have been shown to be effective <i>in vitro</i>.</p>	<p>Separation of the diseased individuals from those clinically healthy animals; serologic and molecular testing of the exposed individuals; six months quarantine and serological testing; avoiding species mixing and crowding; disinfection of the enclosures with virucidal agents.</p>	No

Fact Sheet compiled by: Francesco C. Origgi

Sheet updated on: 30 August 2013; updated 2018

Fact Sheet Reviewed by: Douglas R. Mader

Susceptible animal groups: All tortoises are considered susceptible.

Causative organism: Testudinid herpesviruses (TeHVs) (previously known as tortoise herpesviruses) have four currently known genotypes: TeHV1, -2, -3 and -4. In diseased individuals, TeHV1, -2 and -3 have been detected or isolated. The TeHV3 genotype includes at least two genogroups (A, B) that might be characterized by distinct virulence but both lethal. Genogroup A is the most common, genogroup B is putatively the most virulent. More recently, TeHV4 has been detected in a clinically ill leopard tortoise (*Stigmochelys pardalis*).

Zoonotic potential: No

Distribution: TeHV-1 and -3 have been detected worldwide. TeHV-2 has been detected only in the US until now. TeHV-4 has been detected in the US in a clinically healthy, imported Bowsprit tortoise and in a captive-bred leopard tortoise coinfecting with *Mycoplasma* sp. in Europe. Both Bowsprit and leopard tortoises are African tortoises. TeHV-1 and -3 have been detected more commonly in the genus *Testudo* but also in several other tortoise species not autochthonous of the Eurasian area. TeHV-2 has been detected only in desert tortoises (*Gopherus agassizii*) up to date. It is likely that at least for some of the genotypes, the current area of distribution might only partially overlaps with their original ones because of the extensive movements that the species have undergone especially because of the pet trade. According to multiple studies conducted on wild tortoises, the following geographical classification of the genotypes has been proposed: TeHV-1 and -3, Eurasian; TeHV-2, American; TeHV-4, African.

Incubation period: Following experimental infection in Greek tortoises, the first clinical signs were recorded 7 to 12 days post inoculation. The overall course of the disease lasted 2.5 weeks.

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Clinical signs: The disease originally was called “stomatitis and rhinitis disease” or “running nose” and it was named after its most common clinical signs that include a diphtheronecrotic stomatitis and a glossitis with nasal, oral and eye discharge. The discharges are generally clear, intermittent and recurrent. Monolateral or bilateral recurrent conjunctivitis is also common. Cervical edema and epistaxis in severe cases has been reported. The oral plaques have been shown to develop and eventually fully regress in the survival animals within a 2.5 weeks period of time during an experimental transmission study in Greek tortoises. Following the loss of the crusts covering the healing plaques, no scar can be detected after the resolution of the oral lesions. A band of chalky white material can be observed along the rim of the external aspect of the mandible after the oral discharge has resolved. Respiratory and neurological signs might follow along more general signs such as lethargy and anorexia. Not all the clinical signs described above can be detected in the same diseased individuals and some infected tortoise might not develop obvious clinical signs at all. Similar clinical signs were observed also in a recent transmission study carried out with TeHV3 in Herman’s tortoises.

It is important to consider that none of the clinical signs described above are specific for TeHVs, since similar oral plaques have also been described in tortoises infected with less common iridovirus and virus X (topivirus-tortoise picornavirus). Additionally, nasal discharge and conjunctivitis, in absence of stomatitis, are commonly described in tortoises infected with *Mycoplasma agassizii* (an etiologic agent of the upper respiratory tract disease-URTD).

Post mortem, gross, or histologic findings: Classic gross lesions include diphtheronecrotic plaques over the mucosa of the oral cavity and the tongue, occasionally extending over the mucosa of the esophagus and of the trachea up to the lung. Hepatomegaly and enteritis have also been described. The histologic hallmark of the disease is the presence of eosinophilic to amphophilic intranuclear inclusions in most of the epithelial tissues. Inclusions can also be observed within the central nervous system either associated or not with inflammation. Inclusions are likely to be detectable for a limited time during the initial phase of the disease.

Diagnosis: The clinical diagnosis relies on the detection of the clinical signs described above. The clinical diagnosis requires the confirmation by laboratory testing. Multiple molecular and serological tests are available for the diagnosis of Testudinid herpesvirus infection.

Serology: The available serological tests comprise an ELISA and a serum neutralization test (SNT), while the molecular diagnostic tests comprise multiple PCR protocols for the partial amplification of different herpesvirus genes. The ELISA test has been developed to detect TeHV3 exposure and it has been validated for Greek and Hermann’s tortoises. The test detects the large majority of the antibody developed by the host against the virus after seroconversion. Although the test can detect the exposure to TeHV3 relatively early following the infection, for a reliable diagnosis it is recommended to test a suspected individual two times no less than 8 weeks apart. A modified version of the same test has been shown to be able to detect TeHV2 exposure in desert tortoises, although in this format the test has not been fully validated. SNT similarly to the ELISA, allows the detection of the exposure of an individual to a Testudinid herpesvirus due to the presence of circulating anti-TeHVs antibody (serum neutralizing in this case) following seroconversion. This test can be applied to any species of tortoise and it requires live virus to be carried out. SNT can detect seroconversion to TeHVs 2-5 weeks after the ELISA test. For this reason, it is suggested to test the suspected animals two times 10-12 weeks apart for a reliable detection of the occurred seroconversion when using the SNT. Both ELISA and SNT show comparable specificity and sensitivity.

Molecular diagnosis: Several PCR protocols have been developed for the diagnosis of TeHVs infection. A PCR test targeting the partial sequence of the viral DNA polymerase gene is available for the specific detection of TeHV1. Another protocol has been developed for the detection of the partial sequence of the helicase gene of TeHV3. The same protocol allows also the detection of the homologous gene of the TeHV1 genotype following a specific modification of the test conditions. A PCR test directed to the amplification of the partial sequence of the ribonucleotide reductase (RR) (large subunit) of TeHV3 has also been developed. This test can also amplify the homologous gene portion of TeHV2, although a specific PCR protocol is also

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available for the partial amplification of TeHV2 RR. Finally a PCR protocol, not specifically developed for TeHVs but more in general for the detection of the members of the family Herpesviridae is also available and allows the detection of all the 4 genotypes of TeHVs known up to date (partial amplification of the DNA polymerase gene; the target is a different region from that used for TeHV-1 amplification). Recently, a PCR protocol has been developed to differentiate viral strains belonging to distinct genogroups (A, B) within the TeHV3 genotype, which might be associated with distinct virulence.

Histopathology: routine histopathologic diagnosis of TeHVs infection is more often considered a post-mortem diagnostic method. It is based on the detection of the classic intranuclear inclusions. An immunohistochemistry and an *in situ* hybridization method are also available for the detection of TeHVs antigen and DNA in tissue, respectively and can be used to enhance the sensitivity of the histopathological diagnosis. Electron microscopy is also commonly used to detect the presence of the virus in tissues.

Viral isolation: Viral isolation of TeHV1 and -3 can be performed on reptilian cell cultures (TH-1 cells, subline B1, ATCC CCL-50). Classic cytopathic effects include cell rounding with cell detachment and lysis (plaque formation).

Material required for laboratory analysis:

Serology: 0.2-0.5 ml serum in plastic tube. Store at 4°C and ship refrigerated.

Virology: Pharyngeal swabs are collected for live animals. For dead animals, it is helpful to send the entire carcass if available or 1 g portions of each organ, ideally. If this approach is not possible, the head (including the tongue) of the suspected individual can be submitted. Place samples in viral transport media with antibiotic (1-2 ml for swabs and 3 ml per each tissue sample). Store the samples at 4°C for very short-term storage and -80°C for long-term storage. Ship samples refrigerated or on dry ice accordingly. Preserve the entire carcass and the head at 4°C and ship refrigerated immediately.

Molecular diagnostic: Same samples described for virology but in this case viral transport media is not required. Samples can be stored and shipped also as described for the virology samples, with the exception that for molecular diagnostic the samples can be stored also at -20°C if -80°C freezers are not available. If only formalin-fixed, paraffin-embedded tissues are available, whole tissue block(s) can be used. If not possible, please send 3 sections 20µ-thick each (per tissue block) in a plastic tube (DNase- and RNase-free). The samples do not need to be refrigerated but should be protected from light.

Histopathology: The entire carcass is preferred if the carcass can be stored at 4°C and shipped immediately refrigerated. If the necropsy is performed *in situ* please collect routine samples of all the organs including the brain. Samples need to be placed into a container with 10% buffered formalin.

Relevant diagnostic laboratories: From US, permits may be required for shipment to international labs:

Wildlife Diagnostic Laboratory at the Centre for Fish and Wildlife Health (FIWI)

University of Bern, Vetsuisse Faculty

Länggassstrasse 122

3012, Bern, Switzerland

+41 31 631 2443

Fax +41 31 631 2635

Francesco.origgi@vetsuisse.unibe.ch

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TESTUDINID HERPESVIRUSES

Staatliches Veterinäruntersuchungamt
Westernfeldtrasse 1
32758 Detmold, Germany
+49 05231 911640
Contact person: Silvia Blahak

Veterinary Laboratory Agency
Weybridge, Woodham Lane, New Haw
Addlestone. Surrey. KT15 3NB
United Kingdom
Contact person: Sally Drury

College of Veterinary Medicine
University of Florida
2015 SW 16Ave.
32610 Gainesville, FL, USA
352 294 4420
childressa@ufl.edu

Treatment: Acyclovir and gancyclovir have been shown to be effective against TeHV-3 *in vitro*. Acyclovir also has been used to treat infected animals at 80mg/kg PO SID or TID. An *in vivo* study in marginated tortoises (*Testudo marginata*) showed that a single administration of this dose acyclovir results in a serum concentration of the drug which is lower than that reported to be effective against the virus *in vitro*. Broad-spectrum antibiotics and supportive rehydration therapy have also been described as part as the therapeutic protocol proposed for TeHVs infected tortoises. The duration of the treatment may vary, but it should be no less than two to three weeks unless otherwise suggested by the clinical evaluation and laboratory testing.

Prevention and control: Tortoises showing clinical signs consistent with TeHVs infection should be isolated from clinically healthy individuals and tested for the presence of TeHVs (PCR, virus isolation) and for the exposure to the virus by ELISA or SNT. All clinical animals should also be treated with antiviral drugs and supportive therapy as appropriate. Serological tests needs to be repeated after 8 (ELISA) or 10-12 weeks (SNT) for all the tortoises showing clinical signs that tested negative at the first sampling.

Clinically healthy exposed animals should be closely monitored and tested for viral exposure similarly to what described above.

Serologically and/or PCR positive animals that have recovered from the disease and clinically healthy animals that have seroconverted (and/or have tested positive by PCR) following viral exposure should be considered carrier for life. Currently, no evidence exists that supports vertical transmission of the virus.

Preventive measures include:

1) All the tortoises entering into established collections should be tested for the presence and exposure to the virus ideally both by molecular and serological tests. Viral isolation is also recommended. Animals should be tested during the quarantine period, which ideally should last no less than 6 months. Molecular diagnostic tests and viral isolation are relevant in the acute stage of the disease and during viral recrudescence when the virus is actively replicating. Serological tests are relevant especially after the acute stage of the disease, during the latency of the virus, when seroconversion has occurred and no active viral replication can be detected. Testing an animal during the acute stage of the disease with serological tests only is likely to provide a false negative result, since seroconversion has not occurred yet. However, this is still recommended since some diseased animals might not be showing a primary infection, but differently a recrudescence of the infection and so they might have already seroconverted. Additionally, for naïve negative animals this would be considered the “pre-immune” serum. Clinically healthy infected animals that have survived the acute stage of the disease are likely to shed the virus only sporadically and this can determine a high rate of false

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negative animals if tested solely by PCR and/or virus isolation at that time. It is recommended to repeat two serological tests no less than 8 or 10-12 weeks apart when running the ELISA test or the SNT, respectively. Occasional “non reactors” do exist.

2) It is very important to avoid mixing of different tortoise species due to evidence of different species-specific resistances/sensitivity to TeHVs. Mixing of different species might be fatal for the less resistant species.

3) It is important to avoid overcrowding, since close contact between tortoises is considered to enhance the transmission of TeHVs.

4) Contaminated soil might contain viable virus for 2-4 up to 9-24 weeks according to the season of the year. Exposure to full sunlight of the contaminated soil might reduce the viability of the virus present.

5) Disposable gloves and shoe cover should be changed when moving from pen to pen and when touching different tortoises. An appropriate hands disinfectant is also recommended.

6) A veterinary health check should be performed ideally every 6 months. For tortoises from temperate climates a health check performed before and after brumation is critical.

Suggested disinfectant for housing facilities: Any proven virucidal compound may be used to disinfect contaminated instrument and/or pen furniture

Notification: TeHVs associated diseases are not reportable at the moment.

Measures required under the Animal Disease Surveillance Plan: N/A

Measures required for introducing animals to an infected animal(s): If a seronegative animal is introduced to a seropositive animal it should be expected that the seronegative animal will seroconvert eventually.

Conditions for restoring disease-free status after an outbreak: N/A

Experts who may be consulted:

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HISTOPLASMOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals	Inhalation (+/- ingestion) of microconidia from environment	Non-specific, reflecting organ involvement	Asymptomatic infection is most common but can progress to rapidly fatal illness	Itraconazole or fluconazole, amphotericin B	Avoid contaminated soil (especially areas where bird feces accumulate in endemic areas)	Not directly but humans are infected from environment

Fact Sheet compiled by: Maria Spriggs

Sheet completed on: 3 August 2011; updated 17 Feb 2018

Fact Sheet reviewed by: Tiffany Wolf

Susceptible animal groups: Mammals, including humans; birds, because of higher body temperature, are not typically susceptible to natural infection although a single case report exists in an Eclectus parrot.

Published zoo/wildlife cases include: dorcas gazelle, snow leopard, Patagonian cavy, skunk, spiny rat, two-toed sloth, nine-banded armadillo, common opossum, paca, African pygmy hedgehog, Bengal tiger, European hedgehog, sea mammals.

Causative organism: *Histoplasma capsulatum*

Zoonotic potential: No, although common-source infection of people and animals is possible

Distribution: Worldwide, except Antarctica. In the US, most common in region of Ohio, Missouri, and Mississippi Rivers. The organism is found commonly in soil that contains bird and bat manure as nitrogen-rich soil supports fungal growth. Bats may play role in spreading disease as they can develop chronic intestinal dissemination and shed yeast in feces. The organism may be inhaled or ingested and may remain within the lungs or disseminate systemically.

Incubation period: 12-16 days

Clinical signs: Subclinical infection is most common. When signs are present, they may be chronic and nonspecific.

Pulmonary form: pneumonia, wheezes, fever, weight loss, cough, depression

Mediastinal lymphadenitis form: hilar lymphadenopathy, cough, respiratory distress

Progressive disseminated form: Any tissue can be involved.

In domestic cats: fever, weight loss, anemia, interstitial lung disease, hepatomegaly, splenomegaly, and, rarely, oral and lingual ulcerations

In domestic dogs: fever, large bowel diarrhea, intestinal blood loss, anemia, depression

Any species: bone lesions, ocular lesions, CNS, skin nodules

Equine abortion: mare appears healthy but placenta involved.

Post mortem, gross, or histologic findings:

Pulmonary form: miliary or larger gray granulomas, may be calcified

Disseminated form: visceral organs are generally thickened, gastrointestinal mucosa hemorrhagic, enlarged liver with variegated pale pattern, lymphadenopathy

Histoplasma organisms are usually numerous in granulomas and infected tissue.

Diagnosis:

Cytology/histopathology (gold standard): Diagnosis can be made by FNA/cytology, especially rectal scrapings, blood film, or abnormal fluids and tissues. With tissue biopsy, organisms are difficult to detect with routine H&E stain, but stain well with PAS, Gomori's methenamine silver, and Gridley's fungal stains. Yeast forms in macrophages and giant cells are round to ovoid structures with thin cell wall and a thin, clear zone between the cell wall and cellular cytoplasm.

Clinical pathology: Non-regenerative anemia, thrombocytopenia, might visualize organism in cells on buffy coat smear.

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Radiography: diffuse interstitial, miliary or nodular infiltrates, hilar lymphadenopathy (dogs>cats), rarely osseous lesions are present and when present, they are more typical in distal limbs.

Culture: lung, skin lesions, or bone marrow give highest yield in disseminated cases

Antigen detection: greatest sensitivity when test both urine and serum, and CSF in CNS cases. Urine *Histoplasma* EIA is highly specific and sensitive in dogs.

Serology: variably reliable, but may be useful in mild cases with negative antigen results

Molecular: PCR not well established, high rate of false negatives in published studies

Material required for laboratory analysis: Serum, urine, tissue or fluid sample for cytology/histopath/culture

Relevant diagnostic laboratories:

MiraVista Diagnostics for antigen testing www.miravistalabs.com (also does azole levels)

Many state and university labs run serology including Cornell, Kansas State

Treatment: Infection can be self-limiting and resolve without treatment, but treatment is recommended. Itraconazole or amphotericin B traditionally is drug of choice. However, fluconazole may be better for ocular or CNS involvement. Wilson et al. (2018) found fluconazole to be an effective treatment in dogs with no difference in survival, remission or disease relapse rates as compared to itraconazole. Posaconazole and voriconazole are newer and effective drugs, but are expensive, and have little information in vet medicine literature. Treatment interval is 4-6 months and at least 1 month after resolution of clinical signs and after antigen concentrations are negative or below 2 ng/mL.

Prognosis is fair to excellent for pulmonary histoplasmosis and guarded to good for disseminated disease.

Prevention and control: Avoid contaminated soil.

Suggested disinfectant for housing facilities: The only proven disinfectant is 3% formalin. If an accumulation of bird or bat manure is discovered in a building, removing the material by hand/broom/shovel is NOT always the best. Leaving the material alone with signs to warn of health risk may be best course of action. Truck-mounted or trailer-mounted vacuum systems are recommended for buildings with large accumulations to reduce risk of dust exposure. It is recommended to use a vendor experienced in removal of infectious materials. See: <http://www.cdc.gov/niosh/docs/2005-109/>

Notification: None required

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Direct transmission from infected animal to human or other animal is unlikely because yeast phase is not as infectious as mycelial phase.

Conditions for restoring disease-free status after an outbreak: N/A

Experts who may be consulted:

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HUMAN METAPNEUMOVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals: all primates should be considered susceptible, particularly great apes. <i>In vivo</i> virus replication in small rodents.	Aerosols, fomites, direct contact with nasal and respiratory secretions. Transmission via feces also possible.	None to severe: lethargy, lack of appetite, upper and lower (less common) respiratory signs e.g., nasal discharge, sore throat, fever, cough.	Mild (prior exposure) to fatal (naïve animals).	Supportive care and treatment for secondary bacterial infections - <i>Streptococcus pneumoniae</i> documented in chimpanzees	Proper and strict enforcement of biosecurity measures; early detection; isolation of infected animals if feasible	Yes

Fact Sheet compiled by: Owen M Slater

Sheet completed on: 24 January 2018

Fact Sheet Reviewed by: James G. Johnson III

Susceptible animal groups: Disease documented in chimpanzees, mountain gorilla, Sulawesi crested macaques and brown-headed spider monkeys. However, all primates should be considered susceptible. Fatalities reported in wild and captive great apes.

Causative organism: Human metapneumovirus (Paramyxoviridae; Pneumovirus)

Zoonotic potential: Yes

Distribution: Worldwide, with highest occurrence between winter – early summer in North America.

Incubation period: Approximately 5-7 days

Clinical signs:

Animals: None to severe including lethargy, decreased appetite, cough (dry or productive), nasal discharge, and sneezing. Clinical signs indistinguishable from respiratory syncytial virus (RSV) or other respiratory viral infections.

Humans: Signs usually consistent with upper respiratory tract infection, and sometimes lower respiratory tract infection (e.g., bronchitis, bronchiolitis, pneumonia), such as fever, wheezing, cough, nasal congestion, and less so, dyspnea, diarrhea, and vomiting, particularly in children. Immunocompetent adults typically have mild clinical signs. Almost all children have been exposed to the virus by 5 years of age and ~100% by 10 years. Reinfection common.

Post mortem, gross, or histologic findings: Gross findings: Nasal discharge, dehydration
Histologic findings: Among those cases with evidence of pneumonia: marked subacute to acute necrotizing bronchiointerstitial pneumonia characterized by bronchial epithelial hyperplasia and necrosis, diffuse alveolar damage with hyaline membranes and type II pneumocyte hyperplasia. Secondary, purulent bronchopneumonia not uncommon.

Diagnosis: Ante mortem: PCR on respiratory specimens and feces; immunofluorescence or enzyme immunoassay on respiratory secretions. Serology on acute and convalescent sera. Post mortem: PCR, VI

Material required for laboratory analysis: Nasopharyngeal swab, oropharyngeal swab, nasal wash, tracheal aspirate or BAL, feces, serum. Pathologic specimens (e.g. lung tissue) also acceptable

Relevant diagnostic laboratories: Labs capable of performing PCR testing on human respiratory viruses.

Treatment: Supportive care especially for treatment of dehydration and secondary bacterial infections. Several fatal cases of hMPV in chimpanzees often have underlying *Streptococcus pneumoniae* infections.

Prevention and control: It is recommended that all employees working in close contact with non-human primates wear gloves and face masks always. Personnel with symptoms consistent with a respiratory infection should stay home and/or not visit free-ranging primate troops. Isolate infected and cohort-infected animals if practical and review and/or enhance biosecurity protocols.

HUMAN METAPNEUMOVIRUS

Suggested disinfectant for housing facilities: Routine disinfection protocols. 0.1% sodium hypochlorite.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: No required measures. However, it is recommended that no introductions occur during active disease. Serology can be performed prior to any new introductions to determine if any animals are naïve and therefore, more likely to develop severe disease.

Conditions for restoring disease-free status after an outbreak: Virus is cleared by infected animals. Disease free status restored after each outbreak.

Experts who may be consulted:

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Robert Koch Institute
Nordufer 20
Berlin, 13353
Germany

Dr. Eileen Schneider
Centers for Disease Control and Prevention
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Atlanta, GA 30333
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INCLUSION BODY DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Snakes, especially members of Boidae and Pythonidae; usually in human care.	Undetermined, likely through direct contact; vertical transmission; <i>Ophionyssus natricis</i> mites may be vector.	Variable: asymptomatic to severe disease. Non-CNS: anorexia, regurgitation, stomatitis, pneumonia, and lymphoproliferative disorders. CNS: disorientation, ataxia, head tremors, and opisthotonos.	Usually fatal.	None. Supportive therapy may be elected in individual cases.	Maintenance of a closed group or strict quarantine of new arrivals and testing with the intent to cull infected snakes; mite control.	No

Fact Sheet Compiled by: Erica Wilson Lipanovich and Rich Sim

Completed on: updated 11 January 2019

Fact Sheet Reviewed by: Rachel Marschang, Tara Harrison

Susceptible Animal Groups: Almost exclusively snakes of the Boidae and Pythonidae families. Similar inclusions have also been reported in palm vipers (*Bothriechis marchi*), Eastern kingsnakes (*Lampropeltis getulus*), and corn snakes (*Elaphe guttata*).

Causative Organism: Family *Arenaviridae*, genus *Reptarenavirus*; this etiological agent has only recently been identified.

- It seems that pathogenic and non-pathogenic reptarenaviruses exist, and pathogenic ones can affect species differently. Arenaviral coinfections may be common in snakes clinical for IBD.
- Older literature suspected a retrovirus was causative.

Zoonotic Potential: No

Distribution: Worldwide.

Incubation Period: weeks to years.

Clinical Signs: Highly variable – infected animals can be asymptomatic or develop severe disease, which can include anorexia, regurgitation, neurological signs (disorientation, ataxia, head tremors, opisthotonus, inability to right itself, and flaccid paralysis), or secondary bacterial infections (stomatitis and pneumonia).

- Pythons are reported to have a more acute, severe, and CNS-involved disease progression without regurgitation or other gastrointestinal signs
- Boas, especially boa constrictors, commonly have regurgitation, anorexia and CNS signs. Boas can maintain high levels of viremia and accumulated widespread intracytoplasmic inclusions without clinical signs, and may have more chronic disease course.
- Lymphoproliferative disorders have recently been associated with IBD in boa constrictors.

Post mortem, Gross or Histological Findings:

- Eosinophilic to amphophilic intracytoplasmic (IC) inclusions in H&E-stained tissue sections; composed of ~68 kDa reptarenaviral nucleoprotein (NP).
- Cells with inclusions can frequently be observed in the absence of associated inflammation.
- Tissue tropism can be diverse. Within the CNS, inclusions may be observed in neurons and glial cells. With acute neurologic disease (e.g. pythons), inclusions are often limited to the CNS. With

INCLUSION BODY DISEASE

chronic disease (e.g. boa constrictors), inclusions can be seen in epithelial cells (enteric, respiratory, and renal), hepatocytes, pancreatic acinar cells, and mononuclear cells.

Diagnosis: As detailed above, finding IC inclusions or virus by PCR in a live python can be challenging; less so for a boa.

- Ante-mortem, diagnostic options include:
 - Light microscopy exam of a peripheral blood smear for IC inclusions in WBCs with H&E or Wright Giemsa stain,
 - [Arenavirus RT-PCR at University of Florida](#) of an esophageal swab or whole blood sample,
 - Tissue biopsies (esophageal tonsils, liver, kidney) obtained via endoscopy with IC inclusions seen on H&E stain +/- [Arenavirus RT-PCR at University of Florida](#).
- For post-mortem, histopathology and/or PCR on brain, kidney, liver, pancreas are recommended.

Of note, immunohistochemical testing of blood and tissues for NP used to be available at University of Florida, but has been discontinued.

Material Required for Laboratory Analysis: Blood, esophageal swabs, serum or biopsies from the liver, tonsils or gastric mucosa. Contact laboratory for handling and shipping instructions.

Relevant Diagnostic Laboratories:

University of Florida Veterinary Diagnostic Lab
April Childress

University of Florida; 2015 SW 16th Ave.; Building 1017 Room V2-186; Gainesville, FL 32608
Phone: 352-294-4420

Please contact April Childress (childressa@ufl.edu) prior to sample submission.

[Sample Submission Form for PCR and sequencing](#)

Treatment: There is no effective treatment. Supportive measures include antimicrobial and fluid therapy. Appropriate environmental conditions, including temperature and humidity, are essential. Diazepam may be useful for treatment of seizures.

Prevention and Control: Quarantine of all incoming snakes for a minimum of 90 days. Good hygiene, prevention of exposure to infected animals, pest control and removal of infected animals. See below for disinfection recommendations. Snakes suspected of IBD should be isolated. Diagnostic samples mentioned above should be collected and submitted for evaluation. Infected snakes should be euthanized and necropsied.

Suggested Disinfectant for Housing Facilities: Arenaviruses are an enveloped RNA virus; they can be inactivated by most detergents and disinfectants including 1% sodium hypochlorite, phenolic compounds, 3% acetic acid, lipid solvents and detergents (e.g. sodium dodecyl sulfate [SDS]), formaldehyde, and glutaraldehyde (2%). Arenaviruses, in general, are inactivated with heating to 56–60°C (122–140°F), exposure to gamma or UV irradiation, exposure to pH less than 5.5 or greater than 8.52, autoclaving, incineration, and boiling.

Notification: No.

Measures Required under the Animal Disease Surveillance Plan: None.

Measure Required for Introducing Animals to Infected Animal: Not recommended.

Conditions for Restoring Disease-Free Status after an Outbreak: Sick snakes should never be introduced into an established collection. It is unknown what percentage of snakes with reptarenavirus infection will develop clinical signs and how many will remain clinically healthy.

Experts Who May Be Consulted:

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INCLUSION BODY DISEASE

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American Association of Zoo Veterinarians Infectious Disease Manual
INFECTIOUS HEMATOPOETIC NECROSIS VIRUS (IHNV)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Salmonids	Horizontal - usually by direct contact with mucus, urine, feces, eggs/milt. Vertical transmission is suspected. Gills may be important portal of entry as virus survives >1 mo in water and sediment. It also is transmitted via insect, annelid, and crustacean vectors.	Lethargy with sporadic hyperactivity, ascites, white fecal casts, dorsal darkening, petechiation, coelomic distension, hemorrhage, exophthalmia, and pale gills. Acute mortalities occur and scoliosis is observed in survivors.	Varies by strain and temperature. Highest mortality in younger fish at 8-15°C. Older animals present lower mortality rates and fewer clinical signs.	Increase temperature to >15°C if possible; consider euthanasia of affected animals.	OIE reportable disease. Excellent biosecurity (isolation and disinfection). Egg disinfection. Culling and disinfection in the face of an outbreak. Increase temperature to >15°C.	No.

Fact Sheet compiled by: Catherine Hadfield

Sheet completed on: 28 November 2010; updated: 5 July 2013

Fact Sheet Reviewed by: Brent Whitaker, E. Scott Weber III

Susceptible animal groups: Salmonids – both freshwater and saltwater, and especially rainbow trout (*Oncorhynchus mykiss*), Atlantic salmon (*Salmo salar*), chinook salmon (*O. tshawytscha*), sockeye salmon (*O. nerka*), and chum salmon (*O. keta*). Generally considered resistant are lake trout (*Salvelinus namaycush*), arctic char, (*Salvelinus alpinus*) and coho salmon (*O. kisutch*).

Causative organism: Family Rhabdoviridae, genus *Novirhabdovirus*, IHNV. Several clades of virus exist with certain clades or strains being isolated within certain geographic regions.

Zoonotic potential: None.

Distribution: Endemic to Pacific coast of North America (Alaska to California). It is now endemic to Japan and continental Europe. Outbreaks in other parts of the US and Asia have occurred.

Incubation period: Temperature dependent, ~5-45 days.

Clinical signs: The clinical presentation is more common in fry and fingerlings. Lethargy with sporadic hyperactivity is seen. Coelomic distension presents due to ascites. Pale fecal casts are observed trailing from vent. Darkening, petechiation, erythema, exophthalmia, and pale gills due to anemia are observed. Rapidly escalating mortalities occur which may reach >90%. Scoliosis and lordosis are common in 5-60% of fry and fingerling survivors. On hematology, leukopenia, neutropenia, and anemia with increased numbers of bilobed erythrocytes may be observed.

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Post mortem, gross, or histologic findings: Petechiation, erythema, and pallor may be observed grossly. Necrosis of renal hematopoietic tissue and spleen; possible focal necrosis in liver and gastrointestinal tract can be seen. Degeneration and necrosis of granular cells in the lamina propria, stratum compactum, and stratum granulosum of the gastrointestinal tract is sometimes considered pathognomonic for IHN. Pleiomorphic intracytoplasmic and intranuclear inclusions in the pancreas can be observed. Older fish show fewer histologic lesions.

Diagnosis: Presumptive diagnosis is based on species, clinical signs, age, temperature, and geographic location. Definitive diagnosis for OIE requires viral isolation followed by molecular or immunologic identification. Other tests are available, e.g., virus neutralization, indirect fluorescent antibody testing, RT-PCR, and staphylococcal coagglutination, but are not approved for surveillance. Of these tests, the staphylococcal coagglutination is the most rapid.

Material required for laboratory analysis: Live fish – mucus or eggs. Dead fish – the same as live and also kidney and spleen by sterile collection or whole fish. Pool tissues from up to 10 fish (>0.5 g) with viral transport media and antibiotics (e.g., 4ml 10% fetal calf serum and 200 IU penicillin, 200 µg streptomycin, and 200 µg kanamycin per ml). Transport at 4°C ASAP.

Relevant diagnostic laboratories: State Fish Health Laboratories; university laboratories specializing in fish virology, e.g., UC Davis Fish Health Laboratory.

Treatment: Increase temperature to >15°C if possible.

Prevention and control: Excellent biosecurity is important prevention measure. For stocking, only acquire disinfected eggs (commonly iodophor disinfection) or from IHNV-free stock. Use virus-free water, or disinfect with ozone or UV. Sterilize feed (e.g., by heat). Consider non-susceptible species in endemic areas; surveillance of the young-of-the-year and female broodstock; and selective breeding to maintain virus-free stock. Commercial vaccine (Novartis) available in US and several products are under trial. In the face of an outbreak, cull and disinfect affected animals and increase temperature for remaining animals.

Suggested disinfectant for housing facilities: Virus is inactivated by formalin, sodium hypochlorite, iodophors, gamma and UV irradiation, pH <4 or >10, or temperatures >60°C for 15 minutes. Resistant to ethanol.

Notification: Reportable disease, must notify the OIE.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Do not introduce susceptible fish to affected animals.

Conditions for restoring disease-free status after an outbreak: When the disease is first detected, an infected zone is established and a buffer zone is established peripheral to the problem. All infected animals are either culled or removed from the infected zone to reduce the risk of disease transmission and the area is disinfected. Biosecurity measures are reviewed and modified as needed within the infected zone. Surveillance is established until no virus is detected for at least 2 years.

Experts who may be consulted:

Dr. James R. Winton
Chief of the Fish Health Section
Western Fisheries Research Center, United States Geological Survey, Seattle, WA
(206) 526- 6587
jwinton@usgs.gov

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KLEBSIELLA

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All vertebrates	Environmental sources; nosocomial infections; fecal-oral; respiratory; cockroach or house fly vector; normal intestinal or oropharyngeal flora	Wide range of clinical presentation; septicemia, abscessation, multiple organ inflammation. Hypermucoviscous phenotype of <i>K. pneumoniae</i> is an emerging pathogen of humans, primates, and free-living seals	Variable; hypervirulent strains emerging pathogen of humans, domestic animals, primates, otariid and phocid seals	Antibiotics with Gram-negative activity pending appropriate sensitivity testing. Multidrug resistant strains of emerging importance in humans and animals.	Good sanitation, disinfection protocols for commissary and medical equipment, pest control with emphasis on roach and fly control.	Potentially zoonotic; close genetic relationships exist between humans, livestock, birds, and free-living seals.

Fact Sheet compiled by: Kelly Helmick

Sheet completed on: 3 August 2011; updated 17 December 2012, updated 21 February 2018

Fact Sheet Reviewed by: June Olds, James Steel

Susceptible animal groups: All vertebrates are susceptible. Epizootics in captive and free-ranging primates, captive rabbits, captive and free-ranging rodents, free-ranging otariid seals, domestic carnivores, domestic ungulates, and humans. Isolated reports in captive civet, gecko lizard, American alligator, brown tree frog. Isolated from wild and rehabilitated seabirds, captive healthy garter snake. *K. pneumoniae* hypermucoviscosity phenotype is an emerging disease of humans, nonhuman primates, and otariid seals associated with increased invasiveness and pathogenicity. Multidrug resistant strains of *K. pneumoniae* emerging in human and animal isolates. Multidrug resistant pathogenic strains of *K. pneumoniae* isolated from a variety of confiscated psittacine and passerine species.

Causative organism: *Klebsiella* sp. (gram-negative bacteria, Enterobacteriaceae); *K. oxytoca* and *K. pneumoniae*. Hypervirulent *K. pneumoniae* strains with a gene-regulated hypermucoviscosity (HMV) capsular phenotype that is a significant contributing factor to pathogen virulence. The HMV phenotype strain is emerging as an important pathogen of humans, nonhuman primates, and otariid seals. Multi-antibiotic *K. pneumoniae* resistant strains are emerging pathogens of veterinary and human importance.

Transmission: Normal inhabitant of soil and water, benign inhabitant of the gastrointestinal tract and oropharynx, can colonize medical equipment, contamination of foodstuffs. Hospital and nosocomial infections in humans and domestic animals. Houseflies and cockroaches carry multidrug resistant strains of *K. pneumoniae*. Fecal contamination is a common transmission method for cattle with mastitis. Human microbial marine pollution is a suspected source for isolates obtained from free-living seals. Dissemination through the respiratory tract occurs in humans, African green monkeys (*Chlorocebus aethiops sabaesus*), and is suspected in California sea lions (*Zalophus californianus*). A hypervirulent *K. pneumoniae* was isolated on oropharyngeal swab of a captive black-and-white ruffed lemur (*Varecia variegata*) that survived infection, but no isolates obtained from fecal and oropharyngeal swabs of unaffected conspecifics. *K. oxytoca* and hypervirulent *K. pneumoniae* detected on rectal swab from free-living African green monkeys (*Chlorocebus aethiops sabaesus*). Hypervirulent *K. pneumoniae* detected in the oropharyngeal tissues, intestine, and renal tubules of California sea lions; findings may represent normal flora and/or other potential routes of transmission for this species. Bacterial virulence factors and host factors that promote *Klebsiella* infection and disease not fully understood.

Zoonotic potential: Zoonotic potential of *K. pneumoniae* HMV phenotype is unknown, but is an emerging disease of humans and has been isolated from domestic animals, nonhuman primates, phocid seals, and otariid seals with clinical illness. Close genetic relationships between *K. pneumoniae* isolates from humans, livestock, birds, and free-living seals.

Distribution: Worldwide

KLEBSIELLA

Incubation period: Undetermined
Clinical signs: Causes a wide range of clinical presentation in affected vertebrates: anorexia, lethargy, pneumonia, septicemia, hypopyon, endophthalmitis mastitis, metritis, meningitis, peritonitis, urinary tract infections, abscessation. Common clinical presentations of domestic animals include mastitis (cattle); bacteremia (calves); metritis (horses); septicemia and pneumonia (foals); pneumonia, urinary tract infection, and enteritis (dogs); polyarthritis (goats); associated with stomatitis and dermatitis in reptiles; suppurative otitis in lemmings (<i>Dicrostonyx</i> spp.). Marine mammals: Mortality from natural infection with hypervirulent multidrug resistant <i>K. pneumoniae</i> caused acute to subacute respiratory infection in stranded juvenile, subadult, and adult California sea lions (<i>Zalophus californianus</i>); meningoencephalitis in New Zealand sea lion (<i>Phocarctos hookeri</i>) pups; cervical abscessation, pyothorax, omphalitis, and peritonitis in stranded common seals (<i>Phoca vitulina</i>). Primates: Mortality from natural infection with hypervirulent multidrug resistant <i>K. pneumoniae</i> caused septicemic infection and suppurative meningoencephalitis in a captive cynomolgus monkey (<i>Macaca fascicularis</i>); suppurative peritonitis in a captive gold-handed tamarin (<i>Saguinus midas midas</i>); bronchopneumonia and bacteremia in a free-ranging golden-headed lion tamarin (<i>Leontopithecus chrysomelas</i>); peracute mortality and meningitis in captive lemurs (<i>Varecia variegata</i>); hepatic and abdominal abscessation in captive African green monkeys (<i>Chlorocebus aethiops sabaesus</i>). Mortality with multisystemic abscessation African green monkeys (<i>Chlorocebus aethiops sabaesus</i>) noted following experimental infection with a hypervirulent <i>K. pneumoniae</i> .
Post mortem, gross, or histologic findings: Necropsy findings include abscessation of liver, lung, abdomen, or other organs, septicemia, thoracic or abdominal effusions, or other suppurative changes. Gram-negative bacilli with a prominent capsule.
Diagnosis: Bacterial culture, PCR, IHC. Culture should include sensitivity testing due to variable antibiotic susceptibility patterns. Hyperviscous <i>K. pneumoniae</i> produces abundant capsular material on blood agar, such that a mucoid string > 5mm is lifted off the agar plate (string test). Phenotyping and molecular characterization for hypervirulent and multidrug resistant strains recommended.
Material required for laboratory analysis: Blood, exudate, tissue, feces, sputum, urine, CSF.
Relevant diagnostic laboratories: Routine microbiology laboratories for culture. Genotyping recommended in epizootics.
Treatment: Empirical antibiotic treatment with drugs exhibiting a Gram-negative spectrum of activity, modified based on susceptibility testing. Resistant strains are emerging; resistance to cephalosporins and monobactams through extended-spectrum β -lactamases and resistance to almost all β -lactams (including carbapenems) through carbapenemases. Hypervirulent strains may also complicate treatment and success rates.
Prevention and control: Good sanitation and biosecurity. Fly and cockroach control. Appropriate sterilization or disinfection of medical equipment. Appropriate food handling and commissary disinfection protocols.
Suggested disinfectant for housing facilities: No special requirements other than good hygiene practices, biosecurity, and disinfection protocols.
Notification: No special requirements
Measures required under the Animal Disease Surveillance Plan: None
Measures required for introducing animals to infected animal: No special requirements; animals should be free from clinical illness and re-evaluation with culture diagnostics as appropriate
Conditions for restoring disease-free status after an outbreak: No special requirements
Experts who may be consulted: None.
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KYASANUR FOREST DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals: primarily, rats, squirrels, porcupine and bats, shrews (<i>Suncus murinus</i>) are reservoir hosts. Monkeys are amplification hosts- gray langurs (<i>Semnopithecus</i> sp.) and red-faced bonnet macaques (<i>Macaca radiata</i>) Domestic cattle, sheep, and goats can be affected.	Vector: Hard ticks, primarily nymphal stages of <i>Haemaphysalis spinigera</i> . Other <i>Haemaphysalis</i> sp. and <i>Ixodes</i> sp. soft ticks of <i>Ornithodoros</i> sp. and <i>Argas</i> sp. Direct contact with an infected animal (rodent, monkey)	Biphasic: fever, tussis, dehydration, encephalitis, epistaxis, diarrhea, shock, death	Mild to fatal	No specific treatment. Supportive care especially for treatment of dehydration and hemorrhage	Vector control, including insect repellents and protective clothing	Yes, with mortality for humans living in enzootic areas.

Fact Sheet compiled by: Owen Slater

Sheet completed on: 17 August 2013; updated 12 November 2012 and 24 January 2018

Fact Sheet Reviewed by: Douglas P. Whiteside

Susceptible animal groups: Mammals: Gray langur, red-faced bonnet macaque, domestic cattle, sheep and goats, and humans. Hosts include white-tailed rat, white-bellied rat, shrew, and bats.

Causative organism: Kyasanur forest disease virus (KFDV) virus (Flaviviridae)

Zoonotic potential: Yes and can be fatal in humans

Distribution: Enzootic in Karnataka, Kerala, Maharashtra and Goa States, India, but also human seropositivity in Andaman and Nicobar islands. Debate exists as to whether viruses identified in Saudi Arabia and the People's Republic of China are KFDV or closely related viruses.

Incubation period: In humans, this period is approximately 3-8 days.

Clinical signs:

Animals: Natural infections of monkeys are commonly associated with substantial mortality and evidence of anal hemorrhage. Other clinical signs noted include epistaxis, diarrhea, encephalitis, shock and death.

Humans: Fever, headache, severe muscle pain, prostration, inflammation of conjunctiva, vesicular eruptions on the soft palate, tussis, vomition, diarrhea, dehydration and bleeding. Decreased platelets, red blood cell and white blood cell counts are noted. Patients sometimes recover after 1-2 weeks but usually a biphasic illness with a second wave at three weeks with above clinical signs and, for some, encephalitis. Fatality rate of 3-10%.

Post mortem, gross, or histologic findings: Gross: Anal hemorrhage, epistaxis, and diarrhea. Histologic: Focal liver necrosis with cytoplasmic inclusion bodies, sloughing of tubular epithelium in kidney (humans), small and large intestinal necrosis, pallor of the adrenal cortex, multi-organ hemorrhage (lung, kidney, brain, adrenal), non-purulent encephalitis with focal microgliosis and perivascular cuffing.

Diagnosis: Serology (Convalescent phase), RT-PCR, qRT-PCR, IgM capture ELISA

Material required for laboratory analysis: Serum, whole blood, tissue

Relevant diagnostic laboratories:

Centers for Disease Control and Prevention
1600 Clifton Rd.
Atlanta, GA 30329

Treatment: No specific treatment for the disease. Supportive care for dehydration and hemorrhage.

KYASANUR FOREST DISEASE

Prevention and control: No vaccine currently available in North America but high success with formalin inactivated virus vaccine was been reported for humans in India. Vector (tick and rodent) control in endemic areas is important. Level 3-4 biosecurity protocols in North America.

Suggested disinfectant for housing facilities: 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde. Virus does not survive freezing

Notification: This is not a notifiable foreign animal disease

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: If importing monkeys, rodents or bats from enzootic areas follow normal quarantine measures and strongly consider serologic testing for Kyasanur forest disease virus.

Conditions for restoring disease-free status after an outbreak: None

Experts who may be consulted:

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Chief, Molecular Biology Laboratory, Special Pathogens Branch, Division of Viral and Rickettsial Diseases
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LEPROSY

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans, 9-banded armadillos	Unclear Indirect or direct contact Respiratory droplets Consumption of or contact with 9-banded armadillos	Primarily affects the peripheral nerves, skin, upper respiratory tract, eyes, and limbs. Sensory loss in skin, muscle weakness. Long term lack of sensation leads to traumatic injury and potential loss of use in hands and feet.	Severity of clinical signs based on immunity of host. Left untreated, it may result in permanent damage to skin, nerves, eyes, limbs.	Multi-drug antibiotic therapy.	Humans treated early in course of disease are no longer infective. Avoid exposure to 9 - banded armadillos. Cleaning and eating their carcasses may pose increased risk.	Yes

Fact Sheet compiled by: Lara M. Cusack

Sheet completed on: 25 May 2011; updated 4 September 2012

Fact Sheet Reviewed by: Richard W. Truman, David M. Scollard

Susceptible animal groups: Humans, 9-banded armadillos (*Dasypus novemcinctus*). Other armadillo species such as 6-banded armadillos (*Euphractus*) common as exotic pets, and 3-banded armadillos (*Tolypeutes*) are not known to be susceptible to *M. leprae*.

Causative organism: *Mycobacterium leprae*

Zoonotic potential: Infectious between people and from 9-banded armadillos

Distribution: Organism is found worldwide. Persons in close contact with patients with untreated, active, predominantly multibacillary disease, and persons living in countries with highly endemic disease have higher risk of disease. Most (75%) of cases originate from Angola, Brazil, Central African Republic, Democratic Republic of Congo, India, Madagascar, Mozambique, Nepal and the United Republic of Tanzania. In the US, cases are documented primarily in Louisiana, Texas, California, New York, Massachusetts, and Hawaii. Infections among wild 9-banded armadillos reported in Alabama, Arkansas, Louisiana, Mississippi and Texas, as well as in Argentina, Brazil, Colombia, and Mexico.

Incubation period: While typical incubation period is approximately 5 years, it can be up to 20 years for clinical signs to appear.

Clinical signs:

Humans: Majority of healthy individuals will not develop disease. Susceptibility to infection appears to be genetic. The form of the disease developed depends on host immunity.
Indeterminate form - Earliest clinically detectable form of leprosy found in 10% to 20% of infected people. Hypopigmented macules, without developed tuberculoid or lepromatous characteristics, are present.
Tuberculoid leprosy (pauci-bacillary leprosy) - Single or few well demarcated hypopigmented skin lesions, frequently with active, spreading edges and a clearing center, are noted. Peripheral nerve swelling or thickening also may occur. Acid fast bacilli rare or not visible.

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Lepromatous leprosy (multi-bacillary leprosy) - Very numerous symmetrically distributed erythematous skin lesions, nodules, plaques, thickened dermis, and involvement of the nasal mucosa (congestion, nose bleeds). Acid fast bacilli are always present and may be found in dermal nerves. High titer of antibodies to *M. leprae* but little cellular immune response to the bacillus. Changes in immunity and/or treatment can lead to worsening of clinical signs.

Borderline - Few or several, asymmetrical, hypopigmented, erythematous or coppery skin lesions that are usually positive for acid fast bacilli. These cases may be further sub-divided according to the number and cellularity of the lesions. Borderline Tuberculoid (BT) are usually well demarcated, somewhat dry, and few in number. Borderline Lepromatous (BL) have many roughly symmetrical, shiny macules, nodules, or plaques with sloping or poorly defined edges.

All forms will involve some degree of peripheral neurological damage, leading to sensory loss in skin and muscle weakness. In long term cases, lack of sensation leads to repeated traumatic injury and potential loss of use in hands and feet. Left untreated, may result in permanent damage to skin, nerves, eyes, and limbs.

9-Banded Armadillos: Cutaneous lesions are discerned only in the late stages. One may observe repeated foot ulcers or scrapes around the nose, eyes or legs that do not respond well to normal therapies. Armadillos generally manifest a diffuse lepromatous form of the disease with systemic involvement of reticuloendothelial tissues. Impression smears or swabs of skin lesions can reveal acid fast bacilli or may PCR as *M. leprae*. Leprous armadillos have been reported to show an increase in basal metabolic rate. With one of the lowest metabolic rates of any placental mammal, the cost of infection may represent an important impact but studies to date are undecided as to ecological consequences in wild population. It does not appear to infect young animals which may be due to incubation period.

Post mortem, gross, or histology findings: *Mycobacterium leprae* is an obligate intracellular, acid-fast, Gram-positive bacillus with an affinity for macrophages and Schwann cells. Interaction with Schwann cells induces demyelination and stimulates a chronic inflammatory reaction. Swelling occurs in the perineurium, leading to ischemia, fibrosis, and axonal loss. Sensory fibers are affected prior to motor nerve involvement and the induced insensitivity can contribute to secondary trauma.

Infection in the armadillo is characterized by an insidious microcytic hypochromic anemia, with elevated LDH, ALT, and AST. On gross exam, the liver, spleen, and lymph nodes may be enlarged extensively, they may have a granular texture, and can contain massive numbers of acid fast bacilli. In late stages of disease, no organ system is spared and large numbers of bacilli can be found in all tissues.

Diagnosis: Clinical signs - Localized skin lesions have demonstrated sensory loss, thickened and enlarged peripheral nerves. Acid-fast bacilli in skin or dermal nerve, obtained from the full-thickness skin biopsy of a lepromatous lesion, can be demonstrated. In many cases, rod-shaped, red-stained leprosy bacilli, which are diagnostic of the disease, may be seen in the smears taken from affected skin when examined under a microscope after appropriate staining (weakly acid-fast; Fite stain better than Ziehl-Neelsen). Serology and PCR - not widely performed, fail to reliably detect early/mild forms of the disease.

9-Banded Armadillos: Ear notches- preserve in 100% ethanol for genetic screening and in 70% ethanol for (PCR) analyses to detect *M. leprae* DNA. Serum or eluted whole blood- ELISA test or immunoglobulin M (IgM) antibodies to *M. leprae*. Confirmation made with PCR.

Material required for laboratory analysis: Skin, blood, affected tissues (spleen, liver, lymph node), dermal swabs, and impression smears.

Relevant diagnostic laboratories: Any capable of performing acid fast stain or PCR for *M. leprae*.

Treatment: Hansen's disease is a mild disease when treated early and prior to sensory impairment. Multidrug therapy (MDT) with dapsone, rifampicin, and clofazimine, is daily treatment and prolonged - multi-bacillary cases treated for 2 years, pauci-bacillary cases treated for 1 year.

Drugs provided free of charge by the National Hansen's Disease Programs (NHDP) 1-800-642-2477,

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http://www.hrsa.gov/hansensdisease/clinicalcenter.html .
Prevention and control: Early treatment for atypical skin rashes refractory to treatment should be sought, especially if sensory involvement. Patients are no longer infective after two weeks of MDT (WHO/NHDP). Avoid exposure to/contact with blood or flesh of 9-banded armadillos.
Suggested disinfectant for housing facilities: Organism loses infectivity after 30 min exposure to most disinfectants and UV light. Disinfectants effective against <i>Mycobacterium tuberculosis</i> are likely also effective against <i>M. leprae</i> .
Notification: Nationally, it is a Notifiable Disease (CDC).
Measures required under the Animal Disease Surveillance Plan: Currently none.
Measures required for introducing animals to infected animal: Maintain infected animal in a quarantine situation until treatment initiated.
Conditions for restoring disease-free status after an outbreak: Disinfection of infected environment. Minimize contact with infected persons until treatment is initiated.
<p>Experts who may be consulted:</p> <p>Dr. Richard W Truman, PhD National Hansen's Disease Program LSU School of Veterinary Medicine Tel 225-578-9848, Fax 225-578-9856 rtruman@hrsa.gov</p> <p>Dr. David Scollard, MD, PhD Chief, Clinical Branch National Hansen's Disease Programs Tel 225-756-3713, Fax 225-756-3819 dscollard@hrsa.gov</p>
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LEPTOSPIROSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Fatal Disease	Treatment	Prevention and Control	Zoonotic
Mammals	Contact with urine of shedding host-adapted/ carrier animal or urine-contaminated water; organism can penetrate macerated or wounded skin and intact mucous membranes; potential, but limited, transmission transplacental, transmammary	None or modest in host-adapted/ carrier animals; inapparent to severe in acute infections in non-host adapted animals. Renal signs most typical and include acute renal failure; up to 20% of cases present concurrent hepatitis.	Fatal disease can occur in non-host adapted species.	Antibiotics – usually doxycycline.	Personal hygiene, especially handwashing, and prevention of contact with host-adapted/ carrier animal urine; control of free-ranging wildlife and pests which are often these host-adapted carriers.	Yes

Fact Sheet compiled by: Kathryn C. Gamble

Original date: 12 March 2011; updated 14 July 2013; updated 11 February 2018

Fact Sheet Reviewed by: Kenneth Harkin; June Olds

Susceptible animal groups: Mammals; recent literature assessment published that 10-20% prevalence had been reported in most mammalian families, although *Muridae*, *Canidae*, and *Bovidae* were over-represented; *Felidae* appear more resilient, but recent assessments are that domestic felids are detected more often with sub-clinical disease than recognized previously, and personal author experience with clinical disease in two large exotic felids; reservoir situations, increasing contact with humans through urbanization and conversion to an omnivorous diet is associated with increased prevalence for some taxa, such as *Phalangeridae* (brush-tail possum). Additional reservoirs, including birds and reptiles, have been identified.

Causative organism: *Leptospira* spp. (250± serovars) are spirochaete bacteria which share a common lipopolysaccharide antigen but differ by surface agglutinating antibodies that allows classification. Currently, some of the most common pathogenic leptospiral serovars for U.S. mammals are identified as (*L. kirshneri*) Grippotyphosa, and (*L. interrogans*) Pomona, Bratislava, Hardjo, Icterohemorrhagicae, and Autumnalis.

Zoonotic potential: Infectious to people from animals; though generally comes from a common point source (i.e., rodents, contaminated water) when both animal and human are involved.

Distribution: Worldwide distribution with moist environments most conducive, especially prevalent in tropical countries; occupational and leisure activity risk factors; autumn seasonality observed.

Incubation period: 7-14 days, up to 21 days

Clinical signs: Common reservoir species can have high prevalence of infection – up to 50%. Generally, these individuals do not develop disease or clinical signs, except perhaps mild signs at initial infection. Fatality would not be expected. These animals may shed the organism for a few weeks or intermittently for several years due to chronic infection of the renal tissue. Each serovar tends to have certain host associations as potential natural reservoirs; wildlife and rodents are often implicated in this role during outbreaks.

Acute infections can occur in susceptible species and include most captive zoological species and humans; following infection with the organism, they become ill, moderately to severely. Fatality can occur, especially in untreated individuals. These animals generally do not become carriers. Once the infection has been resolved, especially if these animals are treated, prolonged shedding likely does not occur, although chronic renal damage may be incurred in survivors. Essentially any serovar could infect these individuals and produce disease.

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Endothelial damage is primary source of clinical signs. These signs are non-specific, and many infected animals do not become clinically, or severely, ill. The first signs in humans appear as mild to moderate flu-like with fever, anorexia, malaise and fatigue. Rash may be present but is inconsistent. Other clinical signs are much more severe and related to systemic infection with signs of acute renal disease, including the non-specific, but consistent, clinical signs of infection in the kidneys. Concurrent clinical pathology changes of elevated BUN and creatine, and hyperphosphatemia are present and may be accompanied by hemoglobinuria due to vasculitis. Some infected animals (10-20%) progress to concurrent hepatic disease (Weil's disease) with icterus and increasing hepatocellular enzymes. Pregnant animals may abort. The initial signs may wane with the more serious signs appearing in a biphasic time frame.

As specific taxon focus, equids tend to present with recurrent uveitis rather than renal or hepatic disease; however, reports of acute pulmonary distress as a result of leptospirosis has been reported in foals. Recent studies have also detected leptospiral DNA in vaginal swabs of mares, suggesting potential venereal transmission. Although original association of this organism with black rhinoceros (*Diceros bicornis*) and hemolytic anemia was considered, it has not been proven. Free-ranging California sea lions (*Zalophus californianus*) have a marked predisposition to infection with serovar Pomona with severe renal disease; limited other serovars have been identified in other pinnipeds, but not in cetaceans.

Post mortem findings: These findings are specific to the body system infected and presenting clinical signs at time of illness. Usually, it is evidence of acute renal failure. Acute hepatitis is observed in those animals which had icterus. Scarring ("white spots") in affected organs in chronic cases observed macroscopically in the kidneys of pigs and dogs.

Diagnosis: Diagnosis is challenging and treatment must begin before diagnosis is conclusive. In the literature since the last review, increasing effort to find faster or more point-of-care options was noted. Although direct observation with (silver or fluorescent antibodies (FA)) or without (darkfield microscopy) stain enhancement has been reported as useful, leptospire must be present in sufficient numbers in the sample evaluated, usually urine. The defined gold standard of testing is serologic evaluation by microscopic agglutination testing (MAT) but this testing modality is specific and requires maintenance of the organism with its markedly fastidious culture needs, and it cannot differentiate between vaccine and natural antibody production. However, MAT testing is readily available. A positive status is assigned to a test result >1:100 in an unvaccinated animal, but this low seroconversion requires a four-fold rise in titer over 2-4 weeks for diagnostic support. In a clinically ill animal, a single serologic status of 1:800 is strongly suggestive of leptospirosis. Cross-reactivity is quite common so a panel of likely serovars are assessed, assigning the serovar with the highest titer as the most likely causative agent. Polymerase chain reaction (PCR) of urine is now available which detects specific gene unique to pathogenic serovars. New canine specific tests include indirect ELISA and a commercial lateral flow assay.

Material required for laboratory analysis: Serum is submitted for most testing, but urine can be submitted for PCR. Whole blood and serum can be submitted for PCR or whole blood for culture. Post-mortem tissues – ideally kidney - can be submitted for histology using special silver stains, culture, PCR, or FA. Due to the fastidious nature of leptospira, cultures are often unrewarding, and additional diagnostic methodologies are recommended for confirmatory diagnosis.

Relevant diagnostic laboratories: Leptospire MAT is offered by many commercial and state diagnostic laboratories; Michigan State University Diagnostic Laboratory has an excellent serology panel and consultation services available. PCR testing now is offered routinely by many laboratories, the LipL32 based and 23s rRNA-based PCR have been shown to have false positives from free-catch urine samples.

Treatment: These organisms are generally quite sensitive to most antibiotics, except notably chloramphenicol. First generation cephalosporins (specifically cephalothin) historically were considered less successful for treatment but recently these (specifically cefazolin and cephalexin) have been suggested as effective. Best success occurs when the treatment is initiated promptly and as early in the disease course as possible. Doxycycline for 14 days is most commonly used successfully to treat clinical signs. Supportive care for systemic signs may be needed in more severe cases.

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Prevention and control in zoos: Although vaccines as killed whole cell bacterins are available for pigs, cattle, and dogs, it would be necessary to specifically target the serovar of concern in the particular area. It may therefore be preferred to leave this option to consideration in outbreak control or in areas with higher risk or increased urban wildlife or domestic stray interactions. Serologic testing can be monitored in these situations and during transfers between facilities. More importantly, pest control and exclusion of other carriers from contact with collection animals would be important.

Once an animal is confirmed infected, prompt treatment will minimize or may eliminate shedding. In the treatment interval, appropriate staff protection and personal hygiene is to be utilized to prevent spread within the facility or to staff. Consideration of drainage of the area should be made in this control measure. If the situation were to occur in a contact program area, it is recommended to exclude guests until the situation is treated, and leptospirosis is confirmed resolved.

Suggested disinfectant for housing facilities: Any standard disinfectant technique would be appropriate for cleaning of this organism.

Notification: In the US, Hawaii is the only state currently maintaining this disease as reportable in animals. Centers for Disease Control and local health authorities should be alerted for human cases, especially clusters. USDA apprises WHO of leptospirosis issues in certain production species.

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Infected animals should be maintained as isolated as possible from other mammals until treatment interval is completed. PCR testing on urine would be helpful to confirm that the infected animal was no longer shedding. Serologic monitoring of animals in adjacent areas would be considered prudent.

Conditions for restoring disease-free status after an outbreak: Serologic monitoring of adjacent areas would be considered prudent following return of infected animal to collection to assess for exposure.

Experts who may be consulted:

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LISTERIOSIS (*Listeria monocytogenes*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All	Fecal-oral ingestion, inhalation, direct contact with affected tissues, or indirectly through contaminated milk, cheese, meat, eggs, fruits or vegetables in people. Common route in animals is hay contamination or unstable silage.	Three primary forms: encephalitic, abortion / perinatal mortality, septicemia but can see ophthalmic form. Primarily winter-spring disease in US	Without aggressive treatment, encephalitis and septicemic disease are often fatal or animals recover with permanent brain damage (encephalitic form).	Aggressive and early treatment with antibiotics, supportive care. Recovery rate often ~30%.	Remove spoiled feed or silage. Separate affected animals. Good hygiene practices.	YES. At risk groups are pregnant women, neonates, elderly, immune-compromised and those handling infected tissues.

Fact Sheet compiled by: Diana Boon, DVM

Sheet completed on: 1 December 2010, updated 21 August 2013, updated 24 Sept 2017

Fact Sheet Reviewed by: Clayton Hilton, MS, DVM

Susceptible animal groups: Mammals and birds. *Listeria* has been isolated from fish, crustaceans and insects, but these species are likely carriers. Can be cultured from healthy, asymptomatic animals & humans.

Causative organism: *Listeria monocytogenes* (gram-positive bacilli)

Zoonotic potential: Zoonotic potential exists when handling aborted tissues or removing infected brain during necropsy. Food-borne illness most common and at-risk groups are pregnant, elderly, and immune-compromised people.

Distribution: Worldwide

Incubation period: Approximately 10 days – 3 weeks, but clinical signs have been within 5 hours of exposure in poultry.

Clinical signs: Several forms are possible:

Encephalitic form (adult ruminants): a.k.a. “Circling Disease”, early signs of depression, anorexia, disorientation, decreased milk production, fever which can progress to seizures, unilateral trigeminal and facial nerve paralysis, circling, cerebellar signs [ear droop, deviated muzzle, flaccid lip, lowered eyelid on affected side(s)], salivation, deviated muzzle, flaccid lip, and death.

Abortion (adult ruminants) fever, hypo- to anorexia, late-term abortions & stillbirths. Retained placentas with secondary metritis.

Septicemic form (typically neonates and monogastrics): diarrhea, focal hepatic necrosis, death.

Septicemic form (poultry-rare): lethargy, depressed mentation, diarrhea, myocardial or hepatic necrosis, death.

Ophthalmic form: secondary to nerve damage [decreased tear production secondary to changes in special visual efferent (SVE) system in medulla, eyelid paralysis with secondary exposure keratitis] or direct contact (keratoconjunctivitis, retinal changes).

Post-mortem, gross, or histologic findings:

Encephalitic form: few gross lesions (some congestion of meninges), histologic lesions consistent with encephalitis or meningoencephalitis with micro-abscessation and organisms present (predilection for pons, medulla, brain stem and cranial spinal cord).

Septicemic form: evidence of sepsis, +/- focal hepatic necrosis and hemorrhagic gastroenteritis

Abortion form: third trimester abortion common, gross lesions are placentitis (most severe lesion), metritis, and subtle fetal infection. Histologic lesions are suppurative and necrotizing placentitis and small necrotic foci in any fetal organ, especially liver, with fetal necrotizing colitis not common but very supportive of listeriosis. Gram-stain and culture of abomasal contents may be positive for bacteria. Maceration of the fetus can occur

LISTERIOSIS (*Listeria monocytogenes*)

with retained abortions. The herd and dam generally do not present with the encephalitic or septicemic forms concurrent with the abortion form.

Fetal lesions: slight to marked autolysis, fluid in serous cavities, small necrotic hepatic foci (often in right half), erosion in abomasal mucosa. Complete maceration of fetus is common.

Diagnosis:

Pre-mortem - Clinical signs, CSF tap for cytology and culture. CSF will have increased protein concentration (0.6-2.0g/L) with mild pleocytosis composed of large mononuclear cells.

Post-mortem – culture of affected tissues* (very suggestive if grows at 4°C), IFA affected tissues* gram-stain (gram-positive pleomorphic bacteria, not always diphtheroid coccobacillus), immunohistochemistry of brain tissues.

*Affected tissues include brain (pons & medulla), nasal discharge, placenta & associated fluids, abortus, urine, feces, milk, meat, silage, and other sources.

Serology not routinely due to low specificity.

DDx: Trichomoniasis, pregnancy toxemia (ewes), ketosis (cattle), BSE, histophilosis, poliоencephalomalacia, sporadic bovine encephalitis, lead poisoning, rabies, brain abscess (cestode).

Material required for laboratory analysis: Aborted fetuses and placentas; brain can be submitted and request cold enrichment method for culture. ***Wear gloves and protective clothing when handling tissues.***

Relevant diagnostic laboratories: As the diagnosis is mostly post-mortem, all diagnostic labs should be able to perform testing. Remember to request *Listeria* culture (cold enrichment method*) if initial culture results do not correspond to clinical presentation. *may take 3 months to get results

Treatment: Dependent on prompt diagnosis at early stage of disease as death can occur within 24-48 hours of onset of clinical signs. High doses of penicillin (first choice), oxytetracycline, ceftiofur, erythromycin (not in dairy cattle), trimethoprim/sulfonamide. Supportive care for clinical signs is needed.

Prevention and control: Reduce fecal contamination of feed and monitor sewage contamination. Discard spoiled feed and hay. Improve sanitation of pens, water supply, pasture, food refrigerators, and housing facilities. Limit access of wild birds (as possible vector for bacteria) if possible. Isolate aborting females. Pasteurization of milk for human consumption or bottle feeding, but may not be 100% effective. *Listeria* vaccine developed for oncology patients.

Suggested disinfectant for housing facilities: No specific disinfectants suggested but good hygiene standards should be maintained, including rodent control.

Notification: Reportable to local health authorities within a few days of disease confirmation - see specifics for each state.

Measures required under the Animal Disease Surveillance Plan: None defined. Most measures are currently for prevention of *Listeria* introduction into the human food supply.

Measures required for introducing animals to infected animal: Organism can be shed intermittently in milk (without signs of mastitis), feces, and vaginal secretions for >1 month but no recommended measures for reintroduction.

Conditions for restoring disease-free status after an outbreak: Segregate affected animals, remove affected silage or feed. Disease may continue to be sporadic as is found in the soils. Organism tends to display a seasonal pattern (February – April) of infection.

Experts who may be consulted:

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LUMPY SKIN DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primarily cattle, but also African ruminants	Mainly via biting insects, but less frequently direct contact with infected animal. Virus may be present in semen of infected bulls for extended periods of time.	None to severe: fever, skin and internal nodules, enlarged lymph nodes, anorexia, rhinitis, conjunctivitis, brisket and leg edema	Typically, it is mild but may be severe. Most animals slowly recover but may take months. Morbidity can be 1 - 95% within a herd. Mortality rate usually low, but may be up to 25%.	Subject to regulatory approval as infected animals outside endemic area may be destroyed. Supportive care as virus runs its course. Appropriate antibiotics for secondary bacterial infections	Import restrictions (mainly shipments from Africa); proper quarantine and testing of animals imported from endemic areas. Live and attenuated vaccines exist in endemic areas	No

Fact Sheet compiled by: Jackie Gai

Sheet completed on: 25 January 2011; updated 1 April 2013

Fact Sheet Reviewed by: Carlos Romero, Anna Rovid Spickler

Susceptible animal groups: Cattle (*Bos taurus*). European breeds (Jersey, Guernsey, Ayrshire, Holstein, etc) of thin skin are more susceptible than zebu cattle (*Bos indicus*). A few cases have been reported in Asian water buffalo. Suspected clinical disease has been reported in Arabian oryx (*Oryx leucocoryx*) in Saudi Arabia, springbok (*Antidorcas marsupialis*) in Namibia, and oryx (*Oryx gazelle*) in South Africa. Wildlife probably not important in the epidemiology of the disease. Antibodies have been found in 6 out of 44 wildlife species tested in Africa: African buffalo (*Syncerus caffer*), greater kudu (*Tragelaphus strepsiceros*), waterbuck (*Kobus ellipsiprymnus*), reedbuck (*Redunca arundinum*), impala, springbok, and giraffe, although these may have been due to cross-reaction to similar Capripoxviral exposure. Experimental infection has been induced in sheep and goats.

Causative organism: Lumpy skin disease virus (LSDV) of cattle is classified within the genus *Capripoxvirus*, subfamily *Chordopoxvirinae*, family *Poxviridae*. The virus is morphologically identical to the other two known capripoxviruses; sheeppox and goatpox viruses, to which it is highly antigenically related.

Zoonotic potential: None. No evidence exists that capripoxviruses are transmitted to humans.

Distribution: Originally described in 1929 in sub-Saharan Africa and Madagascar, over the last 70 years it has spread north and south of this region. Recent outbreaks have occurred in Egypt (1988, 2006), Mauritius (2008), Vietnam, and Lebanon and Israel (2012).

Incubation period: Thought to be two to five weeks under natural conditions, but this is an estimate. Experimentally infected animals developed fever within 6-9 days and skin lesions at the inoculation site in

LUMPY SKIN DISEASE

4-20 days. However, not all experimentally infected cattle develop clinical signs or skin lesions, indicating that there are other unknown factors involved in the pathogenesis of the disease.

Clinical signs: Signs may range from inapparent to severe clinical disease. Pyrexia followed by the development of multiple, painful nodules 2-5 cm in diameter over entire body, especially on head, neck, udder, perineum, and legs. Nodules involve the full thickness of the skin and may initially exude serum, developing into necrotic plugs. Rhinitis, conjunctivitis, and hypersalivation may be seen. Agalactia or marked reduction in milk yield may occur. Generalized lymphadenopathy and limb edema that makes animals reluctant to move. Pox lesions may develop on mucous membranes of mouth. Pregnant cattle may abort, and aborted fetuses may have skin nodules. Anorexia and emaciation.

Post mortem, gross, or histologic findings: Greyish pink skin nodules may turn into conical, necrotic plugs which penetrate the full thickness of the hide ("sit-fasts"). Flat or ulcerative lesions may be found in mucous membranes of the oral and nasal cavities, epiglottis, and trachea. Nodules may also be found in the gastrointestinal tract (especially abomasum), udder, urinary bladder, lungs, kidneys, and reproductive organs. Pleuritis and enlargement of mediastinal lymph nodes in severe cases. Enlargement of lymph nodes that drain affected areas, with lymphoid proliferation, edema, congestion, and hemorrhage. Synovitis or tendosynovitis with fibrin in synovial fluid. Temporary or permanent sterility may occur in bulls and cows.

Diagnosis: Confirmation of LSD in a new area requires virus isolation and identification. Biopsy or scrapes of skin lesions and nasal and oral swabs are the most useful samples for virus isolation and rapid identification by PCR and sequencing or by staining of infected cell cultures with specific labeled antiserum. Intracytoplasmic inclusion bodies can be seen on electron microscopy. The gold standard for detecting specific antibodies to capripoxviruses is the virus neutralization test.

Material required for laboratory analysis: Scrapings and biopsies of skin lesions and nasal, pharyngeal and conjunctival swabs. Lymph node biopsies may be useful when there is generalized adenopathy.

Relevant diagnostic laboratories:

Within the US:

Foreign Animal Disease Diagnostic Laboratory
USDA-APHIS
40550 Route 25
Orient, NY 11957
631-323-3256

International shipments:

USDA
Attn: FADDL Lab Director
c/o Port Veterinarian, APHIS VS
230-59 Rockaway Blvd #101
Jamaica, NY 11413
718-553-1727

Outside the US:

Institute for Animal Health
Pirbright Laboratory
Ash Road
Woking, Surrey
GU24 0NF
Great Britain

LUMPY SKIN DISEASE

<p>Treatment: Subject to regulatory approval as infected animals outside endemic area may be destroyed. No specific treatment, provide supportive care. Antibiotics such as sulfonamides to prevent or control secondary infection</p>
<p>Prevention and control: Stringent import restrictions on livestock, carcasses, hides, and semen are in place. When importing animals from endemic countries, adhere to regulatory pre-shipment and quarantine requirements. Report all suspected cases to the appropriate regulatory agency in your area immediately. The disease is mainly transmitted mechanically by biting arthropods, contrary to sheep pox and goat pox that are mainly transmitted by direct contact with infected animals. The infection may also be transmitted by contaminated semen. Animals that recover from the natural disease are immune for life. Live-attenuated sheep/goat pox vaccine (Kenya SGPV strain), as well as South African LSD live vaccine (Neethling strain) are used in cattle in endemic countries.</p>
<p>Suggested disinfectant for housing facilities: Sodium hypochlorite (2-3%), iodine compounds (1:33 dilution), Virkon® (2%), quaternary ammonium compounds.</p>
<p>Notification: Lumpy Skin Disease is a reportable disease which must be reported immediately to the appropriate regulatory body, i.e. Department of Food and Agriculture, USDA-APHIS or State Veterinarian.</p>
<p>Measures required under the Animal Disease Surveillance Plan: Currently none.</p>
<p>Measures required for introducing animals to infected areas: None – this is a reportable disease and animals with confirmed infection outside of endemic area will be destroyed.</p>
<p>Conditions for restoring disease-free status after an outbreak: To be determined by governmental authority. Life-long immunity occurs in recovering individuals. Buffalo may serve as viral reservoirs.</p>
<p>Experts who may be consulted: Douglas Gregg, DVM, PhD Foreign Animal Disease Diagnostic Laboratory NVSL, APHIS, USDA Greenport, NY 11944 USA</p> <p>Dr. Eeva Tuppurainen Institute for Animal Health, Pirbright Laboratory Ash Road, Pirbright, Woking, Surrey GU24 0NF UNITED KINGDOM (44.1483) 23.24.41 Fax: (44.1483) 23.24.48 eeva.tuppurainen@bbsrc.ac.uk</p> <p>Dr. Baratang Alison Lubisi Onderstepoort Veterinary Institute, Agricultural Research Council Private Bag X5, Onderstepoort 0110 SOUTH AFRICA Tel: (27.12) 529.92.33 Fax: (27.12) 529.94.18 Lubisia@arc.agric.za</p>
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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Marine Mammals Birds Snakes Nonhuman primates Canids	Direct	In many cases, animals are asymptomatic; however, in severe infections, they may show signs of upper or lower respiratory disease depending on the host and species of parasite involved.	Dependent on the intensity of infection. Mortality is low, but can be higher if bacterial or fungal co-infection develops.	Ivermectin	Reduce population density, hand rear young.	No

Fact Sheet compiled by: Sara Childs-Sanford

Sheet completed on: updated 2017

Fact Sheet Reviewed by: Rich Sim

Susceptible animal groups:

Marine Mammals: Pinnipeds (phocid seals, otariids, walrus), sea otters.

Birds: Numerous species, including companion passerines (especially exotic finches), wild passerines, and galliformes.

Snakes: reported in *Elaphe schrencki* (Russia), *Crotalus* and *Pituophis* spp. (southern United States), *Natrix trigri* (Korea).

Nonhuman Primates: Old World monkeys (esp. *Macaca mulatta*), apes.

Canids: reported in a fox (Norway) – only documentation in a species other than the domestic dog.

Causative organism:

Pinnipeds:

- Phocid seals: *Halarachne* spp., including *H. halichoeri*.
- Otariids, walrus: *Orthohalarachne* spp., including *O. attenuata* and *O. diminuata*.

Sea otters: (*Halarachne miroungae*).

Birds:

- *Sternostoma tracheacolum*: Captive birds, primarily finches and canaries. Also reported in numerous wild passerine species as well as wild Gouldian finches in Australia following introduction via domestic canaries. Numerous other species of *Sternostoma* have been reported in wild passerines.
- *Cytodites nudus*: pheasants, chickens, turkeys, ruffed grouse, canaries, finches, cockatiels, budgerigars, pigeons.
- Numerous *Ptilonyssus* spp. have been reported in wild passerines in North and South America, and in captive canaries.

Snakes: *Entonyssus* spp., including *E. squamatus*, *E. halli*, *E. koreansis*, *E. vitzthumi*.

Primates: *Pneumonyssus* spp., including *P. simicola*, *P. duttoni*, *P. africanus*.

Canids: *Pneumonyssoides caninum*.

Zoonotic potential: One report describes a case of human ophthalmic acariasis, after getting sneezed on by a walrus, which resulted in ophthalmalgia and corneal abrasion.

Distribution: Worldwide.

Incubation period: Unknown.

Clinical signs:

Marine mammals:

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- Pinnipeds: nasal discharge, sneezing, facial pruritus, head shaking, and, if lung involvement, dyspnea.
- Sea otters: may be predisposed to sinus or turbinate infections.

Birds: wheezing, gasping, open-mouth breathing, head shaking, loss of or change in voice, cessation of singing, dyspnea. Nonspecific signs such as weight loss, weakness, and sudden death may occur.

Snakes: usually asymptomatic.

Primates: usually asymptomatic, but may be predisposed to other pulmonary diseases due to bronchiolar epithelial changes, and sneezing and coughing. In advanced cases (especially in aged or immunocompromised animals) death may occur.

Canids: in domestic dogs, sneezing is common but may also have facial pruritus, excessive lacrimation, and nasal discharge.

Post mortem, gross, or histologic findings:

Marine mammals: histologically, erosion and inflammation of the nasal turbinates and nasopharynx may be seen associated with mites. Sinusitis, rhinitis, bronchopneumonia. *O. attenuata* adults primarily occupy the nasopharynx, while *O. diminuta* are found in the lungs.

Birds:

- *Sternostoma*: Black mites can be found in trachea, air sacs, and lungs. Histologically: tracheitis, air sacculitis, multifocal pneumonia.
- *Cytodites*: Mites can be visualized macroscopically as small white spots within bronchi, lungs, and air sacs. Severe infections may result in granulomatous pneumonia.
- *Ptilonyssus*: Mites within the trachea, with mucosal sloughing, epithelial deciliation and necrosis, and tracheal cartilage degradation.

Primates: Small (1-5mm) pale yellow foci containing mites throughout the lungs. In advanced cases, cavitation of the lungs may be present. Gross lesions may resemble those of tuberculosis. May be a cause of pulmonary bullae. Histologically: presence of macrophages containing brown to black pigment and multifocal eosinophilic granulomatous bronchiolitis, with intralesional arthropods.

Diagnosis: Antemortem diagnosis is difficult.

Marine mammals: identification of larval mites in sputum or nasal exudate, or at necropsy. Rhinoscopy may be useful.

Birds: following wetting of the cervical feathers with alcohol, tracheal illumination may reveal the mites as small black spots within the lumen. Failure to visualize mites with this method does not rule out infection. On necropsy, mites can be identified macroscopically in the tracheal lumen, lungs, or air sacs.

Snakes: lung wash, necropsy.

Primates: tracheobronchial lavage, necropsy. Radiographic lesions may include an interstitial pattern with increased bronchial thickness, pleural thickening, pleural adhesions, and cavitating pulmonary lesions. Pneumothorax is a common complication of pulmonary acariasis and is frequently unilateral.

Canids: nasal swabbing, rhinoscopy, necropsy, use of an antibody ELISA has been reported.

Material required for laboratory analysis: Depending on the species and location of infection: sputum, nasal discharge, lung wash, lung tissue.

Relevant diagnostic laboratories: Any veterinary diagnostic laboratory with a parasitologist on staff.

Treatment: Ivermectin.

Marine mammals: 200µg/kg twice, 2 weeks apart.

Birds: ivermectin or doramectin. Can be given as an injection, or in small birds, can be applied topically on the bare skin at the base of the neck (dilute 1:10 with propylene glycol and apply 1 drop per bird up to 50g, repeat in 7-10 days).

Primates, Canids: 200µg/kg subcutaneously.

Prevention and control: Antemortem diagnosis and prevention are difficult, since infected animals are often asymptomatic and identification of those with a low mite burden is unlikely. Can consider

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prophylactic treatment of newly acquired captive animals during quarantine. High population density facilitates transmission. Animals can be raised free of infection if they are separated from the mother soon after birth and hand-reared.

Suggested disinfectant for housing facilities: Appropriate acaricides (e.g. pyrethroids).

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Infected animals should be treated prior to introduction to disease-free animals.

Conditions for restoring disease-free status after an outbreak: Successful treatment of all potentially exposed susceptible animals.

Experts who may be consulted: While no specific researchers are currently reporting expertise in this parasite, parasitology staffs at veterinary colleges would be a good option.

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**LYMPHOCYTIC CHORIOMENINGITIS VIRUS/
CALLITRICHID HEPATITIS**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
New World nonhuman primates (NHP)- <i>Platyrrhini</i> - of the families Callitrichidae and Callimiconidae; humans and rodents	Horizontal due to ingestion of infected mice with LCMV (including wild rodents) Vertical transmission of LCMV to an aborted nonhuman primate fetus.	Lethargy, jaundice, anorexia, weakness, dyspnea	High fatality rate (morbidity and mortality)	None	Rodent control; avoid feeding primates on mice	Yes

Fact Sheet compiled by: Enrique Yarto-Jaramillo

Sheet completed on: 4 August 2011; updated 4 February 2018

Fact Sheet Reviewed by: Salomé Cabrera; Lilian Silva Catenacci; Rosalia Pastor; Pierre Rollin

Susceptible animal groups: The common house mouse (*Mus musculus*) is the natural host and principal reservoir of LCMV. Several genera of families Callitrichidae, especially *Callithrix* sp., *Saguinus* sp. and Callimiconidae, especially *Callimico goeldii*, are susceptible to the infection with LCMV. In captive golden lion tamarins (*Leontopithecus rosalia*) and pygmy marmoset (*Cebuella pygmaea*), the virus accounted for 43% and 71% of deaths of animals, respectively. Humans and wild, laboratory and pet rodents (especially mice, hamsters, gerbils, rats and guinea pigs) are susceptible. Infections to humans from pet rodents have been reported. Although rodents can potentially become infected, they often do not show any signs of illness. Hamsters are not the natural reservoir so in young hamsters it causes a chronic fatal wasting disease. Infected mice and hamsters have proven to shed the virus in large quantities through their lives in saliva, feces, urine and nasal secretions. Humans may also acquire this virus from nesting materials from infected pet rodents.

Causative organism: Lymphocytic choriomeningitis virus (LCMV) which is a lipid enveloped single-stranded RNA virus (family Arenaviridae, genera Mammarenavirus) of the Old World's Arenavirus group, is considered the prototypic arenavirus. This group of viruses utilize rodents as their principal reservoirs. LCMV is a virus with high mutation rates and important strain variations. Rodent reservoirs pass the virus to their offspring and shed the virus in urine and oral secretions, which are additional routes of transmission to zoo animals. The other route of transmission to zoo animals has been the domestic mice used to feed non-human primates. Animals not eating mice neither became ill nor seroconvert to LCMV even after close contact with sick primates. Thus, direct primate-primate transmission of LCMV was not observed yet, although such a mode of transmission remains a possibility. Vertical transmission of LCMV to an aborted tamarin fetus, however, was demonstrated in a US zoo.

Zoonotic potential: LCMV is a prevalent human pathogen infecting large numbers of humans according to serological studies which indicate that approximately 5% of adult humans in the USA show antibodies to this virus. Seroconversion with no evidence of clinical disease has been reported in handlers of infected animals, although the infection has been reported to cause substantial neurological disease, especially in immunocompromised humans. In humans, the LCMV causes influenza-like clinical signs, occasionally with neurologic complications alike manifestations of aseptic meningitis. Since this virus has a strong

**LYMPHOCYTIC CHORIOMENINGITIS VIRUS/
CALLITRICHID HEPATITIS**

neurotropism, LCMV is recognized as an important cause of neurologic disease in humans. Infection may be asymptomatic in up to one third of patients, although serious complications often occur in intrauterine infection. Less severe cases of adult human infection are likely underreported and often misdiagnosed. It is also a potential emerging zoonotic agent causing congenital defects in children. Several reports of LCMV acquired during pregnancy have demonstrated severe disruption of brain development. In 2009 the Center for Disease Control and Prevention confirmed a case of LCMV-associated congenital hydrocephalus and chorioretinitis in a child from New York. The mother's history referred exposure to mice during pregnancy. LCMV is recognized as a zoonotic disease associated with exposure to infected hamsters and gerbils. Child neurologists should be more familiar with this virus due to its potential to cause severe neurologic birth defects and so to promote its inclusion within the TORCHS acronym.

In April 2012, the CDC was notified about a patient diagnosed with aseptic meningitis who was an employee at a rodent breeding facility in Indiana and whose testing revealed LCMV. Further testing showed evidence of prevailing or past LCMV infection in 13 out of 52 employees at the same facility.

Distribution: LCMV is found worldwide, probably because of its association with its natural Old World's host, the house mouse, *Mus musculus*. Although antibodies have also been detected in other rodent species, arenaviruses are known to be serologically cross-reactive. Outbreaks have been reported in zoo colonies of callitrichid primates in US and Europe (UK and Germany).

Incubation period: In non-human primates, it is from one to three weeks, but deaths which can reach 100% in an outbreak may occur over a period of weeks to months.

Clinical signs: In infected callitrichid primates (marmosets, tamarins, and Goeldi's monkeys), clinical findings are acute onset of lethargy, anorexia, anemia, weakness, fever, dyspnea and mucus-covered feces along with jaundice, and sometimes hemorrhage. It was also reported abortion and dystocia in captive tamarins and marmosets. Animals having a longer course of the disease may present jaundice and inguinal petechiae. Some authors have reported grand mal seizures or sudden death without prior clinical signs. Clinical laboratory findings: elevated levels of aspartate aminotransferase, alkaline phosphatase and bilirubin, but none of them are specific. Serologic evidence of LCMV in marmosets without clinical signs has been documented. In experimentally-infected rhesus macaques, LCVM-WE strain has led to fatal liver disease which was formerly described as Lassa fever (LF) hepatitis.

Post-mortem, gross, or histologic findings: Gross necropsy findings in NHP may include: hepatitis, hepatomegaly, splenomegaly, pleural and pericardial effusions, lymphadenopathy, jaundice, subcutaneous and intramuscular hemorrhages. Histologic findings include multifocal hepatocyte necrosis with infiltration by lymphocytes and neutrophils and portal vein vasculitis, necrosis of spleen, lymph nodes, adrenal cortex and intestinal tract. Acidophilic bodies (Councilman bodies), that represent apoptotic hepatocytes have been observed in affected liver tissues. Brain tissues may show encephalitis, minimal meningitis and vasculitis.

Diagnosis: In NHP clinical signs, clinical findings and husbandry history (exposure to rodent species or history of being fed suckling mice) are consistent with diagnosis. In humans, confirmatory diagnosis is usually by virus isolation in cerebrospinal fluid (CSF); by PCR on tissues or CSF; anti-LCMV IgM and IgG by ELISA in blood, serum, or CSF. In human genetic analysis LCVM strains have demonstrated these viruses are genetically and biologically highly diverse.

Histopathology, virus isolation, electron microscopy, nucleic acid hybridization analysis, immunofluorescence and immunoblot in liver biopsy and other tissues (spleen, lung, adrenal glands, lymph nodes, intestine, kidneys, urinary bladder, heart and brain) are the reported diagnostic methods for LCMV in NHP. In rodents, few isolates of LCMV have been obtained from wild rodents so little is known about its genetic diversity. Confirmatory diagnosis is by viral isolation or PCR, and antibody detection in the blood/serum by ELISA. In recent experimental studies using different types of macrophages and

**LYMPHOCYTIC CHORIOMENINGITIS VIRUS/
CALLITRICHID HEPATITIS**

hepatocytes, it has been validated that AML-12 hepatocytes are useful in studying the mechanisms of arenavirus-induced hepatitis.

Material required for laboratory analysis: Serum for serology, tissue samples (especially liver and brain) frozen at -70°C for PCR or virus isolation. Formalin-fixed tissues for pathology and immunohistochemistry.

Relevant diagnostic laboratories:

Virus Reference Laboratories Inc.
7540 Louis Pasteur Road
San Antonio, Texas 78229
Phone: (210) 614 – 7350
Fax: (210) 614 – 7355

Treatment: No effective treatment known, although supportive therapy with fluids to correct hypovolemia and electrolyte imbalances might be of benefit. The antiviral agent ribavirin has been used in infected primates (150mg/kg, intramuscularly, once daily for 6 days), but all of them were in an advanced stage of the disease and a clinical response was not observed.

Prevention and control: Avoid feeding callitrichid primates on mice (pinkies), and stringent rodent control programs in zoos and primate centers, particularly in areas housing callitrichids. People using frozen or live rodents to feed other animals should follow safety precautions, including wearing gloves when handling animal products. Washing hands with soap and water after handling animal products is warranted. Once an outbreak has been detected the animal enclosure should be cleaned and disinfected. Proper snap traps of rats and mice as well as spraying dead rodents with disinfectants, double bagging the carcasses and waste disposal should reduce the risk for people.

Suggested disinfectant for housing facilities: A 1:10 bleach solution is effective in killing LCMV. Hypochlorite solution: 1 and ½ household bleach: 1 gallon of water is indicated as a disinfectant for contaminated areas.

Notification: Due to some reports on human patients contracting the virus from transplanted organs as well as LCMV-associated congenital defects, LCMV is a reportable disease in three U.S states (Wisconsin, Massachusetts and Arizona) and one city (New York, New York). Increased physician awareness should improve disease recognition and reporting in human patients.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: It appears that most NHP that become clinically infected succumb to the disease. Horizontal transmission has not been reported in people; however vertical transmission can occur.

Conditions for restoring disease-free status after an outbreak: Strict pest control and removal and control of all rodents and their droppings, urine and bedding. Disinfection of all premises with 1:10 bleach solution.

Experts who may be consulted:

CDC – Viral Special Pathogens Branch
404-639-1115 or 404-639-1510
Dvd1spath@cdc.gov
<http://www.cdc.gov/ncezid/dhcpp/vspb/index.html>

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**LYMPHOCYTIC CHORIOMENINGITIS VIRUS/
CALLITRICHID HEPATITIS**

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MARBURG HEMORRHAGIC FEVER

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primates, including humans	Direct contact with body fluids, sexual activity, droplets and aerosolized virus	Fever, rash, malaise, vomiting, diarrhea, shock, multiple organ dysfunction syndrome, hemorrhage	High morbidity and mortality	No specific treatment	Personal protective equipment, strict quarantine	Yes

Fact Sheet compiled by: Aubrey M. Tauer

Sheet completed on: 1 April 2011; 15 August 2013

Fact Sheet Reviewed by: Christine Fiorello; Pierre E. Rollin

Susceptible animal groups: Primates, including humans. African fruit bat (*Rousettus aegyptiacus*) is a natural reservoir for Marburg.

Causative organism: *Lake Victoria Marburgvirus* (Filovirus)

Zoonotic potential: Yes

Distribution: Natural virus circulation and human cases (isolated or during outbreak) are restricted to Africa (geographical range of the reservoir), although imported monkeys caused an human outbreak in Europe in 1967 and several infected travelers have imported the disease outside the endemic zone.

Incubation period: Generally, it is 8-10 days (range 5-21 days).

Clinical signs: Early signs are similar to influenza and malaria, and the onset of disease is sudden.

Humans: Fever, headache, chills, and myalgia, sometimes followed around the fifth day by possible maculopapular rash on the chest, abdomen, and back. As the illness progresses, nausea, vomiting, abdominal pain, diarrhea, chest pain, sore throat, jaundice, weight loss, and pancreatitis are observed. Many patients develop some form of bleeding and often from multiple sites. Shock, renal failure, liver failure, and multiple organ dysfunctions occur in the most severe cases and usually preceded death. Case fatality rate ranges from 23% to more than 80% in recent outbreaks. In recovered humans, complications such as orchitis, recurrent hepatitis, uveitis, transverse myelitis, have been reported.

Animals: Lymphopenia and elevation of liver enzymes are characteristic. Thrombocytopenia is frequent. Although duikers are susceptible to Ebola virus, a related filovirus, little is known about the host range of Marburg virus. Complications in the few recovering animals are not reported.

Post mortem, gross, or histologic findings: In laboratory non-human primates, maculopapular rash; pulmonary congestion and edema; enlarged friable fatty liver; enlarged, congested and/or hemorrhagic lymphoid tissue; pericardial effusion; pyloric and duodenal congestion and/or hemorrhage; fibrinous interstitial pneumonia; and lymphocytolysis and lymphoid depletion in lymph nodes and spleen have been observed.

Diagnosis: Antigen-capture enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR), and virus isolation on acute-phase blood or tissue specimens from deceased individuals. Recovering animals or human develop IgM (capture ELISA) and IgG (ELISA). Immunohistochemistry can be used on tissue specimens from deceased individuals. PCR method has been used on bone marrow samples of primate carcasses in poor condition in the field for the related Ebola virus. The virus has also been visualized in organ tissues by Electron Microscopy.

Material required for laboratory analysis: Like for Ebola hemorrhagic fever, although the virus could be

MARBURG HEMORRHAGIC FEVER

detected in a large variety of biological samples (saliva, throat swabs, urine, semen, excrement, vomit, and potentially skin biopsies and bone samples), blood and tissues (spleen, liver) are the most important specimens to collect for acute case diagnosis. Protective safety equipment and safe collection methods are mandatory.

Relevant diagnostic laboratories: Marburg diagnosis can only be undertaken at BSL-4 laboratories such as Viral Special Pathogens Branch in the Centers for Disease Control and Prevention (Atlanta).

Treatment: No specific treatment for this disease exists. Supportive care, such as maintaining fluid and electrolyte balance, blood pressure, and oxygenation is the currently recommended practice for human patients. Whole blood and fresh-frozen plasma transfusions can be beneficial for the subset of patients that develop hemorrhage. Culling may be the practice of choice in outbreak situations with animals.

Prevention and control: Strict quarantine procedures for mammals imported from Central Africa should be observed. Follow CDC guidelines if importing species suspected to be reservoirs such as the Egyptian fruit bat. Caution should be observed when handling or shipping blood or tissue samples from known affected species and follow CDC and WHO guidelines. The CDC has detailed instructions regarding disinfection, quarantine, and personal protective equipment.

Suggested disinfectant for housing facilities: Viruses in the *Filoviridae* family are readily inactivated by several virucidal products. 0.5% sodium hypochlorite (10% solution household bleach), glutaraldehyde (2%) and phenolic disinfectants (0.5-3%) are recommended by the CDC. Soaps and detergents can also be used liberally and also inactivate the virus. Care should also be taken to prevent aerosolization of the virus.

Notification: All suspected cases must immediately be reported to the CDC Special Pathogens Branch, (404) 639-1115 as well as local and state health departments. Prior to collecting and sending any laboratory samples for Marburg virus testing, consult with the Special Pathogens Branch as well as the local state health department.

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Inadvisable

Conditions for restoring disease-free status after an outbreak: Under advisement of the CDC and state health department

Expert who may be consulted:

Pierre E. Rollin, MD
 Viral Special Pathogens Branch
 Centers for Disease Control and Prevention
 1600 Clifton Road
 Atlanta, Georgia 30333
 404-639-1124 (office)
 prollin@cdc.gov

References:

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MEASLES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primates, including humans; especially susceptible are colobus monkeys, macaques, and callitrichids	Aerosol	Fever, conjunctivitis, cough, and characteristic rash. Other signs of encephalitis and gastroenteritis/colitis.	Highly contagious with variable species morbidity and mortality.	None aside from symptomatic care.	Proper quarantine of animals; wearing proper protective equipment, especially when known exposure to disease. Vaccination can be considered for non-human primates.	Yes

Fact Sheet compiled by: Natalie D. Mylniczenko

Sheet completed on: 29 January 2011; updated 10 September 2013, 19 April 2018

Fact Sheet Reviewed by: Erika Travis-Crook

Susceptible animal groups: All primates – human and non-human are affected, although humans are the only known reservoir; in humans, usually young children or immunocompromised adults infected. Non-human primates are susceptible with variable morbidity and mortality that is species specific and affected by individual animal health status. With some non-human primate species, only seroconversion occurs.

Causative organism: Measles: paramyxoviridae-morbillivirus (also known as rubeola). It is an enveloped, single stranded RNA virus.

Zoonotic potential: Yes

Distribution: Worldwide, but now it is considered a foreign disease in the US as it was eliminated in 2000. Despite this status, a number of outbreaks occur each year, usually secondary to travel abroad and then spread due to lack of vaccination in groups of children.

Incubation period: Infectious 5-21 d post exposure.

Clinical signs: Disease is often asymptomatic. When clinical signs are present, they resemble influenza such as nasal and ocular discharge, and conjunctivitis. Diarrhea may be present, especially in New World monkeys. Occasionally, dermatitis is present, and rarely Koplick spots or stomatitis. Facial edema, blepharitis and erythema have been documented. Measles is immunosuppressive, therefore other diseases may confound diagnosis. Encephalitis, although rare, occurs acutely and has a rapid clinical course. Rarely further in macaques, abortion can be observed.

Post mortem, gross, or histologic findings: Exanthematous rash is noted grossly. In callitrichids, gastritis and enterocolitis is observed. Evidence of encephalitis is observed with acute measles. Syncytial cell formation and giant cell pneumonia is observed histologically. In macaques that abort, endometritis can be rarely observed.

Diagnosis: Serology IgM and IgG (paired titers with 4 fold increase in IgG titer or if IgM is found), immunofluorescence (urine), viral isolation.

Material required for laboratory analysis: Serum is preferred (frozen or fresh), although plasma is accepted at some labs. Tissue samples-see specific labs for their requirements - are usually oropharyngeal swabs, nasal lavage, or urine.

Relevant diagnostic laboratories: PCR and ELISA testing on varying sample types (contact each group for their requirements) can be done at the following facilities.

Centers for Disease Control and Prevention Measles Virus Laboratory Unit #81
1600 Clifton Road
Atlanta, Georgia 30333

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404-639-1156 or 404-639-3512

Fax: 404-639-4187

jrota@cdc.gov

<http://www.cdc.gov/measles/lab-tools/index.html>

Primate Diagnostic Services Laboratory (PDSL)

Washington National Primate Research Center

University of Washington

Seattle Washington 98195-7330

richard.grant@wanprc.org

<https://www.wanprc.org/primate-resources/pdsl/>

Primate Assay Laboratory (PAL) *Formerly PDL*

California National Primate Research Center

University of California, Davis

Phone: 530-752-8242

E-mail: cnprc-pdl@ucdavis.edu

<http://www.cnprc.ucdavis.edu/primate-assay-laboratory-core/>

Virus Reference Laboratories, Inc.

VRL-San Antonio, USA

P.O. Box 40100

7540 Louis Pasteur, Suite 200

San Antonio, Texas 78229

Office: 877-615-7275

Fax: 210-615-7771

<http://www.vrlsat.com/>

Zoologix Inc.

9811 Owensmouth Avenue, Suite 4

Chatsworth, California 91311-3800

818-717-8880

Fax: 818-717-8881

info@zoologix.com

<http://www.zoologix.com/>

Treatment: Supportive or symptomatic care, as no specific treatments are available.

Prevention and control: Vaccination has minimum age for humans of 1 year and booster is recommended to booster at least 4 weeks later although can be administered up to 4-6 years after the initial vaccinations (See <http://www.cdc.gov/vaccines/recs/schedules/child-schedule.htm#hcp>).

Vaccination in gorillas has shown positive serologic responses. Colobus have been vaccinated without adverse effects per SSP veterinary advisor reports. Vaccination against canine distemper virus in macaques has shown effective protection against measles. Human handlers should be properly vaccinated against disease.

According to human guidelines set by the CDC, pregnant women should not be vaccinated with MMR*; however, this concern is based on miscarriage or premature birth occurring in women with actual disease (<http://www.cdc.gov/vaccines/pubs/preg-guide.htm>).

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Infant macaques are vaccinated at 3 mo of age or older with a modified live vaccine. A second dose is given no sooner than 6 weeks produces protective antibody levels. Adult macaques in quarantine are vaccinated with a single dose.

*Note: monovalent measles vaccine is no longer available in the US so can only be obtained in polyvalent combinations, particularly MMR (Measles, Mumps, & Rubella).

Suggested disinfectant for housing facilities: Short lived virus, so routine disinfection is usually sufficient.

Notification: While this disease is not notifiable in animals, it is a human reportable disease.

Measures required under the Animal Disease Surveillance Plan: While this disease is not notifiable in animals, it is a human reportable disease.

Measures required for introducing animals to infected animal: Once exposed, the animal has a natural immunity and will not become re-infected. Typically, primates contract disease from human handlers.

Conditions for restoring disease-free status after an outbreak: The disease has a rapid spread and short course with no animal reservoirs.

Experts who may be consulted:

Centers for Disease Control and Prevention
1600 Clifton Road Atlanta, GA 30329-4027 USA
800-CDC-INFO (800-232-4636), TTY: 888-232-6348

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MELIOIDOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals Humans	Most common routes of infection are: contamination of wounds, ingestion of contaminated soil, water or carcasses and inhalation.	Skin lesions, pneumonia, internal organ miliary abscesses. Mimics many other diseases.	Can vary widely depending on the site of infection.	Antibiotic therapy; multiple drugs for septicemic cases; pulmonary resection may be considered for chronic cases.	Chlorinate or filter water. Minimize exposure to diseased animals.	Yes - rare

Fact Sheet compiled by: Angkana Sommanustweechai, Tanit Kasantikul, Karn Lekagul

Sheet completed on: 3 February 2011; updated 1 April 2013

Fact Sheet Reviewed by: Ronald Mitchell Bush, Rasana Wongratanachewin

Susceptible animal groups: Infection with *B. pseudomallei* is seen most often in many species of domestic animals especially goats and sheep. While cattle, pig, dog and cat have higher resistance to melioidosis. Although incidences of melioidosis in wildlife are rarely reported, cases have been documented in marine mammals, camels, alpacas, mules, zebra, deer, kangaroos, bear and various non-human primates. Reptiles such as crocodiles, snakes, soft-shelled turtles; birds, including parrots, penguin, and tropical fish can also become infected with the bacteria. Hamsters and guinea pigs can be infected in the laboratory.

Causative organism: Gram negative, flagellated, bipolar-shaped saprophytic bacteria called *Burkholderia pseudomallei*.

Zoonotic potential: Humans can be infected by ingesting contaminated food, inhalation, or direct contact of the contaminant with open wound. Intrauterine and mammary transmissions have also been observed. Arthropod borne transmission has also been described. Horizontal transmission between human to human or animal to human by aerosol is unclear.

Distribution: The organism is ubiquitous throughout southeast Asia, northern Australia, and the South Pacific. Its distribution is predominantly tropical and subtropical with "hyperendemicity" in the top end of the Northern Territory of Australia and northeast Thailand. The true boundaries of its endemicity are ambiguous due to movement of the organism and its ability to travel to and exist in temperate regions (southwest Australia and France), where it may cause sporadic disease and outbreaks. Reports of possible autochthonous melioidosis have also come from India, Pacific islands, Central and South America, the Caribbean, Africa, and the Middle East.

Incubation period: In natural infections, the incubation period in humans can vary from days to months or years. The medical onset time of the disease is usually in the range of 1-21 days (means 9 days). Abscesses may be carried without clinical signs which can be found in some resistant animal species such as pigs and cattle. The incubation period in animal particularly in wildlife is uncertain due to lack of clinical history.

Clinical signs: Called "The Great Mimicker", melioidosis has a wide range of clinical presentation, including fulminating septicemia, and chronic and local suppurative infections. Moreover, relapsing melioidosis can also

MELIOIDOSIS

cause the fulminating sepsis in patient who underwent insufficient eradication phase of treatment. The most common site of infection is acute respiratory form and sepsis through hematogenous dissemination. The chronic septicemia can present as intermittent febricula with chronic respiratory infection. Local infection can be seen as lameness, osteomyelitis, mastitis, orchitis, aortic aneurysms which may possibly induce fulminating septicemia or chronic infection. Subclinical infections are common in animals. The animals mostly undergo chronic illness. Abscesses may be found in asymptomatic animals at slaughter or died shortly after show the clinical signs. The clinical presentation also varies by species. In goat and sheep, a severe febrile reaction accompanied by anorexia, lameness and yellow thick exudate from the nose and eyes. Mastitis is sometimes seen in goats and the superficial lymph nodes and udder may contain palpable abscesses. In horses, neurologic disease, respiratory symptoms, or colic and diarrhea have been described. Neurological signs include walking in circles, nystagmus, blindness, hyperaesthesia and mild tetanic convulsions have been reported in cows, goats, camels and horses. Septicemia or extensive involvement of the vital organs can be fatal. Camels are highly susceptible and can present symptoms of pyrexia, severe depression, septic arthritis, anorexia, mucopurulent nasal discharge with nervous signs. Non-human primates mostly show generalized lethargy, progressive cachexia and respiratory distress with nasal purulent discharge. Most cases in captive marine mammals have been characterized by acute septicemia with anorexia and lethargy followed by death. Pyrexia was often recorded in the last few days preceding death, but respiratory distress was noticed only in a few animals immediately before death. Although birds may be relatively resistant to melioidosis, fatal cases with lethargy, anorexia and diarrhea have been reported in various avian species in Australia.

Post mortem, gross, or histologic findings: At necropsy, the major findings are multiple abscesses containing thick, caseous greenish-yellow or off-white material. These abscesses are generally not calcified. The regional lymph nodes, lungs, spleen, liver and subcutaneous tissues are most often involved, but abscesses can occur in most organs. In animals with respiratory disease, fibrinous pleuritis and exudative bronchopneumonia, consolidation and/or abscesses may be found in the lungs. Suppurative lesions including nodules and ulcers may also be found on the nasal mucosa and septum, as well as on the turbinate bones. These nodules may coalesce to form irregular plaques. Meningoencephalitis, severe enteritis, suppurative polyarthritis and other syndromes have also been reported. Aortic aneurysms and mastitis are common in goats. Splenic abscesses are often found in asymptomatic pigs at slaughter.

Diagnosis: The gold standard method is isolation and identification of the organism from lesions and discharges. The organism is readily cultured on routine diagnostic media such as MacConkey's agar and blood agar. The selective media, Ashdown's agar, can help increase the sensitivity and specificity of this technique. The unique characteristic of *Burkholderia pseudomallei* colony is earth odor. Effective serologic screening tests include complement fixation and indirect hemagglutination. In some species, agglutination tests, indirect hemagglutination, immunofluorescence, and enzyme immunoassays can be used for diagnosis. However, serological end points are not available for each wildlife species. Cross-reactions may occur in serologic tests with avirulent strain, *Burkholderia thailandensis*, which causes a false positive outcome in exposed animals. Although antibody titers cannot be detected in chronically infected animals, new tests using DNA probes and PCR have recently been developed. The specific primers that are designed for conserved regions to 16s rRNA, 16S-23S rRNA intergenic spacer, flagellin and lipopolysaccharide can differentiate between *B. pseudomallei*, *B. mallei* and *B. thailandensis*.

Material required for laboratory analysis: Culture swab from lesions or exudates, infected tissue or organs, serum for serologic testing.

Relevant diagnostic laboratories: Any lab is capable of culturing the organism. Currently, there is no reference lab in the world for Melioidosis listed with the Office International des Epizootics (OIE). This list can be checked at:

<http://www.test.oie.int/our-scientific-expertise/reference-laboratories/list-of-laboratories/>

MELIOIDOSIS

Treatment: The medical treatment which will take at least 4 months, can be divided into 3 phases including post exposure prophylaxis, induction and eradication phases. Treatment of septicemic melioidosis in wildlife is difficult and challenging due to the need for extended, continuous intravenous antibiotics and extra-label use of medicine. Moreover, pharmaceutical treatment can lose their effectiveness after prolonged treatment, often resulting in an unsuccessful cure, with risks of recrudescence once treatment is discontinued in animals.

Prevention and control: In endemic or contaminated areas, contact between the animal and soil should be minimized. Providing safe drinking water is important in endemic areas. Chlorine (1ppm) in the water for 30 minutes is effective in inhibiting bacterial activity in the water supply. Carnivores and omnivores should not be allowed to eat contaminated carcasses. Although there is no effective vaccine, promising vaccine candidates are currently being researched and developed. A routine environment collection for bacteriology will help in the disease surveillance and control

Suggested disinfectant for housing: *B. pseudomallei* can survive for months to years in soil and water, but can be readily destroyed by heat. Moist heat of 121°C for at least 15 min or dry heat of 160-170°C for at least 1 hour is recommended for disinfection. The organism is also susceptible to numerous disinfectants, including 1% sodium hypochlorite, 70% ethanol, glutaraldehyde and formaldehyde.

SPILLS: Allow aerosols to settle; wear protective clothing; gently cover spill with paper towels and apply 1% sodium hypochlorite, starting at perimeter and working towards the centre; allow sufficient contact time (30 min) and clean the area. 40% W/W calcium oxide is proved to be effective in preventing bacterial activity in the environment for 1 year.

Notification: Public health officials and state veterinarians will need to be notified if zoonotic transmission occurs.

Measures required under the Animal Disease Surveillance Plan: Melioidosis is not listed under this plan.

Measures required for introducing animals to infected animal: An infected animal should be maintained in a quarantine situation until the wound has healed. Do not introduce infected animal to an animal with a compromised immune system.

Conditions for restoring disease-free status after an outbreak: Follow the suggestions above for disinfection of facilities and maintaining uncontaminated water sources. Decontaminate waste before disposal; steam sterilization, incineration, chemical disinfection. Quarantine any affected individuals until lesions resolved.

Experts who may be consulted: There are no listed OIE experts for *Burkholderia pseudomallei*. That said, the following people deal with Melioidosis routinely and would be willing to respond to questions from professionals dealing with confirmed or suspect cases:

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MELIOIDOSIS

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MRSA (METHICILLIN-RESISTANT *Staphylococcus aureus*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals including Humans	Contact with contaminated surfaces	Minor to severe: skin redness, pustule red lesions, boils, rash fever, headache, malaise	Typically mild, but may be fatal in the immune compromised. No mortality rates are reported in animals, but disease increasingly common in ICU foals.	Wound care; susceptible antibiotics as determined by testing, when needed	Personal/ environmental hygiene. Wear gloves when handling known infected animal and equipment	Yes

Fact Sheet compiled by: Tara M. Harrison

Sheet completed on: 29 November 2009; updated 7 September 2012, updated 2018

Fact Sheet reviewed by: Dalen Agnew, Christine Fiorello, Donald Janssen

Susceptible animal groups: Mammals, avian (+/-)

Causative organism: Methicillin-resistant *Staphylococcus aureus*, also Methicillin-resistant *Staphylococcus pseudointeritidis*

Zoonotic potential: Yes

Distribution: Crowded living conditions, group work and gyms, closely shared work and locker spaces, long-term care or rehabilitation facilities, hospitals. A captive chimpanzee colony was found to have 69% prevalence of MRSA. There was also a wide variety of asymptomatic mammals that cultured positive at a Copenhagen zoo. MRSA was also isolated from clinical and non-clinical animals at a Belgium zoo. An elephant skin infection was also caused by MRSA in a California Zoo.

Incubation period: Generally, it requires 1-10 days. People (7%) in hospitals and in the community (2%) can have MRSA colonization with no clinical signs. It is thought that <10% to up to 90% of dogs and cats can be non-clinical carriers as well.

Clinical signs: Healthy people and animals typically do not develop disease under normal circumstances.
 Humans: Skin redness, “pimple-like” red lesions, boils, rash, fever, headache, malaise
 Animals: Primarily skin infections or skin wounds although necrotizing pneumonia or other general infection may occur.

Post mortem, gross, or histologic findings: This bacterium can produce a wide spectrum of clinical disease, particularly of the skin. In humans, these diseases include impetigo, folliculitis, furunculosis, cellulitis, abscesses and wound infections. Other diseases include necrotizing pneumonia, endocarditis, septic arthritis, osteomyelitis, meningitis, and septicemia.
 In animals, abscesses, dermatitis, fistulas have been reported; as well as pneumonia, rhinitis, bacteremia, septic arthritis, osteomyelitis, omphalophlebitis, metritis, and mastitis. Post-mortem lesions are similar to any other purulent bacterial infection and vary with the organ or tissue involved in the infection.

Diagnosis: Bacterial culture and antibiotic susceptibility testing

Material required for laboratory analysis: Culture swab or tissue sample of the affected area

Relevant diagnostic laboratories: Any laboratory capable of bacteriologic culturing is capable of diagnosing MRSA.

Treatment: Typically, it is resistant to all β -lactam agents, including cephalosporins and carbapenems. Hospital-associated MRSA isolates often are resistant to multiple commonly used antimicrobial agents, including erythromycin, clindamycin, and tetracycline, while community-associated MRSA isolates are often resistant only to β -lactam agents and erythromycin.
 Treatment specifically depends on the specific MRSA isolate, and its antibiotic sensitivity profile. This will require sensitivity testing on ALL isolates and possibly repeated testing on isolates from a single case.
 In humans: Vancomycin (if not resistant), linezolid, and daptomycin, quinupristin/dalfopristin, rifampin,

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tetracycline, and tigecycline are used for severe MRSA infections or MRSA infections resistant to vancomycin.

Prevention and control: Minimization of indiscriminate antibiotic use would help prevent the development of additional antibiotic-resistant strains.

Follow all wound care procedures recommended by veterinarian or physician. Practice good hygiene; wash hands often. Keep cuts and scrapes clean and cover with bandages, avoid direct contact with cuts and scrapes, use gloves to treat wounds, replace and disinfect items in holding or exhibit frequently. Porous surfaces such as blankets need to be washed in hot water using bleach and a hot air dryer to help kill bacteria. Alcohol-based hand cleaners are effective when hands aren't dirty.

Isolate the patient if possible, to minimize staff contact and exposure. Animal enclosures should be clearly marked with the diagnosis and preventative measures required. Maintain infected animal in isolation or away from other animals until wound(s) are healed or cultures are negative. If treatment of the animal is not possible, humane euthanasia of infected animal may be warranted to minimize risk of infection to staff and other animals.

Suggested disinfectant for housing facilities: After cleaning gross contamination, 1 tablespoon of bleach to one quart of water, fresh daily, leave solution on to dry, or wipe dry after 10 minutes. Other disinfectants effective against *Staphylococcus aureus* or *Staph* are also most likely also effective against MRSA. Check the disinfectant product's label on the back of the container to verify it is effective against it.

Notification: Public health officials may need to be notified if zoonotic transmission occurs, depending on the state. Notification to the state may be required if the person is admitted to an acute care ICU or person dies from MRSA or it is *not* associated with the following: been hospitalized, had surgery, had dialysis, been in long term care within the last year, has an indwelling catheter, or has a percutaneous medical device at the time of culture.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Maintain infected animal in a quarantine situation until the wound is healed. Do not introduce infected animal to an animal with a compromised immune system.

Conditions for restoring disease-free status after an outbreak: Clean infected environment with diluted bleach to the extent possible. Minimize contact of infected staff with animal.

Experts who may be consulted:

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans; nonhuman primates; rodents (especially prairie dogs); CDC recommends that all mammals be considered susceptible	Contact with an infected animal, human, or contaminated materials through broken skin, respiratory tract, or mucous membranes	Papulovesicular dermatitis; upper respiratory disease; blepharophlebitis (prairie dogs); fever; lethargy; decreased food/water; lymphadenopathy; asymptomatic	High case fatality rate in prairie dogs; variable mortality in other species; African rodents are suspected reservoir	No specific treatment	Guidelines available from the CDC	Yes

Fact Sheet compiled by: Kelly Helmick

Sheet completed on: 21 February 2018

Fact Sheet Reviewed by: Jennifer Kilburn, Chris Hanley

Susceptible animal groups: The reservoir and full host range is unknown, but African rodents are suspected in transmission. Old and New World primates and rodents have shown susceptibility to experimental and natural infection. Virus recovery from naturally infected animals outside of the U.S. has been limited to a clinically ill rope squirrel (*Funisciurus* sp., 1985, Democratic Republic of Congo) and a dead infant mangabey (*Cercocebus atys*, 2012, Tai National Park, Cote d'Ivoire). Viral testing during the 2003 U.S. outbreak identified infection in imported rope squirrels (*Funisciurus* sp.), dormice (*Graphiurus* sp.), and African giant pouched rats (*Cricetomys* sp.), and in exposed prairie dogs (*Cynomys* sp.). Prairie dogs appear very susceptible to infection. Chinchillas (*Chinchilla lanigera*) and coatimundis (*Nasua nasua*) developed antibodies after exposure. Serological evidence of monkeypox virus infection has been detected in non-human primates, rodents, and squirrels in Africa. Experimentally infected rope squirrels and Gambian pouched rats shed large quantities of virus. Experimentally infected marmosets (*Callithrix jacchus*) and ground squirrels (*Marmota bobak*) developed typical clinical signs. Currently the CDC recommends that veterinarians consider all mammals susceptible to monkeypox virus.

Causative organism: Monkeypox virus (*Orthopoxvirus*, family Poxviridae). Two clades: Central African and West African.

Transmission: Contact with an infected animal, human, or contaminated materials through broken skin, respiratory tract, or mucous membranes. Animal-to-human: bite, scratch, bush meat preparation, needle sticks; direct contact with infected fluids or lesion material; indirect contact (contaminated bedding). Cutaneous transmission implicated in the 2003 U.S. outbreak. Human-to-human: respiratory droplets shared via prolonged face-to-face contact; direct contact with infected fluids or lesion material; indirect contact (contaminated clothing, bedding).

Zoonotic potential: Yes. Rare zoonotic viral disease endemic to central and west Africa. The West African clade is associated with limited human-to-human transmission, milder symptoms, and lower mortality compared to the Central African clade. Human-to-human transmission of the Central African clade is well-documented. The most recent human cases occurred in Sierra Leone (2014) and Sudan (2005). Monkeypox is endemic in the Democratic Republic of Congo.

2003 U.S. outbreak: A human outbreak involving the West African clade of monkeypox occurred in the U.S. in 2003. Introduction of monkeypox virus occurred through a shipment of small mammals originating in Ghana. CDC testing isolated virus from rope squirrels (*Funisciurus* sp.), dormice (*Graphiurus* sp.), and African giant pouched rats (*Cricetomys* sp.). Prairie dogs (*Cynomys* sp.) housed in proximity to imported

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animals were also infected and sold as pets prior to developing signs of infection. All human cases were associated with contact with infected prairie dogs.

Distribution: Monkeypox virus is endemic to central and west Africa. A 2003 outbreak occurred in the U.S. involving prairie dog-to-human transmission traced to contact with newly imported infected African rodents.

Incubation period: The incubation period in humans is typically 7-14 days but can range from 5-21 days.

Clinical signs:

Humans: Fever, headache, muscle aches, lethargy, chills, and swollen lymph nodes appear first. A rash develops approximately 1-3 days later, usually on the face and then spreading to other areas of the body. Lesion progression is macules, papules, vesicles, pustules, then scab formation. Symptoms last approximately 2-4 weeks. In Africa, human mortality occurs in approximately 1 of 10 cases. Symptoms mimic smallpox; lymphadenopathy occurs in monkeypox but not smallpox.

Rodents: In naturally infected prairie dogs and experimentally infected rope squirrels: fever; respiratory symptoms of coughing, nasal discharge, ocular discharge; rash beginning as papules progressing to pustules then crusts affecting head, extremities, trunk; oral ulcers; blepharophlebitis (naturally infected prairie dogs); lymphadenopathy may or may not be present (experimentally infected rodents); lethargy; reduced food/water intake; elevated serum liver enzymes. Some animals exhibited minimal clinical symptoms while others died.

Nonhuman primates: Fever; rash beginning as papules progressing to pustules then crusts typically on the face, limbs, hands, feet, tail; respiratory symptoms of coughing, nasal discharge, dyspnea; anorexia, facial edema; lymphadenopathy. Similar symptoms to rodents observed in experimentally infected non-human primates (*Cynomolgus* sp.).

Postmortem, gross, or histologic findings: Lymphadenitis, skin rash, and evidence of upper and lower respiratory disease on gross postmortem exam. Lower respiratory epithelium is the target cell for virus replication with lymphoid tissue a secondary site for replication and lymphatogenous spread. Trachea, nasal mucosa, skin, hepatocytes, and macrophages can demonstrate high levels of monkeypox virus presence (marmosets, ground squirrels). Infected epithelial cells show prominent ballooning degeneration and dense, eosinophilic, intracytoplasmic granules (prairie dogs). Eosinophilic cytoplasmic granules (Guarnieri-like inclusions) require IHC or EM to confirm orthopoxviral inclusions. Necrotizing bronchopneumonia, conjunctivitis, and tongue ulceration (prairie dogs). Bronchopneumonia, papulovesicular dermatitis, ulcerative stomatitis, colitis, gastritis, secondary bacterial septicemia (experimentally infected *Cynomolgus* monkeys). Use appropriate PPE when examining or collecting diagnostic samples from animals known or suspected to have monkeypox virus.

Diagnosis: Clinical symptomology in rodents, animals originating from endemic regions, or animals housed in proximity to African rodents originating from endemic regions. In humans, monkeypox differs from smallpox by the presence of lymphadenopathy. RT-PCR, immunohistochemistry, virus isolation, and electron microscopy. There is no commercial assay to detect monkeypox virus.

Material required for laboratory analysis: Tonsillar swab; nasopharyngeal swab; aspirate of vesicles; biopsy of lesions; scab or crust collection; serum or whole blood (EDTA) collection. Wear appropriate PPE and practice appropriate biosecurity. Formalin-fixed samples can be held and shipped at room temperature. All other samples are held and shipped at 4°C. Do not use viral transport media. Pack and ship according to IATA rules and regulations for diagnostic specimens. Use appropriate PPE when examining or collecting diagnostic samples from animals known or suspected to have monkeypox virus.

Relevant diagnostic laboratories:

Veterinarians should contact their local or state health departments regarding monkeypox virus testing before contacting the CDC. There is no commercial assay to detect monkeypox virus. Culture based testing for monkeypox virus should be limited to the CDC.

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Treatment: There is no specific treatment for monkeypox virus in humans or animals. Provide supportive treatment with appropriate biosecurity and PPE guidelines.

Prevention and control: Foster good hygiene practices (hand washing), utilize PPE (gloves, masks), and follow biosecurity protocols. Limit contact by humans or mammals with known or suspected infected animals and bedding material, especially animals arising from regions where monkeypox virus is endemic. Utilize practices to limit or eliminate animal bites, scratches, needle sticks, or other injuries. Isolate suspected animals. The CDC bans importation of all African rodents into the U.S.

Suggested disinfectant for housing facilities: Contact state or local health authorities for guidelines when monkeypox virus infection is known or suspected. Consult with state or local public health officials for proper waste disposal; do not dispose of contaminated waste in a dump, landfill, or by routine hospital waste disposal methods. Conduct environmental cleaning using any EPA-registered hospital disinfectant used for health care facilities or environmental sanitation. Laundry can be cleaned using hot water, detergent, and bleach using a standard washing machine.

Notification: Report suspected or confirmed cases to the appropriate local or regional animal and public health authorities.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: None – not recommended.

Conditions for restoring disease-free status after an outbreak: Euthanasia, quarantine, and/or disinfection and incineration protocols as recommended by local, state, and/or federal health and regulatory agencies.

Experts who may be consulted:

Center for Disease Control: <https://www.cdc.gov/poxvirus/monkeypox/index.html>

State animal health officials: <http://www.usaha.org/federal-and-state-animal-health>

Local public health agencies

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ATYPICAL MYCOBACTERIOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Fish Amphibians Reptiles Birds Mammals	Ingestion; inhalation; waterborne; environmental exposure via defects in respiratory, integumentary, or urogenital systems; direct extension via bite wounds	Variable to none; Cutaneous lesions, ascites, pneumonia, mastitis, lymphadenopathy, lameness, emaciation, lethargy	Asymptomatic to chronic disease or acute death	May not be advised. Antibiotics: aminoglycoside; quinolone; macrolide	Good sanitation, good wound care, prevent contact with contaminated water, soil, or feed	Yes

Fact Sheet compiled by: Elizabeth Manning

Sheet completed on: 15 April 2011; updated 21 July 2013, updated 2018

Fact Sheet Reviewed by: Kurt Volle; Shannon Cervený

Susceptible animal groups: Fish, amphibians, reptiles, birds, mammals

Causative organisms: This group includes all *Mycobacteria* except *M. tuberculosis* complex and *M. leprae*. Non-tuberculous mycobacteria – *Mycobacterium avium*, *M. intracellulare*, *M. marinum*, *M. fortuitum*, *M. chelonae*, *M. porcinum*, *M. farcinogenes*, *M. smegmatis*, *M. scrofulaceum*, *M. xenopi*, *M. kansasii*, *M. simiae*, *M. genavense*, and others - are slender, nonmotile, acid-fast bacilli that are classified as slow growing or rapidly growing.

Zoonotic potential: Yes. Many of these bacteria species may infect people who have a genetic predisposition or diminished immune function. Typically, they are not transmitted between humans or between animals and humans. Most infections are acquired from environmental sources, but infection may result secondary to abrasions, cuts, or similar disruption to surfaces.

Distribution: Ubiquitous worldwide

Incubation period: Typically, two weeks to greater than 2 months, however the Runyon Group IV (*M. chelonae*, *fortuitum*, *smegmatis*) are rapid growing and need less than 7 days for incubation.

Clinical signs: Variable clinical signs are observed which depend on species infected and site of infection. Asymptomatic to acute death presentations are possible. Other signs include: lethargy, emaciation, and other non-specific signs of illness; cutaneous ulcers, abscesses, and granulomas; enlarged abdomen and ascites; cough, dyspnea, pneumonia; mastitis; lymphadenopathy; and lameness due to bone infections.

Post mortem, gross, or histologic findings:

Gross: Granulomas in multiple organs, cutaneous ulcers and/or abscesses, ascites, pneumonia, mastitis, lymphadenitis, osteomyelitis, tenosynovitis, arthritis

Histologic: Granulomatous inflammation

Diagnosis: From cytology or histopathology samples, acid-fast bacilli can be demonstrated and tissue culture can be followed by biochemical identification of the bacteria. Polymerase chain reaction (PCR) is available.

Material required for laboratory analysis: For culture, fresh tissue samples are required. For histopathology, formalin-fixed tissue samples are submitted which can then be used for PCR. Direct lesion sampling by swabs can also be used with PCR.

Relevant diagnostic laboratories: National Veterinary Services Laboratories
1920 Dayton Avenue, Ames, Iowa, 50010, USA
515-337-7266 NVSL_Concerns@aphis.usda.gov
http://www.aphis.usda.gov/animal_health/lab_info_services/

Treatment: Due to possibility for development of antibiotic resistance and safety concerns for personnel in close contact with affected animals, treatment may not be recommended. Treatment when attempted should be based on antimicrobial susceptibility testing but empirical treatment options include: aminoglycosides,

ATYPICAL MYCOBACTERIOSIS

quinolones, and macrolides. Radical surgical excision of cutaneous lesions in conjunction with long-term antibiotic therapy has been described.

Prevention and control: Once diagnosed, excellent sanitation measures and permanent quarantine of known positive animals should be introduced. Appropriate wound care and prevention of wound contact with potentially contaminated water, soil, and feed will minimize these infections.

Suggested disinfectant for housing facilities: Tuberculocidal products as listed by the US EPA
http://www.epa.gov/oppad001/list_b_tuberculocide.pdf

Notification: Not required

Measures required under the Animal Disease Surveillance Plan: None required

Measures required for introducing animals to infected animal: None required

Conditions for restoring disease-free status after an outbreak: Due to ubiquitous nature of the etiologic agents, chronic profile, and inability to diagnose carrier state, disease-free status is not possible.

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AVIAN MYCOBACTERIOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All birds, some mammals	Fecal-oral, environment, inhalation	Emaciation, weakness, lethargy, hepatosplenomegaly Rarely skin lesions and respiratory disease	Variable. Severe in the individual with end stage disease	Not recommended as this organism is resistant to most, if not all human antimycobacterial drugs. Euthanasia may need to be considered	Cleaning of the environment. Decreasing load in the environment. Maintaining good immune systems and good husbandry	Yes, but humans have a high resistance to <i>M. avium</i> unless immune compromised. Treatment may be difficult.

Fact Sheet compiled by: Nancy Carpenter

Sheet completed on: 1 February 2011; updated 1 March 2013

Fact Sheet Reviewed by: Erika Travis-Crook, M. Scott Echols

Susceptible animal groups: Birds, some mammals, such as pigs, mink and rabbits.

Causative organism: *Mycobacterium avium* complex (MAC) consisting of *M. avium* and *M. intercellularae*. *M. genavense* can also cause disease in birds.

Zoonotic potential: There is potential, however humans appear to be highly resistant unless immune compromised.

Distribution: Worldwide. However in North America the distribution favors the North Temperate Zone.

Incubation period: There is not a definitive incubation period because the resultant disease is dependant upon immune response to exposure. Exposure does not guarantee disease. Typically an animal suffering from disease caused by Mycobacteria may have had the disease for many years before signs are recognized or, more likely, it is an incidental finding on necropsy.

Clinical signs: Emaciation, weakness, lethargy, hepatosplenomegaly.

Post mortem, gross, or histologic findings: Emaciated carcasses, hepatosplenomegaly, nodular disease in affected organs. Nodules are typically white to yellow and solid to soft or crumbly in consistency. Liver, spleen, lung and intestines are most commonly affected but joints, skin, and respiratory tract may also show lesions.

Diagnosis: Elevated white blood cell counts >60,000 can be an indicator of mycobacteriosis. Antemortem screening can be performed via coelomosopic examination focusing on the liver, spleen and intestines. Biopsy any plaque like lesions or the liver for histopathological screening. Diagnosis is attained through the identification or culture of acid fast organisms or histopathology as the most common route. However, acid fast staining of prepared feces can also be done but is not a definitive test since other organisms can be acid fast positive confounding results. Tuberculin testing is not recommended. PCR assays detect the actual disease causing organism and are considered to be the fastest, most sensitive method for detecting *M. avium*. ELISA assays detect specific antibodies for *M. avium* and help determine exposure. These assays can be performed on whole blood, feces, serum, vent and throat swabs depending upon the laboratory and the test to be run. (Feces for Zoologix; whole blood, serum, vent and throat swabs for Avian Biotech International)

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Material required for laboratory analysis: Acid Fast staining of slides from a suspect nodule, feces or touch prep of affected tissues; formalinized tissue for histopath examination; culture swab for acid fast testing and culture (Lowenstein Jensen media required for culture). Feces for PCR by Zoologix or whole blood, serum, vent and oral swabs for Avian Biotech International for PCR or ELISA. Post mortem sampling includes liver, spleen and lungs and/or suspect areas.

Relevant diagnostic laboratories:

Avian Biotech International (www.avianbiotech.com)

Zoologix (www.zoologix.com). See the Avian and Livestock Assay Data Sheet

Treatment: Typically, control is more desired as treatment can be unrewarding and possibly cause further spread of the disease. Some antibiotic resistance can be expected. The ethics of treatment must be considered as treatment may be life long and may not prevent shedding.

Prevention and control: Try to maintain a clean environment and be diligent in screening via necropsy and testing for acid fast bacteria. Maintain a thorough quarantine protocol.

Suggested disinfectant for housing facilities: Cidex appears to be the product that is the standard efficacy comparison in most studies. Equivalent disinfectants include Sactimed sinald (a quaternary ammonium compound) Steris 20 (a peracetic acid compound) and Pentapon DC1 (a beta-ene compound) are equally effective. Persafe (a tertiary amine that is classified as an HLD High Level Disinfectant) is also reported to be as effective as Cidex. Virkon was NOT effective. Roccal D does not list *M. avium* as being susceptible to that product. Some of these may not be applicable for premise application. Sukusept Plus (Ecolab) is a glucoprtoamin based disinfectant and has effectiveness against all mycobacteria at 2500 ppm for 15 minutes. It is also effective against a glutaraldehyde resistant *M. chelonae* but at a concentration of 5000 ppm for 15 minutes, or at 2500 ppm for 60 minutes. Note that this product may not be available in the US. 1 Stroke Environ B (Vestal Labs), Virostat TBQ, Steris TBQ, Husky QT 814 are other premise disinfectants with efficacy against mycobacteria. During premise disinfection it is recommended that a protective face covering i.e. respirator is worn due to the route of infection for these organisms is through aerosolization.

Notification: Check your individual state for reporting requirements

Measures required under the Animal Disease Surveillance Plan: This is not one of the listed diseases as of 2013.

Measures required for introducing animals to infected animal: If an animal is known to be infected, euthanasia may need to be considered. It is not recommended to mix a known infected animal with a healthy animal unless the risk for infection is considered acceptable. Studies show that there is an increased incidence of disease when an animal is housed with a known positive.

Conditions for restoring disease-free status after an outbreak: As this bacterium is ubiquitous, this condition is unachievable. Efforts should be concentrated on decreasing the environmental load of this bacterium and enhancing the immune response for those living in the contaminated environment through good nutrition and proper husbandry. Screening of all deaths for mycobacteria, having sentinel animals in the enclosure, and periodic liver biopsies have all been done.

AVIAN MYCOBACTERIOSIS

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TUBERCULOSIS IN ELEPHANTS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, including elephants	Aerosol of infectious droplets	Chronic weight loss, excessive mucus discharge from the trunk and respiratory system.	Variable ; Most infected elephants have <u>no</u> clinical signs after many years of chronic infection.	At least three drugs should be used when initiating treatment. Toxicity related to drug treatment has been reported, including hepatopathy, icterus, bone marrow suppression, and anorexia.	Quarantine testing: while culture is the only definitive test, ancillary tests include elephant STAT-Pak and other <i>in vitro</i> assays	Yes

Fact Sheet compiled by: Gary West and Charles O. Thoen

Sheet completed on: 31 January 2011; updated 31 January 2013

Fact Sheet Reviewed by: Linda Peddie; Dennis Schmitt; Paul P. Calle; Michele Miller

Susceptible animal groups: Mammals, including elephants

Causative organism: Predominantly *Mycobacterium tuberculosis* and rarely *M. bovis* have been associated with these infections. *M. avium* complex and certain other mycobacteria (*M. szulgai* and *M. elephantis*).have been isolated from elephants

Zoonotic potential: Yes

Distribution: Worldwide distribution in captive animals and in free-ranging animals in close contact with humans (i.e., working elephants in Asia). Chronically infected and shedding elephants and their caretakers have been noted. Animals traditionally have been relocated without rigorous quarantine. Trunk washes should be collected from animals for mycobacteriologic examinations (ie. PCR and culture) before integration into new herds.

Incubation period: Weeks to years.

Clinical signs: The most commonly observed sign is chronic weight loss. Elephants may also have mucoid sputum discharge from trunk and partial anorexia. However, often no premonitory signs of illness are present until the disease is in the very advanced stages.

Post mortem, gross, or histologic findings: Primarily lung and associated thoracic lymph nodes are observed with chronic granulomas with caseocalcaerous and cavitated lesions. Lesions often are paucibacillary on acid fast staining.

Diagnosis: Laboratory examinations on trunk wash by acid fast staining and culture of the fluid. Ancillary tests include Elephant TBstatPAK® and MAPIA™ for serology. In the US, elephant TBstatPAK testing should coincide with the trunk wash collection per current USDA guidelines. Although not permitted for the official USDA testing, sputum samples or mucus from the trunk can also be cultured and may be useful. Additionally, mycobacterial organisms rarely have been isolated from other body fluids such as vaginal secretions. Post-mortem cultures should be performed.

NOTE: Some elephants with chronic inflammatory conditions have tested positive on the Elephant

TUBERCULOSIS IN ELEPHANTS

TBstatPAK and have not been positive on culture for *Mycobacteria* species. Trunk wash should be collected from these animals if they originate in a herd with a history of tuberculosis or of unknown source as the animals could be infected and not shedding the tubercle bacillus. Intradermal tuberculin testing is not recommended for elephants due to the non-specific reactions observed in this species.

Material required for laboratory analysis: Saline wash of the trunk for mycobacteriologic examination. Culture, PCR on lesions collected at necropsy or on biopsy. Blood may be collected for Elephant TBstatPAK and MAPIA and other *in vitro* supplemental tests. Consult the current Guidelines to Control Tuberculosis in Elephants for additional information.

http://www.aphis.usda.gov/animal_welfare/downloads/elephant/elephant_tb.pdf

Relevant diagnostic laboratories: NVSL (National Veterinary Services Laboratory, Ames, IA) in the US, or similar reference laboratories in other countries.

Treatment: Empirical treatment with at least three drugs (isoniazid, rifampin, ethambutol, and pyrazinamide) while susceptibility tests are pending. Drug resistance has been a concern in a few cases; therefore fluoroquinolones can also be used in combination with other medications. Pyrazinamide is ineffective against *M. bovis*.

Toxicity related to drug treatment has been observed and may include signs of gastrointestinal discomfort, hepatopathy, bone marrow suppression, malaise, and joint stiffness. Prior to beginning treatment, it is recommended that clinicians consult with others with experience in elephant mycobacterial treatment to ensure that the latest information is incorporated into the treatment plan.

Prevention and control: Elephants with chronic unexplained weight loss and identified shedders should be isolated from other animals. Quarantine and test new arrivals to the institution. Anti-tuberculous disinfectants should be used for cleaning. Consideration for staff should be given to wearing HEPA-filter masks that are certified to protect against tuberculosis when collecting trunk wash samples or when in close contact with infected elephants.

Suggested disinfectant for housing facilities: EPA approved tuberculocidal agent.

Notification: USDA in the US, appropriate regulatory officials in other countries.

Measures required under the Animal Disease Surveillance Plan: Specific guidelines are in place in the US http://www.aphis.usda.gov/animal_welfare/downloads/elephant/elephant_tb.pdf

Measures required for introducing animals to infected animal: Follow USDA/APHIS or similar guidelines as above under surveillance. Animals that are not actively shedding *Mycobacteria* and are undergoing treatment may be considered for reintroduction to the herd which may decrease the stress of the animal.

Conditions for restoring disease-free status after an outbreak: see USDA/APHIS Guidelines to Control Tuberculosis in Elephants http://www.aphis.usda.gov/animal_welfare/downloads/elephant/elephant_tb.pdf

Experts who may be consulted:

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TUBERCULOSIS IN ELEPHANTS

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- 12.

**MYCOBACTERIUM AVIUM SUBSP. PARATUBERCULOSIS
(JOHNE'S DISEASE)**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ruminants	- Fecal-oral - Raw colostrum - <i>In utero</i>	-Weakness -Weight loss -Diarrhea in some species	Eventually fatal	None	-Quarantine or cull infected animals -Regular herd testing -Good sanitation	Inconclusive

Fact Sheet compiled by: Elizabeth Manning
Sheet completed on: 19 April 2011; updated 21 July 2013
Fact Sheet Reviewed by: Genevieve Dumonceaux; Patrick Pithua
Susceptible animal groups: Ruminants are affected and though other mammals and birds may become infected, they rarely develop clinical disease
Causative organism: <i>Mycobacterium avium</i> subspecies <i>paratuberculosis</i> (MAP) which is a Gram-positive, acid-fast positive small rod-shaped bacterium that grows in clumps of 10-100 cells and a member of the <i>Mycobacterium avium</i> complex (MAC).
Zoonotic potential: As an opportunistic pathogen in immunocompromised individuals, zoonotic status is possible but it is very unlikely in the zoo setting as organism must be swallowed by immunosusceptible human. Much controversy exists regarding the relationship between MAP and Crohn's disease in humans.
Distribution: Global
Incubation period: Months to years
Clinical signs: These signs are observed primarily in adults although animals are usually infected in the first months of life or <i>in utero</i> if dam is infected. Early course of infection is asymptomatic. Severe and abrupt weight loss occurs as disease progresses with weakness secondary to emaciation. Some species present with chronic diarrhea.
Post mortem, gross, or histologic findings: <u>Gross:</u> These findings range from none to many including: corrugated, reddened, thickened gastrointestinal tract; enlarged mesenteric lymph nodes and enlarged lymphatic vessels; and emaciation with lack of fat stores. <u>Histologic:</u> These findings range from minimal to extensive granulomatous inflammation. Variable numbers of acid-fast positive rods in giant cells of the ileum and mesenteric lymph nodes can be found and, in some species, aortic mineralization is noted.
Diagnosis: <u>-Organism-based:</u> culture (feces, tissue samples, environmental samples), PCR (feces, paraffin blocks of tissue samples, culture isolate identification), histopathology (acid-fast positive rods within macrophages infiltrating the caudal gastrointestinal tract and mesenteric lymph nodes) <u>-Serology:</u> ELISA for cattle, sheep, goats, bison, deer
Material required for laboratory analysis: <u>Ante-mortem:</u> Feces, serum, milk, colostrum, and environmental samples (soil, water, grass) <u>Post-mortem:</u> Fresh mesenteric lymph node, ileum samples for culture; in formalin for histopathology
Relevant diagnostic laboratories: -Animal and Plant Health Inspection Service (APHIS)-Approved Laboratories are listed at: http://www.aphis.usda.gov/animal_health/lab_info_services/approved_labs.shtml -Lab familiarity with zoo collection, diagnostics, and husbandry is helpful

**MYCOBACTERIUM AVIUM SUBSP. PARATUBERCULOSIS
(JOHNE'S DISEASE)**

Treatment: None
Prevention and control: Adherence to principles driving guidelines in the Voluntary Johne's Disease Herd Status Program (http://www.aphis.usda.gov/animal_health/animal_diseases/johnes/downloads/johnes-umr.pdf) and including: <ul style="list-style-type: none">-Screening incoming adult ruminants during quarantine period (if < 1yr. old, test dam).- Feed only pasteurized colostrum or approved commercial colostrum replacers to neonatal ruminants.-Bottle feed neonatal ruminants with pasteurized milk or commercial milk replacer.-Prevent exposure of ruminants less than 6 months of age to adult manure including through water drainage or feed contamination.-Use sod from pastures grazed by Johne's disease-free ruminants.-Establish ruminant enclosure status (all adults in enclosure/barn).- Establish and use a dedicated maternity area separate from lactating cows to reduce the risk of transmission-Examine all adult ruminants dying on site for Johne's disease even if other cause of death known – including culture of mesenteric lymph node.-Learn MAP status of source herds for petting zoo/farm animals as high prevalence in domestic ruminants-Institute excellent sanitation measures.
Suggested disinfectant for housing facilities: Tuberculocidal products as listed by the US EPA http://www.epa.gov/oppad001/list_b_tuberculocide.pdf
Notification: Reportable to the World Organisation for Animal Health (OIE), USDA APHIS, and many state veterinarians.
Measures required under the Animal Disease Surveillance Plan: None required although voluntary program participation exists.
Measures required for introducing animals to infected animal: It is not recommended
Conditions for restoring infection-free status after an outbreak: Following quarantine or cull infected animals and environmental decontamination it would be useful to demonstrate negative test results in adult ruminant(s) over multiple years.
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(JOHNE'S DISEASE)**

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TUBERCULOSIS IN NON-HUMAN PRIMATES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Non-human primates	Inhalation and ingestion predominates ;fomite potential documented	Varies: rough hair coat, weight loss, cough, lymph-adenopathy	Highly variable: asymptomatic to severely debilitating disease.	Limited efficacy even with multi-modal treatment; but may be considered for extremely valuable animals. However, culling of positive animals highly recommended	Skin test is routine and gold standard; but nonspecific responses occur.	Yes – and anthroponotic

Fact Sheet compiled by: Patrice Frost, Heather Cole, Charles O. Thoen

Sheet completed on: 3 August 2011; updated 31 January 2013

Fact Sheet Reviewed by: Paul P. Calle; Hilton Klein; Ana Cristina Leandro

Susceptible animal groups: All primates, including humans.

Causative organisms: *Mycobacterium tuberculosis*; *M. bovis*, *M. avium ss avium*.

Zoonotic potential: Yes

Distribution: Old World non-human primates and great apes – usually with typical mycobacterial infection; New World non-human primates – usually with other mycobacterial infection(s).

Incubation period: Variable from weeks to months; animals can develop latent infections with reactivation in weeks, months or even years later. Development of disease is dependent on organism, route of infection, dose and immunologic status of animal. Susceptibility, morbidity and mortality are variable for different species.

Clinical signs: The clinical signs are often nondescript and ill-defined. Tuberculosis can imitate a multitude of diseases such as pneumonia, neoplasia or fungal infections. The clinical spectrum of signs range from asymptomatic to multi-symptomatic; the profile is highly dependent on the route of exposure, the system involved and the infecting agent. General signs can include a roughened hair coat, anorexia, depression, lethargy, fever (low grade; intermittent or persistent), weight loss, hepatomegaly, splenomegaly, and local or general lymphadenopathy which may or may not have draining tracts. A chronic or paroxysmal cough and dyspnea indicate pulmonary involvement, which mirrors acute bronchitis, or pneumonia. Neurological presentation with signs including anisocoria or ataxia may implicate meningitis or central nervous system involvement, and paresis to paralysis can indicate a peripheral neurological component that may be a result of spondylitis.

Post mortem, gross, or histologic findings: At necropsy, tuberculosis indications vary with the duration and degree of disease. Organs of predilection are the lung and adjacent hilar lymph nodes. Dissemination occurs to the spleen kidney, liver and associated lymph nodes. Additional sites less frequently seen include omentum, ovary, cerebrum, spinal column, peripheral lymph nodes skin and mammary gland. The extent of the lesions can range from no detectable lesions to wide dissemination of caseous granulomas varying in size from pinpoint to large coalescing lesions. Appearance of lesions within the lung can be focal, coalescing or cavitary.

TUBERCULOSIS IN NON-HUMAN PRIMATES

Lesions in parietal pleural with adjacent adhesion maybe caused by collapse of large granulomas expelling contents into the adjacent airway in this process referred to as cavitation.

Diagnosis:

Intradermal tuberculin skin test (TST): Using Mammalian Old Tuberculin (mOT) produced by Symbiotics, Inc. is currently the only USDA approved tuberculin for non-human primates. Intradermal injection of 0.1 ml of MOT using a 26 gauge needle in the palpebrae. In small primates, reduced dose (0.05ml) can be used. Injection sites are observed at 24, 48 and 72 hours post injection for hyperemia, edema, and induration. (Grading systems can be found in the Guidelines for the Prevention and Control of Tuberculosis in Nonhuman Primates). The test is interpreted as positive when palpebral swelling is present in conjunction with droop. A minimum of two weeks should occur between skin tests.

Detection of positive animals is difficult in early infections and in advanced stages of disease animals may be nonresponsive. An immunologically competent animal is required for the test to be effective. False positives may occur due to trauma during administration of antigen or nonspecific response caused by cross reactivity with nonpathogenic *Mycobacteria* or previous exposure to Freund's Complete adjuvant. A comparative TST - using biologically balanced purified protein derivatives (PPD) of *M. bovis* and of *M. avium* - placed into separate palpebrae or at separate sites on the abdomen is useful in differentiating nonspecific sensitization. Limitations to testing can be challenged by the quality and purity and volume of the tuberculin injected, skill in administration, thorough recording of bruise or palpebral trauma, visual access in group settings, accurate interpretation at all time periods, inadequate interval between tests or lack of documentation. All of these can jeopardize a surveillance program. Thoracic radiographs facilitate diagnosis in conjunction with additional diagnostics.

Laboratory testing: This methodology can augment TST.

PRIMAGAM (Prionics USA, Inc.) Cell mediated Immunity IFN- γ assay-fully licensed by USDA in 2007 for *in vitro* testing of cynomolgus and Rhesus macaques and tests for *M. tuberculosis*, *M. bovis* and *M. avium*. No antigen is administered to the animals so re-testing can be conducted immediately. Questionable ability to detect latent.

T-SPOT.TB (Oxford Immunotec, Oxford, UK) Cell Mediated Immunity IFN- γ assay for use in macaques. Response is to *M. tuberculosis*-specific antigens and shows some promise for the diagnosis of latent and active infections.

PrimaTB STAT-PAK Assay (Chembio Diagnostic Systems, Inc., Medford, NY) which detects IgM and IgG antibodies, rapid (20 minute) lateral flow immunoassay. Licensed by USDA in 2007 for use in nonhuman primates. Advantage test uses serum, plasma or whole blood and requires small quantity (30 μ l) although interpretation is difficult due color of blood. Test is used to detect *M. tuberculosis* and *M. bovis*. A combination of diagnostic techniques may provide for an improved diagnosis.

Material required for laboratory analysis:

Antemortem: Polymerase chain reaction (PCR) may be conducted on lesion or granuloma, feces, bronchoalveolar and gastric lavage. Culture and speciation: To optimize isolation of organisms from specimens it is recommended that the samples be centrifuged at 3,500 rpm for 30 minutes in sterile polypropylene conical tubes. Success of isolation is dependent on quality of specimen, appropriate processing and culture techniques in the laboratory. The process requires 4- 8 weeks for isolation and longer to speciate. Microbiological staining: Specimens include lesions or granulomas in lymph nodes (i.e., broncho-alveolar) and gastric lavage. Fine needle aspirates, impression smears or tissue suspensions that are air dried in a thin layer on slides that is heat fixed and stained for the appearance of acid fast bacilli.

Post-mortem: All primates euthanized or found dead should receive complete necropsies to include gross examination and histological examination of lesions including acid fast stains.

PCR, culture and staining for organism: blood, lesion or granuloma, feces, lymph nodes and bronchial or

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gastric lavage.

Products Available:

AFB Kinyoun Kit (Polysciences, Inc.): stain of slides for acid-fast bacilli.

PRIMAGAM (Prionics USA, Inc.): heparinized whole blood

T-SPOT.TB (Oxford Immunotec, Oxford, UK): heparin PBMCs

Relevant diagnostic laboratories:

National Veterinary Services Laboratories (NVSL)

1920 Dayton Ave.

Ames, Iowa 50010

Provides IFN- γ , Histopathology, Isolation and PCR. NVSL is the reference center for U.S. animal health and contribute to public health by ensuring that timely and accurate laboratory support is provided by their nationwide animal-health diagnostic system.

PCR-Zoologix Primate Diagnostics

Zoologix Inc.

9811 Owensmouth Ave., Suite 4

Chatsworth CA 91311-3800

info@zoologix.com

Treatment: Isoniazid, ethambutol and rifampin is usual starting point. However, even this combination has limited efficacy and is not recommended for tuberculous animals.

Prevention and control: Non-human primate colonies should be maintained closed and have minimal direct contact with public. Establish a routine surveillance program using the skin testing to identify infected animals; additional diagnostics may augment TST. Segregate or cull positive animals during confirmation. Identify designated quarantine area for all new nonhuman primates; hold animals for a minimum of 30 days and retest using TST. Animals of unknown source or high risk animals should be quarantined for longer duration for retest.

Suggested disinfectant for housing facilities: All primate primary housing, clinics and caging should incorporate tuberculocidal products. The Environment Protection Agency Antimicrobials Division Test oversees the testing of these products for efficacy. Consideration for product selection will depend on surfaces, caging and equipment needing tuberculocidal products.

Notification: USDA

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: It is not recommended to introduce new animals to collections holding tuberculous animals.

Conditions for restoring disease-free status after an outbreak: Cull all positive animals or treat all extremely valuable animals in isolation. Continue to conduct routine surveillance testing to include TST and other diagnostic testing. Maintain proper PPE and Occupational Health Program for all people in contact with nonhuman primates.

Experts who may be consulted:

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TUBERCULOSIS IN NON-HUMAN PRIMATES

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MYCOBACTERIOSIS (piscine)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Fish Mammal (includes humans) Reptile Amphibian	Ingestion is probably the major route of infection in fishes; other species, direct contact with infected individuals or contaminated objects. Bacteria may be found in aquatic biofilm.	Fishes: hyperemia, pale to dark coloration, morbidity, mortality; granuloma or ulcers of the skin and subcutaneous tissues.	Mild to severe in fishes, causing a wide range of gross and microscopic lesions Mild to moderate in humans (usually restricted to extremities)	Fishes: generally not attempted due to systemic nature of disease at diagnosis, poor response to treatment and zoonotic potential; long-term antimicrobial therapy with appropriate compounds can be tried. Humans: appropriate antimicrobial therapy accompanied by surgical debridement in some cases	Proper hygiene, disinfection, biosecurity, quarantine, protective apparel. Manage environment to reduce stressors on fish.	Yes

Fact Sheet compiled by: Gregory A. Lewbart and Melanie L. Church

Sheet completed on: 16 March 2011; updated 20 August 2012

Fact Sheet Reviewed by: Stephen A. Smith, Leigh A. Clayton

Susceptible animal groups: Fishes, mammals (including humans), reptiles, amphibians

Causative organism: *Mycobacterium* spp. with approximately more than 120 species recognized in the genus *Mycobacterium*. Common isolates include: *M. marinum*, *M. chelonae*, *M. neoaurum*, *M. fortuitum* and *M. haemophilum*. The organisms are Gram-positive and acid-fast staining.

Zoonotic potential: Yes; moderate

Distribution: Global and most commonly associated with aquatic environments. The optimal temperature range is 24-28° C (76-82° F). The bacteria can survive for up to 2 years in the environment.

Incubation period: Varied, weeks to months in fishes; 2 days to 6 weeks in humans.

Clinical signs:

Fishes: Chronic progressive infection is most typically reported and may include skin hemorrhage, ulcerations and granulomas and/or white nodules on viscera; hyperemia of fins; exophthalmos, corneal ulcer, granulomatous endophthalmitis; lethargy, anorexia, weight loss, abdominal edema, cutaneous edema, reduced pigmentation, loss of scales. Acute mortalities may occur with more virulent strains and animals

MYCOBACTERIOSIS (piscine)

<p>may lack substantial gross changes such as granulomas. Animals may be infected without evidence of disease.</p> <p>Humans: Usually causes a chronic infection that is limited to the extremities, such as fingers and hands. A localized skin nodule or granuloma may ulcerate and start to exude a serosanguinous or purulent discharge. Depending on immunological status of infected individual, nodular cutaneous lesions can progress to tenosynovitis, arthritis, and osteomyelitis.</p>
<p>Post mortem, gross, or histologic findings: Gross changes presented in clinical signs. Microscopically, acid-fast organisms are frequently detected in tissues and within granulomas but not all acid-fast organisms are <i>Mycobacteria</i> species. Acid-fast bacilli may be detected in both granulomatous and non-granulomatous tissues. Staining intensity can vary.</p>
<p>Diagnosis: History; signalment; clinical signs; gross lesions; acid-fast staining of tissue touch impressions; histopathology with granulomatous inflammation and acid-fast staining; microbial culture; PCR and DNA sequencing.</p>
<p>Material required for laboratory analysis: Tissue samples for touch impressions, culture, histopathology, and in some cases PCR.</p>
<p>Relevant diagnostic laboratories: Many. National Veterinary Services Diagnostic Laboratory (Ames, Iowa) for culture and sensitivity.</p>
<p>Treatment: Treatment often considered unrewarding for eliminating infection in individual fish or fish populations. Long-term antibiotic including rifampin, erythromycin, streptomycin, as examples, may be considered. Surgical excision and long term antibiotics are usually recommended in humans.</p>
<p>Prevention and control: The disease can be difficult to eradicate. Wear gloves when cleaning aquariums or handling fish. Hands should be washed thoroughly afterwards with 70% isopropyl alcohol and a bactericidal soap. In exhibit settings, may manage certain populations as positive, particularly if animals presenting infrequently with chronic disease in older individuals (consistent with opportunistic infection). Reducing environmental stressors may help reduce clinical disease.</p>
<p>Suggested disinfectant for housing facilities: Ethanol or methanol (70%).</p>
<p>Notification: None required by law.</p>
<p>Measures required under the Animal Disease Surveillance Plan: None.</p>
<p>Measures required for introducing animals to infected animal: Introductions to infected animals should be avoided.</p>
<p>Conditions for restoring disease-free status after an outbreak: Depopulate, disinfect the environment, and then monitor and test sentinel animals.</p>
<p>Experts who may be consulted: Andrew Kane, PhD Emerging Pathogens Institute Aquatic Pathobiology Laboratory University of Florida PO Box 100188 Gainesville, Florida 32610-0188 Phone: (352) 273-9090 Email: Kane@ufl.edu</p> <p>Stephen A. Smith, MS, DVM, PhD Aquatic Medicine/Fish Health Department of Biomedical Sciences & Pathobiology</p>

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TUBERCULOSIS IN UNGULATES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ungulates	Primarily aerosol and ingestion; fomites are a potential	Labored breathing, coughing, lymphadenopathy, wasting	Highly variable, generally slowly progressive to severely debilitating and fatal	Not recommended except in extraordinary cases with endangered species; wide range of antimicrobial combinations utilized based on sensitivity	Isolation and quarantine, test and cull	Yes

Fact Sheet compiled by: Douglas Armstrong and Charles O. Thoen

Sheet completed on: 13 February 2011; updated 31 January 2013

Fact Sheet Reviewed by: Claude Turcotte; John Kaneene; Paul P. Calle

Susceptible animal groups: All ungulates - ruminants and camelids - are susceptible. However, very rare in North American zoo populations due to ongoing monitoring via necropsy programs and tuberculin skin testing during interzoo animal transfer. It is more common in some farmed exotics such as cervids. Pockets of endemic infection in the US in wildlife including white tail deer, elk and bison, specifically in Michigan.

Causative organism: *Mycobacterium bovis*

Zoonotic potential: Significant zoonotic potential

Distribution: Global

Incubation period: Variable, generally slowly progressive

Clinical symptoms: Dyspnea, coughing, lymphadenopathy, lethargy, weight loss.

Post mortem, gross, or histopathologic findings:

Gross: Lesions typically will be yellowish, caseous necrotic areas within nodules of firm white, fibrous tissue. Tuberculous lesions may be accompanied by cavitation and calcification. Miliary patterns of granulomas may be present in some species. Primarily affected organs are lungs and lymphoid system, especially retropharyngeal lymph nodes. However, virtually any organ may be affected.

Histology: Tubercles are granulomatous lesions with a caseous necrotic center bordered by epithelioid cells, some of which may form multinucleated giant cells. Histologic lesions may vary substantially with species. Culture of affected tissue and polymerase chain reaction detection are useful tools in diagnosis.

Diagnosis: All zoo ungulate deaths should be necropsied with the intent to detect this disease if present. See above post mortem description.

Ante mortem: Primary assessment is by delayed hypersensitivity to *M. bovis* PPD tuberculin, 0.1 ml injected intradermally using a 26 gauge needle 3/8 inch in length. In the US, procedures and sites are defined and regulated by United States Department of Agriculture, Veterinary Services, as well as other regulations in the US and other countries. Defined testing procedures and sites for accredited veterinarians for preshipment testing or screening programs include single site caudal tail fold in true cattle species and bison; single site mid cervical region in cervids and antelope species; dorsal lateral edge of base of ear in suidae species; and the axillary region for camelidae.

In the US, comparative tuberculin skin tests can be conducted by USDA approved veterinarians using biologically balanced PPD's prepared from *M. bovis* and from *M. avium* injected in the cervical region. *In vitro* antibody tests have been described; however, the validity of these tests for detecting TB in early stages of

TUBERCULOSIS IN UNGULATES

infection has not been confirmed. Interferon gamma assay is validated and approved only for use in domestic cattle.

Material required for laboratory analysis: Mycobacteriologic examination of material from post mortem lesions or enlarged lymph nodes harvested at necropsy are needed.

Relevant diagnostic laboratories: National Veterinary Service Laboratory, USDA, http://www.aphis.usda.gov/animal_health, and some state veterinary diagnostic laboratories in the US, or similar reference laboratories in other countries.

Treatment: This route is not recommended as it is both ineffective and expensive. Particularly, it is difficult in ruminants since most recommended medications are administered by the oral route. If undertaken for animals of substantial conservation or genetic value, initial protocol of some combination of antimicrobials should be modified subsequently based on culture and sensitivity of organism from tracheal wash possible. Normally use multi-drug regimen includes combinations of isoniazid, ethambutol and rifampin.

Prevention and control: Isolation of suspected infected animals is recommended. For captive populations, and aggressive test and cull program is recommended with depopulation if disease is widespread.

Suggested disinfectant for housing facilities: Cresylics, phenolics and gluteraldehyde based disinfectants labeled to kill pathogenic *Mycobacteria*

Notification: Reportable disease to state and federal authorities

Measures required under the Animal Disease Surveillance Plan: Determined by state and federal authorities, may vary by region. Consult state and regional federal authorities in the US, or similar regulatory authorities in other countries.

Measures required for introducing animals to infected population: Not recommended

Conditions for restoring disease-free status after an outbreak: Repeated tuberculin skin testing to identify reactors and depopulate. Consult state and area federal (APHIS) livestock disease veterinary authorities in the US, or similar regulatory authorities in other countries.

Experts who may be consulted: John B. Kaneene, DVM, MPH, PhD

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**MYCOPLASMOSIS (*Mycoplasma ovipneumoniae*,
M. gallisepticum, *M. agassizi*, and others)**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Sheep, goats, birds, and tortoises most important, others possible	Direct contact between animals most important, fomites possible	Respiratory (pneumonia, coughing), conjunctivitis, polyarthritis	Tends to be chronic; can be severe and result in death.	Macrolides and fluoroquinolones may be effective early in the disease, but ineffective for polymicrobial secondary pneumonia.	Vaccination generally not effective. Health screening by culture and PCR. Prevent close contact	Maybe

Fact Sheet compiled by: Anne Justice-Allen

Sheet completed on: 25 February 2011; updated 1 April 2013; updated 2 January 2018

Fact Sheet Reviewed by: Tom Besser, Bruce Rideout

Susceptible animal groups *M. ovipneumoniae* – bighorn sheep, mountain goats, musk oxen, *M. gallisepticum* – birds especially passerines (house finches) and galliformes, *M. agassizii*, *M. testudineum* – tortoises. Many other *Mycoplasma* spp. exist and new ones are being identified in connection with disease syndromes in mammals, birds, and reptiles. *Mycoplasma mycoides* cluster – sheep, goats, cattle, others. *M. bovis* – bison and bovids. Other *Mycoplasma* spp. may be minimally- or non-pathogenic.

Causative organism: *Mycoplasma* spp. are bacteria with no cell wall and complex growth requirements making traditional culture mediated isolation difficult. Many of the organisms associated with disease have not been fully characterized because identification has been by molecular methods. *Mycoplasma mycoides* cluster organisms cause contagious bovine pleuropneumonia, contagious agalactia of sheep and goats and contagious caprine pleuropneumonia and are considered foreign animal diseases in the United States. *M. agalactiae* is the major cause of contagious agalactia of sheep and goats, but has only been reported sporadically in the United States. Within a species, some strains may vary in pathogenicity as well as in the clinical syndrome that develops. For example, some strains *M. bovis* are linked to calf pneumonia while others will generally cause mastitis. A subcategory of mycoplasmas is the hemoplasmas, obligate red blood cell pathogens such as *Mycoplasma ovis* in sheep, *M. suis* in swine, and novel species in raccoons. Disease caused by hemoplasmas is not considered to be mycoplasmosis.

Zoonotic potential: Marine mammal workers have acquired skin infections suspected to be *Mycoplasma* spp. Humans have their own complement of *Mycoplasma* pathogens and many of those may infect non-human primates.

Distribution: World-wide, often host species specific

Incubation period: 2 to 4 weeks, possibly longer

Clinical signs: Generally, mycoplasmas cause one or more of three clinical syndromes: lymphocytic pneumonia where secondary infection with additional bacteria is common (Pasteurellas for example); polyarthritis; mastitis. Additionally, otitis media and conjunctivitis may occur with some species. In bighorn sheep, and mountain goats signs typically consist of coughing, respiratory distress, otitis, sinusitis, loss of body condition; death is possible in all age classes on first exposure, death in neonates and weanlings in subsequent years, population declines, and poor recruitment. In birds the predominant sign is mild to severe conjunctivitis; death in some cases. In tortoises the predominant signs are nasal discharge (clear to mucopurulent), conjunctivitis, edema of the eyelids, infection often becomes chronic, and may end in death. A fatal multisystemic disease attributed to *M. alligatoris* has been identified in American alligators and related caimans. Additional species (black vultures, skunks, crocodiles) signs consist of polyarthritis

Post mortem, gross, or histologic findings: Epithelial hyperplasia is observed in the affected tissues, lymphoid aggregates and infiltrates which can progress to fibrosis. Lesions can become suppurative and necrotizing with secondary bacterial invasion.

**MYCOPLASMOSIS (*Mycoplasma ovipneumoniae*,
M. gallisepticum, *M. agassizi*, and others)**

Diagnosis: PCR is most reliable with several protocols available. Culture with specialized media (PPLO, SP4, Friis', modified Hayflick) may be utilized. Serology is unreliable for individual animal diagnosis but can be used for screening groups of animals. When comparing disease risk between populations, strain-typing is recommended as pathogenicity has been shown to vary between strains and cross-protection appears to be incomplete.

Material required for laboratory analysis: Tissues, especially lung, trachea, and retropharyngeal lymph nodes; deep nasal or oropharyngeal swabs or washes, middle ear swabs, and sinus swabs; joint fluid or tissue. Swabs (dacron or polyester with a plastic shaft) should be transported in PPLO, TSB with 10% glycerin or specialized mycoplasma/viral transport media (consult the laboratory) and should be sent to the lab promptly (should arrive within 72 hours) on gel ice. For PCR testing, swabs may be shipped without media in cryovials.

Relevant diagnostic laboratories:

Colorado Veterinary Diagnostic Laboratory – Colorado State University

<http://csu-cvmb.colostate.edu/vdl/Pages/default.aspx>

970-297-1281

National Veterinary Services Laboratory

http://www.aphis.usda.gov/animal_health/lab_info_services/about_nvsl.shtml

(515) 337-7266

Mycoplasma Research Lab - Dr. Mary Brown

University of Florida

(352) 294-4029

Lab Telephone: (352) 294-4094 or 294-4071

Texas Veterinary Medical Diag. Lab. – TAMU

<http://tvmdl.tamu.edu/>

(979) 845-3414

Washington Animal Disease Diagnostic Laboratory – Washington State Univ.

http://www.vetmed.wsu.edu/depts_WADDL/

(509)335-9696

Treatment: Azithromycin, erythromycin, tulathromycin; enrofloxacin; beta lactam antibiotics are not effective due to an absent cell wall.

Prevention and control: Population testing with blocking or competitive ELISA is most appropriate for non-domestic species where other serology methods such as AGID or SN have not been validated. Prolonged quarantine as stress increases shedding and repeated attempts at isolation during this interval are recommended. For positive populations, strain-typing should be conducted as isolates can vary in pathogenicity.

Suggested disinfectant for housing facilities: Mycoplasmas are susceptible to most commonly used disinfectants, including Virkon S, quaternary ammonium compounds, or household bleach (1:20 in water). Bleach and to a lesser extent, quaternary ammonium compounds are inactivated in the presence of organic matter so are preferably used on clean surfaces. *Mycoplasma* doesn't survive well in dry conditions or with exposure to sunlight. Some species of *Mycoplasma* will have increased survival in conditions where biofilms develop.

Notification: *Mycoplasma mycoides* cluster organisms cause contagious bovine pleuropneumonia, contagious agalactia of sheep and goats, and contagious caprine pleuropneumonia, all of which are foreign animal diseases in the United States. Immediate notification of USDA and state agencies is required for any suspected cases.

Measures required under the Animal Disease Surveillance Plan: None

**MYCOPLASMOSIS (*Mycoplasma ovipneumoniae*,
M. gallisepticum, *M. agassizi*, and others)**

Measures required for introducing animals to infected animal: Multiple negative cultures/PCR tests from infected animal. Chronic and subclinical carriers highly likely.

Conditions for restoring disease-free status after an outbreak: Difficult to impossible, long-term treatment with appropriate systemic antibiotic; see above. Test-and-cull showing promise experimentally for *M. ovipneumoniae* in bighorn sheep and has been used in other species. Multiple tests should be conducted on individual animals as shedding of organisms is inconsistent.

Experts who may be consulted:

Thomas E Besser, DVM PhD

Professor and Rocky Crate D. V. M. and Wild Sheep Foundation Chair in Wild Sheep Disease Research
Washington State University College of Veterinary Medicine

Phone: 509-335-6075

tbesser@vetmed.wsu.edu

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OESOPHAGOSTOMIASIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Old World monkeys, great apes; ruminants, camelids, and suids.	Fecal-oral: ingestion of third-stage larvae (L3); direct life cycle.	Diarrhea, anorexia, weight loss, lethargy, and abdominal pain.	Variable, but severe infection can result in death.	Ivermectin, pyrantel pamoate, or a benzimidazole. Surgical removal of mass effect.	Quarantine of new individuals; isolation of affected animals; parasite monitoring programs.	Yes, only for some parasite species that infect non-human primates.

Fact Sheet compiled by: Ginger L. Takle; updated by Karen Terio

Sheet completed on: 21 June 2011; updated 13 September 2013, updated 24 May 2018

Fact Sheet Reviewed by: Stephanie McCain

Susceptible animal groups: Great apes, Old World monkeys, suids, camelids, ruminants

Causative organism:

Primates: *Oesophagostomum bifurcum*, *O. (Conoweberia) apiostomum*, *O. (Conoweberia) stephanostomum*, *O. aculeatum*

Ruminants: *O. columbianum*, *O. venulosum*, *O. radiatum* and other species may be found in wild ruminants

New and Old World camelids: *O. venulosum*, *Oesophagostomum* sp.

Suids: *O. dentatum*, *O. brevicaudum*, *O. quadrispinulatum*, and other species may be found in wild suids.

Zoonotic potential: Yes (*Oesophagostomum bifurcum* and *O. stephanostomum*).

Distribution: Worldwide, but most commonly occurs in the tropics and subtropics.

Incubation period: Ova passed in feces hatch and develop into infective L3 in approximately 2-7 days, depending on environmental conditions. After ingestion, the L3 burrow into the intestinal wall forming cystic nodules to granulomas within the submucosa, muscularis and mesentery in which the nematodes molt into fourth-stage larvae (L4). The L4 can then remain in the nodules or return to the intestinal lumen where they develop to the adult stage. Generally, pre-patent period is considered 32-42 days.

Clinical signs:

Primates: Clinical signs can range from intermittent diarrhea to inappetence, severe mucoid bloody diarrhea, pale mucous membranes, weakness, lethargy, weight loss, vomiting, abdominal pain and death.

Ruminants and Suids: fetid diarrhea, anorexia, weakness, emaciation, and death. If chronic infection is present, clinical signs may be seen that are consistent with decreased intestinal motility, stenosis, or intussusception.

Post mortem, gross, or histologic findings: Oesophagostomins are also known as nodular worms due to their gross appearance. The L3 penetrate deep into and encyst in the lamina propria, submucosa, muscularis of the small and large intestine and in some cases the adjacent mesentery. Granulomas (nodules) form around the larvae and can be 5-50mm in diameter. These granulomas may contain reddish brown fluid and a central nematode. In some sections, inflammation is associated with migration tracts and abdominal adhesions or peritonitis may be present. Mesenteric lymph nodes are often enlarged.

Diagnosis: Identification of ova on fecal examination but these are confused easily with 'hookworm' eggs; identification of larvae or adults during intestinal biopsy; morphological identification of adult specimens collected at necropsy, PCR, PCR-RFLP, semi-nested PCR

Material required for laboratory analysis: Fecal sample, larvae or adult worms, nodular intestinal tissues.

OESOPHAGOSTOMIASIS

<p>Relevant diagnostic laboratories: Any diagnostic laboratory with routine parasitologic capabilities should be able to diagnose this infection. These diagnostics are readily available, as in-house fecal flotation or any laboratory performing fecal exams.</p>
<p>Treatment: Ivermectin, pyrantel pamoate, or benzimidazole can be administered. Where possible, surgical excision of the nodules may be performed.</p>
<p>Prevention and control: Quarantine of new animals, parasite monitoring program, isolation and treatment of affected animals, proper sanitation and waste removal can assist with prevention. Free-living larval stages (L1- infective L3) survive in the environment (moisture and temperature dependent).</p>
<p>Suggested disinfectant for housing facilities: Commonly used disinfectants can be used after removal of feces from the area.</p>
<p>Notification: None.</p>
<p>Measures required under the Animal Disease Surveillance Plan: None.</p>
<p>Measures required for introducing animals to infected animal: Treat infected or potentially infected animals prior to introduction to non-infected animals.</p>
<p>Conditions for restoring disease-free status after an outbreak: Negative fecal examinations can be used to identify persistent infections that should be resolved before introductions.</p>
<p>Experts who may be consulted: Parasitologists are well versed in this disease and should be consulted.</p>
<p>References:</p> <ol style="list-style-type: none"> 1. Oesophagostomiasis [Internet]. Center for Disease Control; c 2017 [cited 24 May 2018]. Available from http://www.dpd.cdc.gov/dpdx/html/oesophagostomiasis.htm 2. Boomker J. Parasites of South African wildlife-V. A description of the males of <i>Oesophagostomum mocambiquei</i> Ortlepp, 1964 from warthogs, <i>Phacochoerus aethiopicus</i> (Pallas, 1766). Onderstepoort J Vet Res. 1990;57:169-173. 3. Helminths, In, Bowman, DD (ed.). Georgis' Parasitology for Veterinarians, 8th ed. St. Louis (MO): Saunders; 2003. p. 115-118. 4. Gasser R, De Gruijter J, Polderman A. Insights into the epidemiology and genetic make-up of <i>Oesophagostomum bifurcum</i> from human and non-human primates using molecular tools. Parasitol. 2006;132:453-460. 5. Krief S, Jamart A, Mahé S, Leendertz F, Mätz-Rensing K, Crespeau F, Bain O, Guillot J. Clinical and pathologic manifestation of oesophagostomosis in African great apes: does self-medication in wild apes influence disease progression. J Med Primatol. 2008;37:188-195. 6. Krief S, Vermeulen B, Lafosse S, Kasenene J, Nieguitsila A, Berthelemy M, L'Hostis M, Bain O, Guillot J. Nodular worm infection in wild chimpanzees in western Uganda: a risk for human health? PLoS Negl Trop Dis. 2010;4:e630. 7. Terio KA, Lonsdorf EV, Kinsel MJ, Raphael J, Lipende I, Collins A, Li Y, Hahn BH, Travis DA, Gillespie TR. Oesophagostomiasis in non-human primates of Gombe National Park, Tanzania. Am J Primatol. 2018;80(1). Available from doi: 10.1002/ajp.22572.

OPHIDIAN PARAMYXOVIRUS (OPMV)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All snakes, especially Viperidae	Primarily airborne but fomite, waterborne, fecal-oral transmission can occur. Vertical transmission may occur, but uncertain at this time.	Respiratory compromise ; neurologic signs; anorexia, regurgitation, and chronic “poor doer”; and sudden death	Severe; animals can survive infection with supportive care but death is common	Supportive care, including broad spectrum antibiotics, fluid and nutritional support, and ensure proper husbandry, especially correct thermal gradient	Strict quarantine with separate airspace and utensils, PCR testing on tracheal lavage, and proper husbandry	No

Fact Sheet compiled by: Christopher S. Hanley

Sheet completed on: 19 November 2011; updated 1 April 2013; updated 8 Feb 2018

Fact Sheet Reviewed by: Randy Junge

Susceptible animal groups: All snakes, especially Viperidae

Causative organism: Ophidian paramyxovirus

Zoonotic potential: None

Distribution: Outbreaks have been documented in multiple private and zoological collections worldwide; seroconversion has been documented in wild specimens and importance of which remains uncertain. Outbreaks are more common from January through May.

Incubation period: Seroconversion takes at least 8 weeks but the incubation period has been documented to be at least 10 months in some specimens.

Clinical signs: Acute death, respiratory compromise, blood in the oral cavity or nares, neurologic signs, including head tremors, star gazing, flaccid paralysis, convulsions, and loss of righting reflex. In more chronic cases, anorexia, regurgitation, cachexia, lethargy, and abnormal feces are common. As with other paramyxoviruses, OPMV causes immunosuppression, so secondary infections are common.

Post mortem, gross, or histologic findings: No pathognomonic lesions occur with this disease. Gross findings range from no lesions to respiratory lesions including pulmonary congestion, hemorrhage, respiratory exudates, and pneumonia, pancreatic hyperplasia, and hepatic necrosis and granulomas may all be macroscopically evident. Histologic lesions include hyperplasia of the respiratory epithelium, thickening of the pulmonary septa, inflammatory cell infiltration, evidence of exudates and edema, and rarely eosinophilic intracytoplasmic inclusions. If the CNS is involved, there can be encephalitis with multifocal gliosis, moderate ballooning of axon fibers in the brain stem and spinal cord. Hepatic necrosis or multifocal pyogranulomatous inflammation is often observed. Hyperplasia of pancreatic ducts and acinar cells with cystic dilatation has been observed. The salivary glands can be affected by ductular dilatation, flattening of the ductular epithelium, and accumulation of cellular debris and secretory material in the lumen.

Diagnosis: Definitive diagnosis requires viral isolation from tissues, PCR for viral nucleic acid, immunohistochemical staining for viral antigen, and/or electron microscopy. Tracheal lavages submitted for PCR analysis may provide an antemortem diagnosis.

Material required for laboratory analysis: Tracheal lavage fluid can be submitted for PCR analysis as a screening tool and tissue samples collected at necropsy (especially lung, liver, and pancreas) – both formalin fixed and frozen, depending on test.

Relevant diagnostic laboratories:

University of Florida –2015 SW 16th Avenue, Bldg 1017, Room V2-238, Gainesville, FL 32608 – (352) 392-4700 ext. 5775

OPHIDIAN PARAMYXOVIRUS (OPMV)

<p>Treatment: Supportive care, including broad spectrum antibiotics, fluid and nutritional support, and ensure proper husbandry, especially correct thermal gradient.</p>
<p>Prevention and control: Maintain proper husbandry, especially correct thermal gradients. Quarantine all new animals for a minimum of 60-90 days, using separate utensils and supplies, disinfection or destruction of all materials at the end of quarantine, and usage of a footbath. Obtain OPMV PCR via tracheal wash during quarantine period. Monitor animals closely for abnormal behaviors. Necropsy all animals that die. While OPMV serology is available from multiple laboratories, question has been raised as to the value of this method of testing, especially when comparing results between different laboratories. If used, as with any other antibody titer, serial sampling is required to confirm infection versus just exposure.</p>
<p>Suggested disinfectant for housing facilities: Bleach is recommended for disinfection at 1/2 cup/gallon of water [120ml/L].</p>
<p>Notification: None</p>
<p>Measures required under the Animal Disease Surveillance Plan: None</p>
<p>Measures required for introducing animals to infected animal: Due to the fact that PCR positive animals would be actively shedding the virus, it is not recommended to introduce new animals to those that are infected. A minimum of 60 days (and ideally longer) after the last OPMV death should pass before the introduction of new specimens.</p>
<p>Conditions for restoring disease-free status after an outbreak: PCR positive animals should be isolated and those that are PCR negative should appear healthy and have negative tracheal washes at least 90 days after diagnosis before new animals are introduced into the collection.</p>
<p>Experts who may be consulted: Jim Wellehan, DVM, MS, PhD, DACZM, DACVM (Virology, Bacteriology/Mycology) Zoological Medicine Service University of Florida College of Veterinary Medicine Gainesville, Florida 32610-0126 wellehanj@ufl.edu</p>
<p>References</p> <ol style="list-style-type: none"> 1. Allender MC, Mitchell MA, Dreslik MJ, Phillips CA, Beasley VR. Measuring agreement and discord among hemagglutination inhibition assays against different ophidian paramyxovirus strains in the Eastern massasauga (<i>Sistrurus catenatus catenatus</i>). J Zoo Wild Med. 2008; 39:358-361. 2. Bronson E, Cranfield MR. Paramyxovirus. In: Mader DR (ed.). Reptile medicine and surgery, 2nd ed. St. Louis (MO): Elsevier; 2006. p. 851-861. 3. Ophidian Paramyxovirus (OPMV) [Internet]. Microbiology, Parasitology & Serology UF Diagnostic Laboratories, College of Veterinary Medicine. c2018 [cited 2018 February 08]. Available from http://labs.vetmed.ufl.edu/available-tests/zoo-med-infections/opm/ 4. Pees M, Schmidt V, Marschang RE, Heckers KO, Krautwald-Junghanns ME. Prevalence of viral infections in captive collections of boid snakes in Germany. Vet Rec. 2010;166:422-425.

OXYURIASIS (“pinworm”)

(*Alaeuris*, *Aspicularis*, *Dentostomella*, *Enterobius*, *Oxyuris*, *Probstmayria*, *Passalurus*, *Skrjabinema*, *Syphacia* and *Trypanoxuria* spp.)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals; including humans; reptiles	Fecal-oral by ingestion of eggs; retroinfection by anal entry possible with some species and short pre-patent period	Asymptomatic to substantial irritability as anusitis and pruritus. Equids with severe infection often present with broken tail hairs or peri-anal excoriation or trauma.	Typically mild, but may become highly aggravating to horses and humans.	Anthelmintics, e.g. ivermectin, fenbendazole. Hygiene to prevent re-infection	Personal/environmental hygiene; quarantine of new arrivals and treatment as necessary.	<i>Enterobius</i> is probably transmissible between apes and humans. Zoonotic potential of others are not known.

Fact Sheet compiled by: Christopher J. Bonar

Sheet completed on: 14 February 2011; updated 16 April 2013, updated 2018

Fact Sheet Reviewed by: Kate Pritchett, Christie Hicks

Susceptible animal groups: Mammals, including humans; reptiles, (+/-) avian.

Causative organism: *Enterobius* spp. infects humans and chimpanzees. *Trypanoxuria* and *Enterobius* can also cause disease in New World primates. *Probstmayria vivipara*, *Skrjabinema ovis*, and *S. caprae* in sheep and goats. *Oxyuris equi* infects equids, and *Oxyuris karamoja* infects African rhinoceroses and elephants. *Passalurus ambiguous* is common in the colon and cecum of lagomorphs. *Dentostomella* spp., *Syphacia* spp. and *Aspicularis tetraptera* infect laboratory rodents, although no oxyurid is described in guinea pigs. *Alaeuris brachylophi* has been described in reptiles. *Oxyuronema atelophorum* has been reported in monkeys of the genus *Ateles*.

Zoonotic potential: Yes

Distribution: Parasite is found occasionally in wild and captive chimpanzees, elephants, rhinos, equids, reptiles, domestic and laboratory rodents, and humans.

Incubation Period: *Aspicularis tetraptera*: prepatent period 23 days. *Enterobius vermicularis*: prepatent period 30 days. *Passalurus ambiguous*: prepatent period 56-64 days. *Syphacia muris* prepatent period 8 days. *Syphacia obvelata*: prepatent period 11-15 days.

Clinical signs:

Humans: Perineal and anal pruritus, often worse in the evenings, when oxyurids emerge to lay eggs on the perineum.

Animals: Irritability, anal pruritus, occasionally gastrointestinal impaction in reptiles.

Post mortem, gross, or histologic findings: Parasites are most commonly found incidentally on routine fecal ova and parasite examination, but rarely do they occur in large enough numbers to cause noticeable gross pathology. Occasional reports of infections in lizards and turtles severe enough to cause gastro-intestinal impaction. Hemorrhagic enteritis has been reported in *Ateles* spp. infected with *Oxyuronema atelophorum*.

Diagnosis: “Scotch tape preparation” from anus/perineum, routine fecal O&P examination (floatation). Examination of cecal and colonic contents at necropsy. PCR amplified DNA has recently been demonstrated to be more sensitive than fecal O&P examination.

Material required for laboratory analysis: Egg masses from perineum or from fecal examination or worms and eggs recovered from cecal or colonic contents at necropsy.

Relevant diagnostic laboratories:

Any laboratory equipped with light microscopy and basic supplies for fecal O&P examination can detect oxyurids. PCR capabilities are a useful adjunct.

Treatment: Pyrantel, avermectins, and benzimidazoles are all effective against oxyurids. Fenbendazole

OXYURIASIS (“pinworm”)

(*Alaeuris*, *Aspiculuris*, *Dentostomella*, *Enterobius*, *Oxyuris*, *Probstmayria*, *Passalurus*, *Skrjabinema*, *Syphacia* and *Trypanoxuria* spp.)

medicated feed is commonly used for laboratory rodents.

Prevention and control: Detection on routine fecal ova and parasite examinations, and on scotch-tape preparations on symptomatic individuals, and follow-up treatment on infected individuals should allow for control of the organisms. Good hygiene of enclosures should help to prevent re-infection. Eggs have been shown to be present on laboratory workers hands as well as in the dust found around cages, on air vents, and in animal room ventilation systems. Filter-top cages or individually ventilated cages, therefore, may be useful to prevent airborne transmission in laboratory rodent facilities. Strict sanitation and hygienic measures should be adequate to prevent zoonotic transmission of *Enterobius* spp. between apes and man.

Suggested disinfectant for housing facilities: Eggs may be resistant to routine disinfectants. Heat disinfection of cages and cleaning implements (100°C) is effective in killing eggs. Chlorhexidine (0.5%), formaldehyde vapors, and 75 mg/L chlorine dioxide have also been recommended. Mechanical removal by washing and scrubbing of enclosures, and heat disinfection, where possible, is recommended.

Notification: This disease is not ordinarily reportable.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Routine anthelmintic treatments.

Conditions for restoring disease-free status after an outbreak: Repeated treatment of individual animals and conspecifics, as well as sanitation measures to prevent re-infection via the fecal-oral route should eventually be effective.

Experts who may be consulted:

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MENINGEAL WORM (*Parelaphostrongylus tenuis*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ungulates, notably cervids	Oral - Ingestion of infected intermediate host which includes numerous terrestrial mollusk species (i.e., snails and slugs)	Neurologic	Ranges from mild lameness to recumbency and death. Severity is typically worse in young animals and may vary between species.	High doses of anthelmintics combined with antiinflammatories; supportive therapy	Prophylactic anthelmintic administered every 4-6 wks; exclusion of the natural host (white-tailed deer); elimination or control of mollusk population	No

Fact Sheet compiled by: Rae Gandolf and Julie Ter Beest

Sheet completed on: 1 January 2011; updated 9 October 2012

Fact Sheet Reviewed by: Priya Bapodra

Susceptible animal groups:

Natural host: The white-tailed deer (*Odocoileus virginianus*) serves as the natural host and is rarely clinically affected; they can shed numerous dorsal-spined larvae in their feces. Approximately 80% of white-tailed deer are infected in endemic regions.

Aberrant or dead-end hosts: Other cervid species (moose, caribou, mule deer, elk, Sika deer); camelids (camels, llamas, alpacas); pronghorn; some bovids (many antelope species, bighorn sheep, Angora goats, bison, rarely domestic cattle); and rarely equids (reported in domestic horses) may show severe clinical signs. Overall, these species rarely shed larvae in their feces.

Disease significance: Mortalities in captive species; failed reintroduction of cervid species such as caribou; suppression of elk and moose populations; suspected cause of moose population declines in central and eastern North America.

Causative organism: *Parelaphostrongylus tenuis*, an extrapulmonary lungworm nematode

Life cycle: The natural host (white-tailed deer) acquires the infection through accidental ingestion of mollusks infected with 3rd stage larvae. The larvae migrate from the gastrointestinal tract along spinal nerves and into the spinal cord where they develop to the last larval state. Adult worms then locate on the meninges and in the cranial venous sinuses where they lay eggs. The eggs pass into the venous circulation, develop into 1st stage larvae in lung capillaries, and then migrate into the lung tissue. These larvae are expectorated, swallowed, and passed in the feces. Mollusks acquire larval infection when crawling over feces and the parasite develops into the infective 3rd stage larvae within this intermediate host.

In the aberrant host, infection is acquired by the same route. However, migration of the larvae in the spinal cord tends to be non-directional and larvae often die before reaching the brain. The aimless migration and larval death result in more local tissue damage as compared to the natural host. Larvae infrequently develop into reproductive adults in the aberrant host.

Zoonotic potential: None reported

Distribution: Predominantly associated with deciduous and deciduous-coniferous forests of eastern and central North America, concurrent with white-tail deer populations. It is uncertain why deer of the southeast coastal plains region and of western North America are not infected.

MENINGEAL WORM (*Parelaphostrongylus tenuis*)

Incubation period:

Natural host: pre-patent period 82-137 days, inversely proportional to infection dose.

Aberrant host: signs typically appear in 30-60 days, as short as 5 days reported in experimental infections.

Clinical signs: Neurologic signs are associated with intracranial or spinal cord inflammatory lesions caused by parasite migration. Signs may range from single limb lameness or rear limb weakness to head tilt, ataxia, circling, blindness, progressive loss of motor function and death. Ocular symptoms associated with migration of larvae into the uvea have been reported.

Post mortem, gross, or histologic findings Lesions in the aberrant host consist primarily of histologic changes in the brain and spinal cord. They may include meningitis and encephalitis; perivascular cuffing and infiltrations of eosinophils, lymphocytes, and plasma cells; calcified remains of worms; worm tracks; focal traumatic malacia caused by developing nematodes; gliosis; disruption of the ependyma; neuronal and myelin degeneration. Eggs and larvae may be found associated with the eyes or the roots of cranial nerves, on the leptomeninges, and in brain tissue.

Diagnosis:

Natural host: Modified Baermann technique for retrieving 1st stage larvae from feces. Larvae must then be differentiated from related species using PCR. However, there are limited species of dorsal-spined larvae and they are easy to retrieve, allowing for presumptive diagnosis. In addition to white-tailed deer, moose and elk may shed the larvae in low numbers.

Aberrant hosts: Ante-mortem diagnostic testing is currently unavailable; a serum ELISA is under development at the University of Tennessee, aimed at detecting antibodies against 3rd stage larvae in cervid species. Post-mortem diagnostics include PCR on tissues collected at necropsy, post-mortem recovery of adult worms, or identification of larvae in neurologic tissue.

Material required for laboratory analysis: Post mortem: spinal cord and brain

Antemortem: plasma or serum (aberrant hosts), feces (white-tailed deer, moose and elk)

Relevant diagnostic laboratories:

ELISA (in development) or PCR (tissue): University of Tennessee, College of Veterinary Medicine, Department of Biomedical and Diagnostic Sciences, Knoxville, Tennessee, USA

Treatment: High dose fenbendazole (20-50mg/kg orally once daily for 5 days) and or high dose ivermectin (0.3-0.4mg/kg SC daily for 3-5 days), or levamisole, in addition to supportive therapies including non-steroidal or steroidal anti-inflammatory drugs, vitamin E, and vitamin B complex. Early initiation of treatment is key to success.

Prevention and control:

Captive species: Administration of anthelmintics every 4 -6 weeks to target 3rd stage larvae before they migrate to neural tissue; minimize exposure of captive animals to mollusks by establishing gravel roads or other vegetation breaks to act as snail and slug barriers; use molluscicides with caution due to potential for environmental toxicity; allow non-susceptible species to initiate grazing on new or overgrown pastures; reduce white-tailed deer population and build fences to exclude them.

Free-ranging species: Control of white-tailed deer population to reduce exposure.

Suggested disinfectant for housing facilities: Molluscicides (copper sulfate, metaldehyde, sodium pentachlorophenate) may be used against the intermediate host with caution, as they are potential environmental toxins.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: As no direct transmission of the parasite occurs, and species susceptible to clinical disease do not typically pass larvae, infected animals do not pose a direct threat to un-infected animals. However, white-tailed deer should generally be considered as infected, and exposure of susceptible species to white-tailed deer should be avoided as possible.

MENINGEAL WORM (*Parelaphostrongylus tenuis*)

Conditions for restoring disease-free status after an outbreak: This disease is endemic in white-tailed deer populations of eastern North America.

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Carnivores including: felids, mink, canids, procyonids, viverrids, mustelids, ursids, hyaenids, and possibly sea lions; other species are affected by species-specific parvoviruses, but the diseases differ substantially from the carnivores	Ingestion, primarily (fecal-oral)	Canine parvovirus-2 (CPV-2) and panleukopenia virus: most common signs are vomiting and diarrhea that can result in dehydration and death, immune-suppression is also common. In some cases no clinical signs occur. Respiratory or neurologic signs also can be seen with panleukopenia virus	Non-clinical or mild to severe including death; depends on immune status of animal	Symptomatic treatment to prevent dehydration and prevent or treat secondary bacterial infections	Vaccination when possible; environmental sanitation, prevention of contamination of environment, strict isolation of naïve populations; control can be managed with quarantine for at least 30 days	No

Fact Sheet compiled by: Tara M. Harrison

Sheet completed on: 3 August 2011; updated 7 September 12; updated 6 February 2018

Fact Sheet Reviewed by: Dalen Agnew, Rebecca Smedley, Roger Maes

Susceptible animal groups: Carnivores, such as felids, canids, procyonids, mustelids, ursids, hyaenids, and viverrids, are affected by those parvoviruses described on this fact sheet. Many other groups of animals are affected by various parvoviruses but the disease differs significantly from these disease presentations.

Causative organism: Canine parvovirus type-2 and Feline parvovirus (panleukopenia virus) are discussed here. (Other parvoviruses include but are not limited to: canine parvovirus type 1 (minute virus of canines), mink enteritis virus, mink Aleutian disease parvovirus, ferret Aleutian disease virus, raccoon parvovirus.)

Zoonotic potential: None

Distribution: World-wide distribution

Incubation period: Typically 5-7 day incubation period, but can range from 4-10 days

Clinical signs: For canine and other enteric parvoviruses, puppies are most likely to suffer severe disease and death. However any unvaccinated canid, of any age, can become infected with CPV-2. Clinical signs range from non-clinical to profound depression, lethargy, and inappetence; enteric parvoviruses cause signs of gastroenteritis such as vomiting and severe diarrhea that can be foul-smelling and include mucus, fibrin casts, and blood; may also see pyrexia and dehydration.

Other parvoviruses cause variable disease syndromes such as chronic wasting or neurologic disease seen with (mink) Aleutian disease, or respiratory, neurological, and/or gastrointestinal disease seen with panleukopenia. Most parvoviruses also cause immunosuppression. Most animals that succumb do so within 4-5 days of infection; juveniles have a higher fatality rate than adults.

Clinical pathological, gross, and histopathological findings: Feline parvovirus (panleukopenia): total white counts of ≤ 1000 -2000/ml and neutrophils < 200 /ml. Canine, feline, and other enteric parvoviruses: anemia and hypoproteinemia possibly due to blood and protein loss through the gastrointestinal tract; segmental reddening, hemorrhage, and a granular appearance of the serosa of the small intestine; lymphoid depletion; necrosis and loss of crypts with subsequent villous atrophy, blunting and fusion in the small intestine; involution of the lymphoid tissues in the small intestine, lymph nodes, spleen and thymus; bone marrow may be hypocellular.

Diagnosis: Testing should be performed on acutely infected or recently exposed or high risk canines. In-house fecal ELISA tests are quite specific and sensitive for currently circulating strains of canine parvovirus, and are reportedly useful for feline panleukopenia. Some strains of CPV-2 can infect domestic cats as well as dogs. As

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with any test, false results are possible, and negative results can occur later in the course of disease. Weak false positives may also reportedly occur due to recent vaccination. However, this is uncommon, so positive results should be taken seriously even in recently vaccinated dogs. Shedding can be intermittent, therefore testing more than one animal, or one animal on sequential days is suggested. Other possible tests are latex agglutination, and hemagglutination which is not a specific test. Virus can be detected in serum by hemagglutination and IgG IFA; virus may be detected in tissues using virus isolation, hemagglutination, PCR, immunohistochemistry (IHC) and electron microscopy. Serology is a useful tool to assess risk or to further clarify the need for quarantine of individual dogs. Necropsy (both gross and histopathology) are very useful, especially in a “herd” health situation when mortality is present. A presumptive diagnosis may be made from characteristic gross and histologic lesions and confirmed with positive IHC labeling within the damaged small intestinal crypts and in the epithelial cells of the tongue; tongue can be useful for PCR, IHC, or FA testing in canines and felines if there is marked autolysis in the small intestine.

Material required for laboratory analysis: Enteric parvoviruses: feces, small intestine >> tongue, systemic lymphoid tissues. Serum can also be used but may not be helpful.

Relevant diagnostic laboratories: Most diagnostic laboratories can test for enteric parvoviruses; several in-house diagnostic tests are also available for enteric parvovirus.

Treatment: Enteric parvoviruses: isolate infected animal; provide supportive care to treat dehydration and electrolyte imbalance; prevent secondary bacterial infections, especially in animals with leukopenia.

Prevention and control: Parvoviruses can survive for months in cool, moist areas protected from sunlight, and are very stable when frozen; can persist in feces for 6 months at room temperature and may remain viable in the natural environment for 9-12 months. Vaccination is the cornerstone of parvovirus prevention. In the absence of maternal antibody interference, a single modified live vaccine can confer protection within 3-5 days. Re-vaccination must be performed, especially in high risk situations such as shelters. Both inactivated-adjuvanted and modified live vaccines are available, although the use of modified live vaccines in non-domesticated animals may produce disease and is typically not recommended. Although, modified live vaccines against parvovirus were used in red wolves and produced titers for three years and no adverse reactions.

Suggested disinfectant for housing facilities: Parvovirus must be mechanically removed or can be killed by one of the few effective disinfectants. Disinfection using formaldehyde, glutaraldehyde, potassium peroxymonosulfate (Trifectant or Virkon-S), or chlorine solutions, such as 0.1755% sodium hypochlorite solution; for bleach, 5% household bleach can be used but should be freshly diluted at **1:32** (1/2 cup per gallon). Foot baths can also be used with sodium hypochlorite solutions to prevent spread. Like all disinfectants, bleach must be used and stored correctly to be effective. Application should be only to pre-cleaned surfaces free of organic matter. Independent studies have shown that quaternary ammonium disinfectants do not reliably kill parvovirus.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Do not introduce non-infected animal to infected animal; non-infected animal should be vaccinated prior to exposure to previously infected animal whenever possible.

Conditions for restoring disease-free status after an outbreak: Adequate husbandry practices using an approved disinfectant; in many environments parvovirus may be endemic and it may be difficult to restore a disease-free environment. If the virus is present endemically in other wildlife vectors or in a captive setting, appropriate vaccination and disinfection may assist in controlling potential outbreaks. Quarantine of infected animals for a minimum of 30 days may also help to control an outbreak and restore a disease-free state. References below include information for less common parvoviruses.

Experts who may be consulted:

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PASTEURELLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals Avian	Aerosol, mechanical via bite or scratch wounds, or environmental (food, water). Colonization of lungs by endogenous nasopharyngeal bacteria is described in ruminants and swine due to environmental stressors and/or primary infections due to viruses or <i>Mycoplasma</i> spp.	Primarily depression, fever, coughing, nasal and oral discharge, increased respiratory rates, tachypnea. Arthritis, gastrointestinal disease, otitis media, mastitis, bite wound abscesses and other signs are possible.	Variable. Ranges from subclinical to peracute and fatal.	Supportive care, early intervention with antibiotics, ideally based on antibiotic sensitivity. Drainage of localized abscesses. Organ specific treatment for systemic infections. Peracute systemic infections may be unresponsive.	Sanitation, quarantine, optimization of animal health and management, and minimization of environmental and social stressors. Vaccination for viral respiratory agents that can predispose to pasteurellosis. Some strains may be responsive to vaccination.	Yes, but rare

Fact Sheet compiled by: Glen C. Weiser, David S. Miller, and Susan M. Lindstedt

Sheet completed on: 1 March 2011; updated 1 October 2012, Jan 2018

Fact Sheet Reviewed by: Robert E. Briggs, James J. England, Jack C. Rhyan

Susceptible animal groups: Most notably ruminants and birds, but members of the Pasteurellaceae family can cause disease in many farm, companion and wild animals.

Causative organism:

Members of the Pasteurellaceae family. In ruminant pneumonia, mostly *Mannheimia haemolytica*, *Bibersteinia trehalosi*, *Histophilus somni*, and *Pasteurella multocida* are involved. While each organism is capable of causing systemic and septicemic disease, prominently *P. multocida* in association with avian cholera or hemorrhagic septicemia. In some cases, the incidence of *M. haemolytica* may be underestimated due to proximity dependent inhibition by other organisms (Dassanayake et al., 2010; Bavananthasivam et al., 2012), although this has only been shown *in vitro*. It can be a primary infection, particularly in avians, or secondary to viral or *Mycoplasma* spp. infections and stress. Recent data from free ranging bighorn sheep suggest that *Mycoplasma ovipneumoniae*, rather than Pasteurellaceae, may play a primary role in epizootic pneumonia and predispose to secondary Pasteurellaceae infection (Besser et al., 2012). In free ranging bighorn sheep, lambs appeared more susceptible to pasteurellosis than adults, and β -hemolytic isolates were more likely to be associated with respiratory disease in adults (Miller et al., 2012).

Zoonotic potential: Yes, but rare, primarily in severely immunocompromised individuals. Cat bite infections are more common.

Distribution: Ubiquitous

Incubation period: Various reports indicate 1-8 days, although some strains are carried asymptotically for prolonged periods.

Clinical signs: Serous oculonasal discharge, cough, depression, anorexia, fever, pneumonia, tachypnea, dyspnea. Arthritis, otitis media, gastrointestinal disease, and other signs are possible, particularly with chronic and systemic infections. Localized abscesses in rabbits and cats due to bite wounds.

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<p>Postmortem, gross, or histologic findings: Highly variable, but lesions are most common in the thoracic cavity. Gross signs include pleural effusion, fibrinous adhesions, hemorrhage, necrosis, pulmonary consolidation, thickened interlobular septa, hydropericardium, multifocal liver lesions, and abscesses. Histopathologic lesions include hyperemia, pneumonitis, fibronopurulent bronchopneumonia, coagulative necrosis, and fibrinous pleuritis.</p>
<p>Diagnosis: Bacterial culture. PCR detection methods are available. Laboratories with specific expertise in pasteurellosis for disease investigations. Concurrent testing for respiratory viruses and <i>Mycoplasma</i> spp. is recommended.</p>
<p>Material required for laboratory analysis: Nasal and/or oropharyngeal swabs, tonsillar tissue, lung tissue, or other infected tissues.</p>
<p>Relevant diagnostic laboratories: Most veterinary diagnostic laboratories can complete analysis. In cases involving wildlife, labs with specific wildlife experience should be consulted. Wildlife Pasteurellaceae can differ from domestic animal isolates.</p>
<p>Treatment: Rapid quarantine of infected individuals, prompt administration of appropriate antibiotics (ceftiofur, oxytetracycline, penicillins, florfenicol, enrofloxacin, tilmicosin, azithromycin, or based on susceptibility testing), reduction of stressful environmental and social conditions, general supportive care. Drainage of abscesses or other therapy specific to the clinical presentation.</p>
<p>Prevention and control: Sound management practices (including minimization of stressors, nutritional and environmental control, and vaccination for viral respiratory agents), quarantine of affected animals that prevents fence line and close aerosol contact, quick treatment, or in advanced cases, euthanasia. Specific vaccination is practiced for septicemic disease, avian cholera, atrophic rhinitis, and bovine respiratory disease.</p>
<p>Suggested disinfectant for housing facilities: Thorough physical cleaning, chlorhexidine, bleach or other effective disinfectants.</p>
<p>Notification: None required.</p>
<p>Measures required under the Animal Disease Surveillance Plan: Currently none.</p>
<p>Measures required for introducing animals to infected animal: Quarantine, do not introduce animals with recent or observed clinical disease. Optimize animal health prior to introduction with appropriate nutrition and similar measures, minimize environmental extremes, ensure social compatibility.</p>
<p>Conditions for restoring disease-free status after an outbreak: Absence of apparent respiratory disease. Persistent subclinical infections are difficult to determine.</p>
<p>Experts who may be consulted: Any licensed veterinarian with appropriate experience or university animal extension specialists. Glen C. Weiser (Caine Veterinary Teaching Center, University of Idaho) may be able to provide reference laboratory support, also including <i>Mycoplasma</i> isolation and PCR detection of some species, and isolation and characterization of Pasteurellaceae. Thomas E. Besser (College of Veterinary Medicine, Washington State University) may be able to provide strain typing of <i>Mycoplasma</i> spp.</p>
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PESTE des PETITS RUMINANTS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Goats, sheep, small ruminants	Direct contact with ocular, nasal, oral secretions, urine and feces of affected animals; indirect via fomites is possible; virus fragile in environment, so long-distance transmission is unlikely	Fever, erosive stomatitis, conjunctivitis, gastroenteritis, pneumonia, abortion	Can be mild to severe with up to 100% morbidity and 20-100% mortality	Supportive care; antibiotics for secondary bacterial infections, anthelmintics for parasitic complications	Vaccination w/ PPR modified live vaccine; Eradication from newly infected areas	No

Fact Sheet compiled by: Andrea Goodnight

Sheet completed on: 4 June 2018

Fact Sheet Reviewed by: Douglas P. Whiteside

Susceptible animal groups: Goats – most susceptible and most severely affected (80-100% morbidity and mortality); sheep, small ruminants – less severe disease. White-tailed deer are susceptible experimentally. A few outbreaks in camels and water buffalo. Isolated clinical cases reported in: gazelles (Dorcas, Thomson’s, Rheem, Arabian), bushbuck impala, springbuck, gemsbok, bharal. Sindh ibex, bezoar ibex, Afghan Markhor goat, Nubian ibex, Barbary sheep, Laristan sheep, and Indian buffalo. Cattle, pigs – subclinical infection (dead-end hosts).

Causative organism: Peste des petits ruminants virus (Genus *Morbillivirus*, family *Paramyxoviridae*). Four lineages recognized.

Zoonotic potential: No

Distribution: Historically sub-Saharan Africa, north of the equator, more recently cases spreading, including into North Africa; Middle East (Turkey, Iraq, Iran, Pakistan, Afghanistan) southern Asia including India, Bangladesh and Vietnam; moving into Nepal and China as well.

Incubation period: 2-10 days (typically 3-6 days for clinical signs to appear)

Clinical signs: Acute (most common): High fever (40-41°C); crusting lip scabs, nasal discharge – serous to mucopurulent, eventually occludes nares; purulent ocular discharge; conjunctivitis – profuse catarrhal discharge matting palpebrae closed; necrotizing stomatitis – including lips, gingiva, dental pad, hard palate, cheeks, anterior tongue; gray necrotic foci over shallow erosions; profuse, non-hemorrhagic diarrhea; bronchopneumonia with dyspnea; anorexia, dehydration, emaciation; abortion; death in 5-10 days; long convalescence in survivors

Peracute mortality – Frequent in goats; high fever, severe depression, death

Subacute and chronic – Pneumonia, inconsistent signs (develops over 10-15 days)

Post mortem, gross, or histologic findings:

Gross: Erosions (not ulcerations) – inside of lower lip including gingiva, cheeks near commissures, tongue, hard palate, pharynx, upper 1/3 of esophagus; abomasum and small intestine – moderate erosions; Peyer’s patches – extensive necrosis; large intestine has most severe lesions – extensive congestion (“zebra stripes”) on mucosal folds of colon and rectum; pneumonia; generalized lymphadenopathy

Histologic: Degeneration, necrosis of epithelial cells of mucous membranes; eosinophilic intracytoplasmic and intranuclear inclusions in epithelial cells; lymphoid cell depletion in Peyer’s patches; necrotic/hemorrhagic enteritis; bronchointerstitial pneumonia; eosinophilic intracytoplasmic and intranuclear inclusions in giant cells and alveolar macrophages

PESTE des PETITS RUMINANTS

Diagnosis: Clinical signs are only presumptive so must have laboratory confirmation, especially to differentiate from rinderpest, although rinderpest is considered eradicated worldwide

Material required for laboratory analysis:

Virus neutralization: serum (10 mL)

Virus isolation or PCR (RT-PCR): whole blood in EDTA or heparin (10 mL), tissue (bronchial or mesenteric lymph nodes, tonsil, spleen, lung, intestinal mucosa)

Virus isolation: nasal, ocular, oral, fecal swabs

Ship samples fresh on ice within 12 hours

Histopathology on formalin fixed tissue

Relevant diagnostic laboratories:

National Veterinary Services Laboratories (NVSL)

Foreign Animal Disease Diagnostic Laboratory (FADDL), Plum Island, NY

http://www.aphis.usda.gov/animal_health/lab_info_services/diagnos_tests.shtml

FAO Reference Laboratory for PPR

CIRAD-EMVT Campus International de Baillarguet Montferrier-sur-Lez

BP 5034 34032 Montpellier Cedex 1 France

diallo@cirad.fr

Treatment: Supportive care, antibiotics for secondary infections, anthelmintics for parasitic complications; PPR hyperimmune bovine serum may decrease severity of clinical signs if given early in course of disease; quarantine infected animals; surviving animals – have circulating neutralizing antibodies up to 4 years post infection with likely life-long immunity

Prevention and control: Eradication – quarantine and slaughter, proper carcass disposal (incineration or burial); decontamination of housing facilities; proper disposal of contact fomites; restrict importation of sheep and goats from endemic areas. Vaccination, if approved by the government, strategically or for high risk population, with a homologous PPR vaccine – is protective for 3 years

No current evidence of virus circulation in wild ruminants, unless introduced from domestic sheep and goats.

Suggested disinfectant for housing facilities: PPR virus killed by most common disinfectants. UV light and desiccation likely inactivate the virus within 3-4 days. Virus survives long periods in chilled or frozen tissues.

Notification: In the US and Canada, this is a foreign animal disease which must be reported to state or federal veterinarian

Measures required under the Animal Disease Surveillance Plan: In the US and Canada, this is a foreign animal disease which must be reported to state or federal veterinarian.

Measures required for introducing animals to infected animal: It is not recommended to introduce new animals to infected animals; however, vaccination of introduced animals is recommended if must introduce.

Conditions for restoring disease-free status after an outbreak: Eradication of infected flock and decontamination of facility.

Experts who may be consulted:

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PESTE des PETITS RUMINANTS

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PHOCINE DISTEMPER VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Phocids, primarily harbor seals, but also gray seals. Virus isolated from northern sea otters.	Primarily aerosolization of respiratory secretions, but also possible via fecal, urinary, and ocular secretions via direct or indirect transmission.	Lethargy, fever, coughing, dyspnea, oculonasal discharge, conjunctivitis, keratitis, diarrhea, generalized body tremors and spasms, neurologic signs, increased buoyancy, abortion, inability to dive.	Acute to subacute. Mortality is high in susceptible populations.	Supportive, treat secondary infections.	Vaccination with MLV or killed canine distemper (CD) vaccine or subunit CD vaccine. Vaccination of free-ranging pinnipeds is controversial and difficult to implement effectively. Virus is enzootic in arctic seals.	No

Fact Sheet compiled by: Laurie J. Gage
Sheet completed on: 3 August 2011, updated 19 March 2013
Fact Sheet Reviewed by: Ariana Finkelstein, Kimberly Rainwater
Susceptible animal groups: Phocids and possibly northern sea otters.
Causative organism: Morbillivirus –Phocine Distemper Virus (PDV)
Zoonotic potential: None
Distribution: North Sea, North America (Atlantic coast and North Pacific Ocean)
Incubation period: 5 to 12 days
Clinical signs: Variable body condition, lethargy, fever, coughing, dyspnea, oculonasal discharge, conjunctivitis, keratitis, diarrhea, neurologic signs, increased buoyancy, abortion, inability to dive.
Post mortem, gross, or histologic findings: Bronchointerstitial pneumonia, interstitial and purulent pneumonia, alveolar and interstitial emphysema, alveolitis, generalized lymphodepletion. Less common findings are non-suppurative encephalitis and eosinophilic intracytoplasmic and intranuclear viral inclusion bodies in the brain and predominantly eosinophilic intracytoplasmic inclusion bodies in various organs including lungs, liver, kidneys, pancreas, intestine, and brain. Lymphoid depletion is marked in acute infection. In two pinniped cases of morbilliviral dermatitis, syncytia and eosinophilic intracytoplasmic inclusions were prominent in the epidermis, follicular epithelium, and sebaceous glands.
Diagnosis: Presence of characteristic histopathological lesions, immunohistochemistry, PCR, RT-PCR, ELISA. Paired serum samples with increasing antibody titer.
Material required for laboratory analysis: Fresh or fixed tissue, serum
Relevant diagnostic laboratories: University of Georgia Marine Mammal Diagnostics (Saliki)
Treatment: Supportive

PHOCINE DISTEMPER VIRUS

Prevention and control: Vaccination with canine distemper vaccine. Vaccination with a subunit vaccine is practiced in European rescue centers and appears to be protective.
Suggested disinfectant for housing facilities: No special requirements/standard disinfection protocol
Notification: Not required
Measures required under the Animal Disease Surveillance Plan: None required
Measures required for introducing animals to infected animal: Not recommended
Conditions for restoring disease-free status after an outbreak: No special requirements
Experts who may be consulted: Jeremiah T. Saliki (jsaliki@uga.edu)
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PLASMODIUM

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Reptiles, birds – especially penguins, and some mammals, including non-human primates and humans	Mosquitoes of different genera; in reptiles, also other biting flies	Lethargy; anorexia; minor to severe anemia; neurologic signs; paralysis	Typically of low virulence in adapted hosts; mild to severe – possibly fatal – disease in non-adapted hosts	Various anti-malarial drugs can be used but are unlikely to eliminate infection at tissue stage; resulting in relapses of parasitemia	Vector control and mosquito proof enclosures	Generally no; in endemic areas, primates can act as a reservoir for humans

Fact Sheet compiled by: Dennilyn Parker; updated by Gediminas Valkiūnas

Sheet completed on: 7 June 2011; updated 3 August 2013

Fact Sheet Reviewed by: Sam Telford Jr.; Arnaud Van Wettere

Susceptible animal groups: The parasite is reported in birds of the majority of avian orders. Species that have been relocated from habitats without vector or parasite, or in areas where the vector or parasite have been introduced are especially vulnerable, e.g. penguins, other captive Arctic or Antarctic species, species from Hawaii or other islands.

Infection of mammals is most common in tropical countries; diversity is greatest in Africa, where *Plasmodium* parasites have been reported in primates, rodents, ungulates and bats. One report in wild and captive capybaras in South America has been documented. Humans and non-human primates are infected mainly in tropical Africa, Asia and South America.

Reptiles have been seen infected mainly in tropical countries – primarily lizards, some snakes, and reported anecdotally in tortoises, but has not been reported in turtles or crocodiles.

Causative organism: *Plasmodium* spp. (Plasmodiidae, Haemosporida) >200 species.

Zoonotic potential: No zoonotic risk exists from avian or reptilian species. Although no evidence of zoonotic risk from non-human primate species, primates can carry the same species that infect humans, so reservoir exists.

Distribution: Worldwide, except Antarctica due to absence of mosquitoes and low temperature.

Incubation period: Avian – usually 5-7 days.

Clinical signs: Cases in most species of adapted hosts are often of low virulence. Importantly, the same lineages of *Plasmodium* sp. cause diseases of markedly different severity in different avian hosts that should be taken in consideration in conservation projects. Susceptible non-adapted avian species (e.g. penguins and some endemic Hawaiian birds) present lethargy, dyspnea, anorexia, vomiting, ruffled feathers, anemia where hematocrits may fall by more than 50% and regenerative hemolytic anemia is observed. Biliverdinuria may occur. Partial or total paralysis and convulsions can present terminally.

Post mortem, gross, or histologic findings:

Avian: Blood and reticuloendothelial system – hemolysis, splenomegaly, hepatomegaly, and pulmonary edema. Macrophages, lymphocytes and plasma infiltrate in liver and spleen. Exoerythrocytic meronts in endothelial cells with possible blockage of brain and lung capillaries. Hemozoin pigment in Kupffer cells and splenic macrophages.

Primates: virulence of different species and strains markedly vary in different hosts. Macroscopic pathology of the brain and endocardium might show hemorrhages, and the liver and spleen often are enlarged.

PLASMODIUM

<p>Microscopic pathology usually shows sequestration of pigmented parasitized red blood cells in the vessels of the cerebrum, cerebellum, heart, kidney and other organs. The spleen and liver contains abundant pigment containing macrophages and parasitized red blood cells. During acute infections, the kidney often has evidence of tubular necrosis.</p>
<p>Diagnosis: Identification of intracellular red blood cell parasite on a smear, but difficult to detect low intensity chronic infections by microscopy; gold standard – Giemsa stained blood smear – erythrocytic meronts and gametocytes with pigment granules. PCR is more sensitive but may still not identify low level parasitemias and often does not read co-infections; small subunit ribosomal ribonucleic acid and mitochondrial cytochrome b genes are definitive targets for malarial parasite ID and used to determine genetic relationships. Immunoblotting can be used to ID antibodies to <i>Plasmodium</i> but only to the level of parasite genus. ELISA available for <i>P. relictum</i> in penguins.</p>
<p>Material required for laboratory analysis: Giemsa stained blood films (microscopy) and whole blood or tissue (i.e. liver and/or spleen) (PCR) are most often used.</p>
<p>Relevant diagnostic laboratories: Any laboratory performing complete blood counts is capable of diagnosis <i>Plasmodium</i> spp. on blood smears. DNA testing is not widely performed commercially at this time, but is available in many research laboratories that manage wildlife parasites.</p>
<p>Treatment: <u>Avian</u> – Chloroquine phosphate, primaquine phosphate, pyrimethamine-sulfadoxine combinations, mefloquine, and atovaquone - proguanil hydrochloride – canaries, penguins, raptors and wild passerines. Sulfamonomethoxine – suppresses parasitemia but does not protect from mortality if given after circulating parasites are present, sulfachloropyrazine – reduces mortality but has no effect of parasitemia. Halofuginone – delays parasitemia but only minor suppression of it – turkeys. Mefloquine, and atovaquone - proguanil hydrochloride are highly efficient for blood stages, but does not affect exoerythrocytic (tissue) stages. <u>Primates</u> - drugs which are used for human malaria treatment can be used for treatment of malaria in primates (chloroquine phosphate, quinine sulfate plus doxycycline or malarone, and other drugs).</p>
<p>Prevention and control: Housing susceptible species indoors. Vector (mosquito) control. Prophylactic treatment of highly susceptible species can be considered. Vaccines development is under trial. Preventive treatment for primates has not been used extensively.</p>
<p>Suggested disinfectant for housing facilities: Disinfection is not appropriate for this disease.</p>
<p>Notification: None.</p>
<p>Measures required under the Animal Disease Surveillance Plan: None.</p>
<p>Measures required for introducing animals to infected animal: Isolate infected animals with vector control to prevent spread to susceptible animals.</p>
<p>Conditions for restoring disease-free status after an outbreak: Difficult or impossible as wildlife acts as a reservoir.</p>
<p>Experts who may be consulted: Centers for Disease Control and Prevention Center for Global Health, Division of Parasitic Diseases and Malaria 1600 Clifton Road Mailstop A-06 Atlanta, Georgia 30333 770-488-7788 or 855-856-4713 (toll-free) Fax: 404-718-4815 malaria@cdc.gov www.cdc.gov/malaria</p>

PLASMIDIUM

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PNEUMOCOCCOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primates, including humans; multiple domestic and lab mammal species; dolphins	Aerosol; direct contact	Primarily respiratory, neurologic, and septic	Asymptomatic to severe	Antibiotics and symptomatic treatment	Vaccine is available for humans. No vaccines have been used routinely for animals. Prevent contact with sick animals or people.	Theoretical

Fact Sheet compiled by: Ellen Wiedner

Sheet completed on: 3 August 2011; updated 18 September 2013

Fact Sheet Reviewed by: Jim Wellehan; Ramiro Isaza; Steve Unwin

Susceptible animal groups: Humans, multiple non-human primate species (both wild and captive), dogs, cats, rats, mice, guinea pigs, cattle, horses, dolphins.

Causative organism: *Streptococcus pneumoniae* is an alpha-hemolytic strep (encapsulated, facultative anaerobe, optochin sensitive and bile soluble) with more than 90 serotypes

Zoonotic potential: This risk is theoretical but unproven. Animals have developed disease both from human serotypes as well as animal-specific serotypes. One report documented in a human included several clinically ill house pets with *S. pneumoniae*. Suspected reverse zoonosis from a keeper to zoo chimpanzees have occurred.

Distribution: Worldwide.

Incubation period: Carrier status confirmed. With asymptomatic but infective carriers, it is unknown in animals how long carrier state lasts. Clinical disease can occur within 96 hours of exposure.

Clinical signs: Pneumonia, meningitis, sepsis, conjunctivitis, sinusitis, otitis media, other respiratory disease, polyarthritis, endocarditis, pericarditis, and sudden death. Clinical disease often more severe with a viral co-infection.

Post mortem, gross, or histologic findings: Fibrinous bronchopneumonia, pericarditis, necrotizing cerebral vasculitis.

Diagnosis: Bacterial identification of isolates using DNA sequencing, latex agglutination tests and others. Positive Gram staining of respiratory samples with lancet-shaped diplococci. Serotyping recommended.

Material required for laboratory analysis: Respiratory secretions, CSF, or blood. If immediate (< 1hr) transport to laboratory is not possible, samples should be inoculated into growth media and kept cool. The laboratory should be consulted first about appropriate media.

Relevant diagnostic laboratories: Any laboratory that performs cultures and sensitivities on a routine basis can complete testing for this organism. PCR testing can be found at many major commercial and veterinary diagnostic laboratories.

Streptococcus Laboratory
Centers for Disease Control and Prevention
1600 Clifton Rd

PNEUMOCOCCOSIS

<p>Atlanta, GA 30333 404-639-1237</p>
<p>Treatment: Historically, penicillins were recommended. However, severe multidrug resistance reported in many serotypes to beta-lactam, fluoroquinolone and macrolide antibiotics so culture and sensitivity should be collected and submitted prior to treatment.</p>
<p>Prevention and control: Vaccination programs for humans have decreased rates of <i>S. pneumoniae</i> severe and fatal disease cases. No vaccines have been tested in animal species. Prevention of contact with infected animals and good hygiene is recommended in zoos and similar animal facilities. If vaccination considered, bacterial typing required before vaccination to confirm polyvalent vaccine applicable.</p>
<p>Suggested disinfectant for housing facilities: The bacteria is susceptible to many disinfectants: 70% ethanol, 2% glutaraldehyde, 1% sodium hypochlorite and others. However, it can live in sputum at room temperature for one week and in dust particles for up to 25 days.</p>
<p>Notification: The disease is reportable nationally. CDC and several states currently conducting surveillance of resistant strains.</p>
<p>Measures required under the Animal Disease Surveillance Plan: None at this time.</p>
<p>Measures required for introducing animals to infected animal: Do not introduce new animals to an infected animal.</p>
<p>Conditions for restoring disease-free status after an outbreak: In human nursing homes, vaccination and treatment of close contacts with prophylactic antibiotics is done. However, no studies on this approach have been documented in animals.</p>
<p>Experts who may be consulted: <i>Streptococcus</i> Laboratory Centers for Disease Control and Prevention 1600 Clifton Rd, Atlanta, GA 30333 404-639-1237</p> <p>Fabian Leendertz Robert Koch Institute Postbox: 650280 D-13302 Berlin, Germany Nordufer 20, 13353 Berlin, Germany +49 (0)30 - 18754-2592 leendertzf@rki.de</p>
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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans, non-human primates, and numerous mammalian species, especially immune-compromised individuals.	Aerosol transmission, environmental exposure, or direct contact with infected individuals.	Dyspnea, dry cough, cyanosis, pyrexia, weight loss.	Can be fatal in immunocompromised individuals.	Trimethoprim-sulfa methoxazole (TMP-SMX) is the drug of choice.	Prophylactic treatment with TMP-SMX.	No as human strain is believed to be host-specific.

Fact Sheet compiled by: Brenda Tesini and Zachary Hoy

Sheet completed on: 3 June 2011; updated 10 September 2013

Fact Sheet Reviewed by: Francis Gigliotti; Remo Lobetti

Susceptible animal groups: Humans, primates and numerous mammalian species. The organism is presumed to be ubiquitous in the environment. Serological evidence shows that most healthy children have had exposure to the organism by 4 years of age. Studies screening numerous zoological, wildlife and laboratory mammalian species have also shown a high prevalence of exposure to the organism. The organism proliferates in the lungs of host species with compromised immune systems. Studies have found an absence of the organism in animals with body temperatures below 35°C and above 41°C. Studies conducted in birds, reptiles, amphibians and fish have not identified the organism.

Causative organism: Human derived: *Pneumocystis jirovecii* (formally known as *P. carinii*). Multiple other mammalian host-specific species exist. For example, *P. carinii* in the rat and *P. murina* in the mouse. The organism was previously thought to be a protozoan, but in 1988, through DNA analysis, it was determined to be a yeast-like fungus. It is unusual when compared to other fungi in that the cell membrane lacks ergosterol and currently is unable to be grown in culture. Genomic and phenotypic differences exist between the organisms that infect different mammalian species indicating that the organisms are host-species specific.

Zoonotic potential: *Pneumocystis* organisms infecting each mammalian species are host specific. No animal reservoir for *P. jirovecii* has been identified and no animal strains have been identified as human pathogens.

Distribution: Worldwide in humans and animals.

Incubation period: 3 to 12 weeks; but unclear if this includes carriage time in healthy individuals as compared to immunocompromised hosts.

Clinical signs: Immunocompetent individuals are most often asymptomatic. Immunodeficient individuals develop *Pneumocystis* pneumonia (PcP), a chronic progressive pneumonia. The most common clinical signs include dyspnea, an unproductive cough, cyanosis, pyrexia, and weight loss. Severe cases can lead to respiratory failure and death. Extrapulmonary lesions occur in a minority (<3%) of patients, involving most frequently the lymph nodes, spleen, liver, and bone marrow. The organisms reside in the alveoli and stimulate both a humoral and cellular immune response. The host's inflammatory response leads to alveolar damage, impaired gas exchange and decreased respiratory function which results in the common clinical signs of this disease.

Post mortem, gross, or histologic findings: Lungs show evidence of interstitial pneumonia. Grossly, the lungs will be edematous and heavy. They will have a pale gray or tan granular, firm, consolidated cut surface.

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<p>Histological examination of lung tissue often reveals a foamy eosinophilic exudate within the alveolar spaces and interstitial fibrosis of the alveolar septa. Basophilic dots within the exudate represent the <i>Pneumocystis</i> cysts. With special stains, the cysts can be identified as ovoid bodies. Additional stains can also be used to identify isolated trophozoites. The organism can be specifically identified using immunohistochemistry, immunofluorescence and PCR assay. Studies have also identified the organism in a large percentage of asymptomatic infants on post-mortem.</p>
<p>Diagnosis: Specific diagnosis is by the recovery and identification of the organism in samples obtained through trans-tracheal aspirate (TTA), bronchoalveolar lavage (BAL), induced sputum or lung tissue obtained through biopsy or necropsy. Identification of the organism via PCR assay, immunohistochemistry, immunofluorescence, or special stains that stain the cyst wall of the organism (Gomori's methanamine silver (GMS), toluidine blue O) or those that stain the nuclei of the trophozoites and sporozoites (Geimsa, Wright, Diff-Quick, polychrome methylene blue, and Gram's stain).</p>
<p>Material required for laboratory analysis: Bronchopulmonary secretions obtained via TTA, BAL or induced sputum. Lung tissue obtained via biopsy or necropsy.</p>
<p>Relevant diagnostic laboratories: Laboratories with the capability to perform nested PCR assay are used to identify the organism. Immunohistochemical methods require the host species-specific monoclonal antibody used to identify the organism to avoid false negative results. Identification of the organism using special stains requires reviewer expertise.</p>
<p>Treatment: Since the organism lacks ergosterol, common anti-fungal treatments are not effective. Trimethoprim-sulfamethoxazole (TMP-SMX) is the drug of choice for both the treatment of infection and prophylaxis. Alternative drugs used for the treatment of infection include pentamidine, trimethoprim plus dapsone, atovaquone and primaquine plus clindamycin. Alternative drugs used for prophylaxis include dapsone, dapsone plus pyrimethamine, pentamidine and atovaquone. Recurrence is common if the immunosuppressive condition of the host persists.</p>
<p>Prevention and control: Avoidance of the organism is impractical since the natural reservoir is unknown and the organism is presumed to be ubiquitous in the environment. TMP-SMX or other chemoprophylaxis can be used as a preventative treatment in susceptible individuals.</p>
<p>Suggested disinfectant for housing facilities: A study found the following chemical disinfectants to be effective in the inactivation of <i>Pneumocystis</i> cysts: 70% ethyl alcohol, 10% iodoform, 1% quaternary ammonium salts, 3% hydrogen peroxide, sodium chlorite and 1% cresol soap.</p>
<p>Notification: None</p>
<p>Measures required under the Animal Disease Surveillance Plan: None</p>
<p>Measures required for introducing animals to infected animal: Prevent exposure of healthy animals to animals exhibiting clinical signs of pneumocytosis.</p>
<p>Conditions for restoring disease-free status after an outbreak: This approach may not be possible since a large percentage of humans and other mammalian species harbor this organism while remaining asymptomatic. Testing can be used to screen individuals for the presence of the organism. Serological screening is not effective since a large percentage of humans and other mammalian species are shown to have had exposure to the organism. Sterilization of any air filters in the area of the outbreak is an important measure to reduce the number of cysts in the environment. Disinfecting the area of the outbreak with appropriate disinfectants will help to inactivate any remaining cysts.</p>
<p>Experts who may be consulted: Brenda L Tesini, MD University of Rochester Medical Center Department of Pediatrics (Infectious Diseases)</p>

PNEUMOCYSTIS

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POLIOVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans; non-human primates	Fecal-oral and respiratory routes. Rapidly spread through feces-contaminated food and water sources.	Range from asymptomatic to paresis and death.	Most severe clinical signs include permanent flaccid paralysis of one or more limbs or muscle groups. Paralysis of respiratory muscles can lead to death.	Symptomatic, supportive care, based on clinical presentation No effective antiviral medications currently advocated.	Vaccination - used extensively in humans. To control spread of infection, use isolation, standard cleaning and disinfection methods, and PPE.	Yes and humans are the primary reservoir

Fact Sheet compiled by: Wynona C. Shellabarger

Sheet completed on: 8 August 2011; updated on 30 August 2018

Fact Sheet Reviewed by: Jennifer D'Agostino

Susceptible animal groups: Humans and non-human primates are affected although cases in NHPs are rare with great apes, in particular chimps, most frequently reported in literature. Macaques and chimpanzees assisted in vaccine development in early 1950's-1960's.

Causative organism: Poliovirus types 1, 2 and 3, family Picornaviridae, subgroup Enteroviridae

Zoonotic potential: Yes, humans are the primary reservoir.

Distribution: Historically, the disease was present worldwide. Western Hemisphere declared free of indigenous poliovirus since September 1991, and the last case of endemic polio in the US was in 1979. This status was achieved through the global efforts of WHO, UNICEF, and an international contingent spearheading the Global Polio Eradication Initiative (GPEI) and widespread use of vaccines in humans. Global eradication is still an active goal of these organizations, and incidence and transmission have continued to dramatically decline with continued use and distribution of vaccine. Currently, three countries maintain an endemic status: Afghanistan, Nigeria and Pakistan; an additional four African countries have continued incidence of imported-wild type polio.

Incubation period: Differs depending on type of polio but ranges from 3 to 35 days. Non-paralytic disease has incubation period of 3 to 6 days; paralytic disease has incubation period of 7 to 21 days. Virus can be shed in the feces for 3 to 6 weeks post-exposure or after vaccination with oral polio vaccine (OPV).

Clinical signs: Although flaccid paralysis is the most noteworthy and potentially severe of the clinical signs described, poliovirus infection in humans can be highly variable and clinical signs are categorized based on presentation. The majority of human infections are asymptomatic (72%). About 24% of infections result in minor disease including those of the upper respiratory tract, gastrointestinal disturbances and flu-like signs with associated fever and muscle aches. In 1-2% of cases, signs are more severe including meningitis, muscle weakness or flaccid paralysis of a single limb to quadriplegia, and respiratory failure with 0.1% of all reported polio cases resulting in the paralytic form in humans. Death occurs, but rarely, at 2-5% in children and 15-30% in adults that contract the paralytic form of this disease. A post-polio syndrome may occur in 25% to 40% of human cases as well, which develops decades after the initial infection. Although rare, poliovirus infection has been described in chimpanzees, orangutans, gorillas, macaques, and colobus monkeys in human care, laboratory, and wild settings, and manifests with similar clinical signs to those of human infections.

Post-mortem, gross, or histological findings: Most severe lesions are associated with paralytic disease and include muscle wasting, inflammation, demyelination, apoptosis, destruction of interior horn cells of spinal cord, brain stem and/or lower motor neurons.

Diagnosis: Culture, intratypic differentiation, genome sequencing, and serology are used for poliovirus

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testing. Viral detection via cell culture or PCR of throat, fecal, or occasionally CSF cultures may be positive within the first week of illness in humans. Real-time reverse transcription PCR is used to distinguish wild-strain from vaccine-induced strain from cell culture isolates. Retrospectively, serologic titers can be used to confirm diagnosis since IgM and IgG titers may take weeks to develop and become detectable.

Material required for laboratory analysis: Diagnostic samples include pharyngeal swabs, feces, CSF fluids, urine, and serum. Contact local and state public health and epidemiology officials for specific NHP sample submissions and guidance.

Relevant diagnostic laboratories:

Diagnosis, isolation and characterization of polioviruses from submitted human samples are coordinated by: Center for Disease Control

Global Polio Laboratory Network/Polio and Picornavirus Laboratory (Division of Viral Diseases)

1600 Clifton Rd

Atlanta, GA 30329-4027 USA

800-CDC-INFO (800-232-4636), TTY: 888-232-6348

Email: CDC-INFO

(404) 639-2749

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www.cdc.gov

Treatment: Symptomatic treatment is based on severity of clinical signs, including pain relief and physical therapy. Mechanical ventilation used in humans with permanent respiratory muscle paralysis.

Prevention and control: Infected animals should be isolated and standard disinfection measures used with personnel protective equipment to minimize exposure to humans or other animals.

Vaccines are used extensively in humans to prevent disease, and vaccine use has reduced incidence of disease worldwide by 99%. Inactivated (IPV) and oral polio (OPV) vaccines are currently available for human use, but since 2000, only IPV has been used in the US to minimize vaccine-associated paralytic polio (VAPP) incidence. The World Health Organization recommends that all children be fully vaccinated.

Current recommendations for childhood coverage are a series of 4 IPV vaccines at 2, 4, 6-18 months, and 4-6 years of age. Naïve adult vaccine recommendations and vaccine information for travelers to endemic countries are also available through the CDC website. OPV is still used in a number of other countries. Routine polio vaccination with OPV or IPV of great apes in human care has been recommended historically but is currently at the discretion of the animal's holding facility. Risk of exposure is low due to human vaccine eradication efforts. Type and schedule of vaccination in NHPs is extrapolated from human ACIP recommendations and vaccines available.

Suggested disinfectant for housing facilities: Poliovirus is known to be susceptible to heat, chlorine, formaldehyde, and UV light. Standard disinfection using a dilute bleach solution or one of the above products should be adequate. Removal of feces and bodily fluids before disinfection is required for effective disinfection.

Notification: If polio is suspected, veterinarians should work closely with local and state public health officials and epidemiologists. Contact CDC directly if local or state authorities are not available.

Measures required under the Animal Disease Surveillance Plan: Currently no measures are required. However, polio is epidemiologically important to monitor due to extensive worldwide eradication efforts in the human population.

Measures required for introducing animals to infected animal: Maintain potentially infected animals in isolation and quarantine conditions until presentation is resolved. Vaccination of conspecific naïve NHPs should be considered.

Conditions for restoring disease-free status after an outbreak: Minimize fecal contamination and clean and disinfect potentially contaminated areas thoroughly for at least 3-6 weeks post-infection and vaccination series. Source of infection should be determined and NHP staff vaccination history should be reviewed and updated if necessary.

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Experts who may be consulted:

Center for Disease Control

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PSITTACINE BEAK AND FEATHER DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Psittacines - Old World more than New World species	<p>Direct contact with infected animals with virus presented by inhalation or ingestion.</p> <p>Indirect contact with contaminated excretions, secretions and feather dust.</p> <p>Virus remains in contaminated environments, particularly air handling systems, for years.</p>	<p><u>Peracute:</u> Particularly common in African grey parrots with pancytopenia and death.</p> <p><u>Acute:</u> Depression followed by appearance of dystrophic feathers and death.</p> <p><u>Chronic:</u> Progressive appearance of dystrophic feathers. Necrotic beak and ulcerations in some long term infected birds. Death occurs in months to years.</p>	<p>Aggressive disease most common in African grey, vasa, and ecleetus parrots, and cockatoos.</p> <p>PCV-1 associated disease is fatal in most Old World psittacines.</p> <p>Chronic and less severe disease in lovebirds, lorries and lorikeets, particularly those birds infected with PCV-2.</p>	Supportive care should be provided in isolated environments where even caretakers have no contact with other birds.	<p><u>Prevention</u> PCR-based testing has reduced spread in managed populations.</p> <p>Developed vaccine has reached governmental approval stage.</p> <p><u>Control</u> Testing and isolation of infected birds; strict entry quarantine protocols.</p>	None known

Fact Sheet compiled by: Branson W. Ritchie

Sheet completed on: 15 November 2011; updated 19 August 2013

Fact Sheet Reviewed by: Thomas N. Tully; Lauren V. Powers

Susceptible animal groups: All psittacines are susceptible to infection. Most New World species develop a rapid immune response and clear the virus, although classic disease has been documented in some New World species (i.e., macaws and Amazon parrots). Classic disease associated with PCV-1 can occur in any Old World psittacine but is most common in cockatoos, African grey parrots, ring-necked parakeets and ecleetus parrots. PCV-2 causes less severe disease and affected birds may recover from disease; infections with this pathotype are most common in lorries and lorikeets. Lovebirds may be infected with PCV-1 alone or with both PCV-1 and PCV-2. Disease progression appears to vary in lovebirds infected with both pathotypes.

Causative organism: Psittacine circovirus - a non-enveloped icosahedral DNA virus belonging to the family *Circoviridae*. Two pathotypes, PCV-1 and PCV-2 must be distinguished for accurate prognosis and patient management. Circovirus infections have also been documented in Anseriformes, Columbiformes, Passeriformes, Galliformes and gulls.

Zoonotic potential: No known human transmission has occurred.

PSITTACINE BEAK AND FEATHER DISEASE

Distribution: Virus likely evolved in Australia and has been disseminated globally through transcontinental movement of infected birds. Virus could be found on any continent with a sufficient population of free-ranging or captive psittacine birds to support virus survival and transmission. Virus will continue to spread in untested or, until available, unvaccinated psittacine birds.

Incubation period: Experimentally, signs appear in 3-4 weeks. However, variation in disease progression can make the incubation period appear longer.

Clinical signs: Most birds infected with PCV-1 develop a transient infection that can be detected by finding viral DNA in whole blood. Most infected birds subsequently respond with an appropriate immune response and clear the virus with no recognizable clinical changes. In unmanaged (untested) populations, infection should be considered relatively common while disease is comparatively uncommon.

Peracute/Acute Form: These forms most commonly occur in young chicks, and may begin with signs unrelated to the beak or feathers. Affected birds are often depressed and regurgitate due to crop stasis. They may develop a diarrhea-causing enteritis, or pneumonia, and die without displaying any lesions of the feathers or beak. This peracute form of the disease is particularly common in African grey parrots that frequently die with acute hepatic necrosis. In the acute form, feather abnormalities in already developed feathers (from causes other than PCV) should be distinguished from abnormalities associated with the developing feather (from the pulp cap to the feather base). Visible developmental feather abnormalities include: retention of the feather sheath, hemorrhage of the pulp cavity, shortened deformed feathers and circumferential constrictions at the feather base. Stress lines are common in affected feathers. Affected feathers are often loose, break easily, may bleed, and elicit a pain response with minimal manipulation. Some chicks die within days to weeks of the first signs of feather abnormalities and others survive with progression to chronic disease.

Chronic Form: Newly developing powder down and contour feathers are the first to show clinical changes in birds that exhibit feather abnormalities after their remiges and rectrices are developed. The visible changes in these feathers are similar to those described above. In psittacines other than lovebirds, feather lesions associated with PCV-1 become progressively worse with each successive molt and if the bird survives for years it may become mostly or completely featherless as feather follicle damage prevents replacement.

In some affected birds, beak abnormalities may occur that typically start as a brownish necrotic area on the inside of rhinotheca. Affected beaks may elongate, becoming progressively deformed, and fracture. Secondary beak and oral infections are common in necrotic areas of the beak. Some affected birds may develop beak elongation in the absence of necrosis. In some birds, the nails can also be deformed or slough.

Birds with the chronic form of the disease may live for months to years. Progressive disease is associated with organopathies that are likely associated with immune suppression and birds usually die from secondary bacterial, fungal, parasitic, or other viral infections.

Birds with Pbfd shed substantial quantities of extremely environmentally stable virus in their feather dander and should not be maintained in environments (aviaries or hospitals) or by care takers that have direct or indirect contact with other birds. Recovery of Old World psittacines with the chronic disease associated with PCV-1 has not been documented. Comparatively, PCV-2 appears clinically less virulent and lorries and lorikeets with moderate feather abnormalities have been shown to recover as indicated by a return to normal feather plumage and no detectable viral DNA in their blood. The PCV-2 pathotype has only been documented as a monotypic infection in lorries and lorikeets. Comparatively, other psittacines, particularly lovebirds, have been documented with both PCV-1 and PCV-2 and the role that co-infection may play in altering the virulence of PCV-1 and thus the progression of classic disease is unknown.

Post mortem, gross, or histologic findings: Gross feather and, less often beak, changes described above are associated with the circovirus infection. In chronic cases, other lesions related to the secondary infections that actually lead to the birds death will be found at necropsy.

PSITTACINE BEAK AND FEATHER DISEASE

Predominant histological lesions include necrosis and ballooning degeneration of epithelial cells in the epidermal collar and epidermal, basal and intermediate zones of the developing feather shaft. The follicular epithelium also may be necrotic, but this lesion is reported less commonly. Feather sheath hyperkeratosis prevents the feather from ex-sheathing resulting in retention of the feather sheath. Feather pulp lesions are characterized by suppurative inflammation, including perivascular accumulations of heterophils, plasma cells, macrophages and rarely lymphocytes. The characteristic basophilic intracytoplasmic - and less commonly intranuclear - inclusions are usually, but not always present in diseased feathers. Granulomatous dermatitis with vesicle formation was described in a group of infected lovebirds.

Histologic lesions in the beak of PBF/D birds are similar to those described in their feathers, including necrosis and hyperplasia of epithelial cells in the basal and intermediate epithelial layers. Hyperkeratosis and separation of the cornified outer layer from the underlying tissues and bone may also be evident, and are often accompanied by secondary necrosis and osteitis of associated tissues

In peracute cases, histologic lesions may be limited to severe bursal or thymic necrosis with the presence of viral inclusion bodies. Feather pathology in these cases may not occur, or may be limited to edema in the follicular epithelium (if present).

In birds with beak disease, necrosis and inflammation of the epithelial lining of the tongue, beak cavity, and crop have also been reported. Secondary Gram-negative bacteria and fungi are commonly isolated from beak lesions and may be associated with acute or chronic inflammatory reactions.

Diagnosis: PBF/D should be considered in any bird presenting abnormal feather loss or developmental abnormalities. PBF/D can only be diagnosed by detection of the virus using *in situ* hybridization, immunohistochemistry or electron microscopy to document the virus or viral components in diseased tissues. For antemortem diagnosis, a biopsy of 3-4 diseased feathers and their associated follicle is recommended. It is critical for the clinician to biopsy diseased feathers. Both diseased and normal feathers can be present directly next to each other and failure to obtain a biopsy of diseased feathers can result in an inaccurate diagnosis. Birds with the peracute and early acute forms of the disease may die before the development of feather abnormalities and disease is documented by histopathologic evaluation of internal organs including the bursa, thymus and liver.

PCR-based testing can be used to detect target segments of viral DNA in the blood of suspect birds before feather abnormalities develop but this condition does not confirm the presence of disease. Most birds infected with PCV develop a transient infection that can be detected by finding viral DNA in whole blood. Most infected birds subsequently respond with an appropriate immune response and clear the virus with no recognizable clinical changes. A bird that is PCR positive for PCV-1 and does not have dystrophic feathers must be retested in 90 days to determine if the bird has cleared the virus. It is important that birds be maintained in a virus free environment during this 90 day period. The author has placed vaccinated (protected) birds in the same room with PBF/D positive birds and viral DNA can be intermittently detected in the vaccinated birds because of persistent environmental exposure to the virus and the subsequent clearing of the virus through the blood that is necessary for any inhaled or ingested virus.

A bird that is PCR positive for PCV-2 and does not have dystrophic feathers must be retested in 180 days to determine if the bird has cleared the virus. Lories with PCV-2 and with dystrophic feathers have been documented to recover from disease but should be maintained in strict isolation during any convalescent period. Virus is being shed in the dystrophic feathers until they are replaced even though viral DNA can no longer be detected in the blood.

For the most current recommendations on testing and interpretation of PCR-based assays, see www.vet.uga.edu/SAMS/IDL.

Material required for laboratory analysis: Biopsy of dystrophic feathers and their associated follicle in formalin for histologic diagnosis. Whole blood collected by venipuncture. Blood samples collected by toe nail

PSITTACINE BEAK AND FEATHER DISEASE

clipping should be considered environmental samples and not a bird specific sample. Feathers submitted for PCR-based testing should also be considered environmental samples and are not bird specific. Viral DNA can be detected by PCR-based testing in environmental swabs. These can be used to document the extent of environmental contamination (air filters, fan motors, nest boxes, etc.) and for evaluating cleaning efforts following an outbreak.

Post-mortem samples include bursa, thymus, liver, spleen, kidney, and dystrophic feathers (if present) in formalin. Swabs of tissues collected from the cut surface of the bursa, thymus or liver can be used for rapid detection of viral DNA. Only disposable scalpel blades should be used for collecting post-mortem samples or swab may be positive because of transfer to the cut surface of the organ from viral contaminated instruments. Prior to shipping, blood samples should be stored refrigerated (4°C/39.2°F). Samples must be shipped in a padded envelope or box. In cooler seasons, samples may be sent by regular mail, but overnight is recommended. For the most current recommendations on sample submission, see www.vet.uga.edu/SAMS/IDL.

Relevant diagnostic laboratories:

Infectious Disease Laboratory
College of Veterinary Medicine
University of Georgia
110 Riverbend Rd
Riverbend North, Room 150
Athens, GA 30602-7390
706 542-8092
Fax: 706 583-0843
www.vet.uga.edu/SAMS/idl/

Treatment: No known specific antiviral treatment.

Prevention and control: Transmission of the virus is primarily through inhalation or ingestion of air or food containing viral contaminated feather or fecal dust. Contaminated clothing, hair and body surfaces of care takers can also serve to disseminate the virus as can contaminated bird carriers, feeding utensils, nest boxes and nesting materials. Two of the most severe modern (post PCR-based testing) outbreaks the author investigated were associated with use of a contaminated grinder for nail grooming and the sale of a contaminated egg incubator. Maternal transmission has been documented. The virus is extremely environmentally stable and for the safety of birds any contaminated environment should always be considered a source of infectious virus. Any diseased birds should be maintained in strict isolation and the care takers of these birds should always be considered contaminated with the virus. Maintain strict quarantine and testing protocols for new birds prior to entering the collection.

PCR-based testing should be used during entry quarantine to detect viral DNA in the blood. See the recommendations above for testing procedures and interpretation. Because of the difficulty in decontaminating a typical clinic, it is not recommended that known diseased birds be evaluated or maintained in the hospital. PCR-based testing of environmental swabs can be used to document the severity of viral contamination in the environment.

A PCV vaccine has been developed by the Emerging Diseases Research Group at the University of Georgia and the vaccine awaits a USDA approved manufacturer to take the necessary steps to register the vaccine for commercial use.

Suggested disinfectant for housing facilities: While specific data on the susceptibility of PCV to disinfectants is unknown, it is known that other circovirus are among the most environmentally stable and disinfectant resistant of all viruses. The goal in a contaminated facility is to wash the virus out of the environment, expose contaminated surfaces to prolong drying and direct sunlight and then seal any remaining virus to a substrate

PSITTACINE BEAK AND FEATHER DISEASE

with paint (or equivalent). Any contaminated surface that is porous (not made of metal or plastic) should be discarded. All metal, concrete and plastic surfaces should be washed with a sodium hypochlorite (e.g. Clorox)-containing detergent, rinsed and allowed to dry in direct sunlight. The procedure should be repeated 3-4 times. Air handling systems should be professionally cleaned by a company experienced with decontaminating hospital air systems. Once repeated cleaning has been accomplished, a pressure painter should be used to coat all remaining surfaces (floor, walls and ceiling). If a diseased bird has been maintained in an incubator, one should make certain that the fan and motor housing are decontaminated and PCR negative for viral DNA before the fan is returned to service. PCR-based testing can be used to evaluate the success for virus removal from the environment.

Notification: Not needed.

Measures required under the Animal Disease Surveillance Plan: Not applicable

Measures required for introducing animals to infected animal: It is not recommended to mix infected and non-infected birds.

Conditions for restoring disease-free status after an outbreak: Remove any birds with feather dystrophy and maintain in isolation while conducting additional diagnostic testing. Remove birds without feather dystrophy from any potentially contaminated environment, wash the birds if feasible and wait 90 days (one could also blood test these birds for the presence of viral DNA immediately but many will be blood positive and clear the virus. Waiting 90 days with the birds in a non-contaminated environment will reduce the number of birds that require additional testing). Follow the current testing recommendations based on the detected pathotype provided at www.vet.uga.edu/SAMS/IDL. PCR-based testing of environmental samples collected during and after the cleaning and decontaminated process as detailed above.

Experts who may be consulted:

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 University of Georgia
 College of Veterinary Medicine
 (706) 206-7931
britchie@uga.edu

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 Emerging Diseases Research Group
 University of Georgia
 706-583-0742
crg@uga.edu

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American Association of Zoo Veterinarians Infectious Disease Manual
PSITTACINE BEAK AND FEATHER DISEASE

for the Isolation and Identification of Avian Pathogens, 5th edition. American Association of Avian Pathologists, Kennett Square, Pennsylvania. 249 pp.

PYTHIOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals: canids, felids, equids, felids, cattle, camelids and sheep. Birds (limited)	Motile, biflagellate zoospore (<i>P. insidiosum</i>) released into aquatic environments.	Gastro-intestinal: weight loss, vomiting, diarrhea, and hematochezia. Other mucosal (e.g. vulvar): weight loss, masses Cutaneous: Non-healing wounds, nasopharyngeal lesions, invasive subcutaneous masses, draining nodular lesion, or ulcerated plaque-like lesions.	Often fatal unless resectable with wide margins.	Surgical resection or amputation of infected tissues with wide margins. Post-operative treatment with antifungals may decrease recurrence when incomplete resection occurs. Addition of corticosteroids to antifungals may be of benefit for nonresectable lesions, Immunotherapy has also been seen to be effective in humans and horses.	As it is environmental exposure, control is difficult.	No; although humans can get it from the environment, infection is rare.

Fact Sheet compiled by: Roberto Aguilar, updated by Leonel Mendoza and Raquel Vilela, updated by Charles O. Cummings

Sheet completed on: 31 January 2011; updated 9 September 2013; updated 11 November 2019

Fact Sheet Reviewed by: Amy Grooters

Susceptible animal groups: Essentially, all mammals are susceptible. Small mammals, cats and dogs have been reported. Horses, cattle, sheep, and camelids present pythiosis with some frequency. Captive wild felids and ursids have been reported although all mammals are potentially susceptible. In zoo species specifically, primary pulmonary pythiosis in a jaguar in Louisiana; spectacled bears in South Carolina; and a lion in Florida have been reported. Mandibular, bulbar, gastric, and vulvar infections in captive camels. In birds, a cutaneous infection in a white-faced ibis and esophageal infection in an ostrich have been described. Ocular and vascular pythiosis is reported in humans.

Causative organism *Pythium insidiosum* (pathogenic "water mold").

Zoonotic potential: Humans would get pythiosis from the environment, but infection is rare. No evidence had been documented that pythiosis can be transmitted from an animal to a person.

Distribution: Globally, pythiosis is most often encountered in Southeast Asia (especially Thailand and Indonesia), eastern coastal Australia, New Zealand, and South America, but has also been recognized in Korea, Japan, and the Caribbean. In the United States it is most often found in the southeastern US but it also has been identified in Wisconsin, New Jersey, New York, Virginia, Kentucky, Arizona, California, Illinois, Indiana, Oklahoma, Missouri, Kansas, and Tennessee.

Incubation period Unknown, but clinical disease likely develops weeks to months after exposure.

Clinical signs:

Gastrointestinal: weight loss, vomiting, diarrhea, and hematochezia. Laboratory abnormalities include eosinophilia, anemia, hyperglobulinemia, hypoalbuminemia, and rarely hypercalcemia. Abdominal

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radiography and sonography usually reveal severe segmental thickening of the gastrointestinal tract, an abdominal mass, and/or mesenteric lymphadenopathy.

Cutaneous: Non-healing wounds and invasive masses that contain ulcerated nodules and draining tracts. In horses, the formation of hard masses (“kunkers”) within the lesions may occur. Nasopharyngeal lesions, invasive subcutaneous masses, draining nodular lesion, and ulcerated plaque-like lesions are found in cats. *Pythium insidiosum* has been also reported affecting bones, lungs, lymph nodes, eyes and blood vessels.

Post mortem, gross, or histologic findings: Histologically pythiosis is characterized by eosinophilic pyogranulomatous inflammation associated with broad (4-7 micron), poorly septate hyphae. Affected tissues contain multiple foci of necrosis surrounded and infiltrated by neutrophils, eosinophils, and macrophages. In addition, there are discrete granulomas composed of epithelioid macrophages, plasma cells, multinucleate giant cells. Hyphae stain well with GMS but less well with PAS. Histologically pythiosis, other oomycoses, and zygomycosis have a similar appearance.

Diagnosis: Veterinarians and physicians with expertise in this disease could suspect pythiosis because the clinical features of the disease. However, a clinical specimen (biopsy, kunkers) is always recommended to support the findings. Confirmation is usually histopathology followed by PCR confirmation of pythiosis in paraffin-embedded tissues. Serology has been performed successfully in canids, exotic felids, and several species of ursids, but this frequently requires species-specific antibodies. Cytologic evaluation of exudates from draining tracts or fine-needle aspirates of enlarged lymph nodes may be suggestive of fungal infection.

Material required for laboratory analysis: Paraffin-embedded tissue, infected tissue, fine-needle aspirates, serum, Gomori's methenamine silver stain (GMS), selective media containing streptomycin and ampicillin for culture

Relevant diagnostic laboratories:

Panfungal PCR and Sequencing from Paraffin-embedded Tissue

Texas A&M

Dermatopathology Specialty Service, TVMDL

Shipping Address (FedEx, UPS, LSO)

483 Agronomy Rd.

College Station, TX 77843-4471

USPS Mailing Address

PO Drawer 3040

College Station, TX 77841-3040

Pythium Serology (Canine and Equine)

158 Greene Hall

Auburn University, AL 36849

(334) 844-2694

Fax: (334) 844-2652

Fungal Culture & Immunotherapy Consultation

Leonel Mendoza

Michigan State University

North Kedzie Hall

354 Farm Lane, Rm 322

East Lansing MI 48824-1031

(517) 432-1234

Fax: (517) 432-2006

mendoza9@msu.edu

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Treatment: Surgical resection of infected tissues with wide margins or amputation for distal cutaneous lesions. Postoperative treatment with itraconazole and terbinafine may decrease the chance of recurrence in lesions that are not completely resected. For inoperable pythiosis, the addition of corticosteroids to terbinafine and itraconazole had resulted in lesion resolution and decreased titers in dogs.

Immunotherapy (Pan American Veterinary Laboratories, <https://pavlab.com/pavlab/pythiosis-insidiosum/>) is often effective for treatment in horses, especially when it is combined with aggressive surgical resection. It is infrequently effective in dogs.

Prevention and control: As it is transmitted via environmental exposure, control is difficult.

Suggested disinfectant for housing facilities: No special requirements for disinfection. Standard disinfection protocols may be used.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: None

Conditions for restoring disease-free status after an outbreak: None

Experts who may be consulted:

Most internal medicine specialists (DACVIMs) and dermatologists (DACVDs) practicing in the American Southeast are familiar with the intricacies of treating pythiosis and other oomycoses.

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**RABBIT HEMORRHAGIC DISEASE (RHD)/
RABBIT CALICIVIRUS DISEASE (RCD)**

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
European rabbit (<i>Oryctolagus cuniculus</i>)	<p>Direct contact with infected rabbits, rabbit products or secretions; insect or animal vectors; fomites; fecal excretion of virus from predators or scavengers.</p> <p>Importation of infected rabbit meat or rabbits that survive infection as they can shed virus for at least 4 weeks.</p> <p>Climate may be important-many outbreaks occur in winter or spring.</p>	<p>Often sudden death with no obvious signs.</p> <p>In symptomatic animals: depression, coma, nervous signs (padding, ataxia, convulsions, opisthotonos), reluctance to move, prostration; serosanguineous discharge from nostrils.</p>	Often high morbidity (up to 100%) and high mortality (50-100%)	None	<p>Avoid contact with infected or contaminated animals, animal products, fomites, or vectors.</p> <p>Perform disinfection, depopulation, surveillance, and quarantine under supervision of state and federal agencies.</p> <p>Vaccinate in countries where available and legal - not so in US</p>	No

Fact Sheet compiled by: Denise McAloose

Sheet completed on: 12 January 2011; updated 6 March 2013

Fact Sheet Reviewed by: Alisa Newton; Ken Conley

Susceptible animal groups: European rabbit (*Oryctolagus cuniculus*); typically affects animals > 2 months of age; animals that survive infection become immune

Causative organism: Family: Caliciviridae Genus: Lagovirus Species: Rabbit hemorrhagic disease virus; single serotype, two subtypes (RHDV, RHDVa)

Zoonotic potential: No

Distribution: Disease has been reported in > 40 countries and is endemic in Australia, New Zealand, Cuba, parts of Asia and Africa, and most of Europe. Sporadic reports in several countries including Mexico (now eradicated), Uruguay (2004), and the United States (Iowa 2000, Utah 2001, Illinois 2001, New York 2001, Indiana 2005, Minnesota 2010), although now thought to have been eradicated in the US. It also has been reported in China, Republic of Korea, India and the Middle East. It is more likely to be detected in large congregations than in single (e.g. individual pet) rabbits; disease spread exacerbated in crowded conditions.

**RABBIT HEMORRHAGIC DISEASE (RHD)/
RABBIT CALICIVIRUS DISEASE (RCD)**

Incubation period: Incubation 1-3 days; death often occurs within 12h-36 hours of fever onset
Clinical signs: Infection may present no apparent symptoms. In symptomatic animals, fever, depression, reluctance to move, neurologic signs, and bleeding from mouth or nostrils may be seen. In animals that survive infection, jaundice, weight loss and lethargy may be seen.
Post mortem, gross, or histologic findings: <u>Gross findings:</u> most commonly seen is friable liver, splenomegaly; and pulmonary congestion, edema and/or petechiae; may also see multifocal petechiae in other organs or serosanguineous tracheal fluid <u>Histologic findings:</u> More common lesions: mild to (more often) severe hepatic necrosis, disseminated intravascular coagulopathy (DIC), splenic congestion, multifocal lymphocytolysis. May also see multifocal acute pulmonary edema, congestion or hemorrhage and multifocal hemorrhage in other sites.
Diagnosis: <u>Serologic tests:</u> hemagglutination inhibition (HI), indirect ELISA (I-ELISA), competitive ELISA (C-ELISA). <u>Pathogen identification:</u> hemagglutination test (HA), electron microscopy (negative staining EM, immuno-EM, immunogold EM), virus detection ELISA, RT-PCR, Western blot, histology, immunostaining, inoculation study (RHDV never grown in cell cultures); <i>in situ</i> hybridizaion
Material required for laboratory analysis: Serum: HI, I-ELISA, C-ELISA Fresh liver (preferred sample), spleen or lung: HA, RT-PCR, inoculation study 10% neutral buffered formalin-fixed, paraffin embedded liver, spleen, lung: histology, immunostain Fresh or fixed liver (depending on procedure): EM
Relevant diagnostic laboratories: USDA-APHIS-VS-NVSL-FADDL 40550 Route 25 (for packages) Orient Point, NY 11957 P.O. Box 848 (for letters) Greenport, NY 11944-0848 (631) 323-3256 Fax: (631) 323-3366
Treatment: Currently there is no treatment for this disease.
Prevention and control: For prevention and control, biosecurity is highly important; avoid contact with imported rabbits and rabbit products; prevent contact between healthy and ill animals; quarantine new animals or animals that have been in contact with other rabbits. With input from State and Federal agencies, control will include disinfection, depopulation, surveillance, and quarantine; elimination of fomites (including insects, other animal vectors); incineration of dead animals, feedstuff, feces; limit or ban animal/animal derivative product movement in face of outbreak. Animals that survive infection are immune but can shed virus for at least 4 weeks. Two vaccines are available in UK; no vaccine available in US. Vaccinated rabbits are protected from disease but due to low mucosal immunity can still develop intestinal infection and shed virus in the absence of clinical disease.
Suggested disinfectant for housing facilities: This process is to be performed in collaboration and under supervision of State and Federal Agencies. RHDV is inactivated by 1% sodium hydroxide (lye) or 1-2% formalin (OIE recommends 3% for disinfecting pelts). Recommended disinfectants are substituted phenolics (e.g. 2% One-stroke Environ®) and 0.5% sodium hypochlorite (bleach).
Notification: Reportable to State and Federal (USDA) agencies; OIE reportable disease

**RABBIT HEMORRHAGIC DISEASE (RHD)/
RABBIT CALICIVIRUS DISEASE (RCD)**

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Due to highly infectious nature, significance of disease, and persistence in the environment, introduction to infected animals is not recommended and may not be permitted by State and Federal agencies

Conditions for restoring disease-free status after an outbreak: State and Federal agencies will make recommendations that may include addition of sentinel animals on treated premises to monitor for persistent virus, minimum post depopulation, disinfection period prior to new animal addition

Experts who may be consulted:

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services, Emergency Management
4700 River Road, Unit 41
Riverdale, MD 20737-1231
Telephone: (301) 734-8073
Fax: (301) 734-7817

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RABIES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals. Major reservoirs in the US include dogs, raccoons, skunks, foxes, and bats. Internationally, vampire bat (Latin America), mongoose species (the Caribbean, southern Africa, and parts of Asia); jackals (parts of Africa); wolves (parts of northern Europe); marmosets (Brazil); ferret badgers (China).	Bites or scratches of infected animals; saliva into open wounds and mucous membranes	Paresthesia or pain at bite site; fever, myalgia, malaise, behavior changes, paresis, seizures and other CNS signs.	Nearly always fatal	Once clinical signs present, no treatment in humans and animals is available although an experimental procedure has been used in humans with limited success.	Eliminating exposure to rabid animals, including vaccination of species for which an approved vaccine exists; providing exposed persons local treatment of wounds and human or equine rabies immune globulin (if not previously vaccinated); vaccinating persons in at-risk professions	Yes

Fact Sheet Compiled by: Erica Lipanovich

Completed on: updated 12 January 2019

Fact Sheet Reviewed by: Donna Ialeggio

Susceptible Animal Groups: All mammals are susceptible.

Causative Organism: The disease rabies is caused by the rabies virus (Family Rhabdoviridae, Genus Lyssavirus) and non-rabies lyssaviruses, such as Australian Bat Lyssavirus, Duvenhage virus, European Bat Lyssavirus, and Mokola virus.

Zoonotic Potential: Bites or scratches of infected animals; saliva into open wounds and mucous membranes

Distribution: Worldwide. Several countries have been declared canine rabies-free. However, the of such declaration is to facilitate waiving the rabies vaccination requirement as these are countries that have not reported recent cases of rabies in land animals and that have adequate disease surveillance for rabies cases, as determined by the CDC. Countries on the list might still have circulating bat lyssaviruses, which can cause the disease in people.

Incubation Period: Incubation is prolonged and variable. The virus typically remains at the inoculation site for a considerable time. In domestic animals, it is generally 1-12 weeks, but can range from several days to months, rarely exceeding 6 months. Virus can be shed for a few days prior to the onset of clinical signs and during illness.

Clinical Signs: Animals will show inappetence, cranial nerve deficits, ataxia, salivating, drooping of lower jaw, acute behavioral changes, such as altered vocalization, aggression, docility, coma, and progressive paralysis. Humans experience pain, paresthesia, and intense pruritus at the bite site; fever, myalgia, malaise, and mood changes that progress to paresthesia, paresis, seizures, coma, and many other neurologic signs. Survival is extremely rare in humans and animals.

Post mortem, Gross or Histological Findings: Gross lesions are often undetectable. Necrotic tonsillitis, necrotic bronchitis, bronchiolitis and alveolitis are commonly seen. Focal areas of necrosis are often found in

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the liver, spleen, lymph nodes and adrenal glands. Histologically, non-suppurative meningoencephalitis is a characteristic lesion in the gray and white matter. Negri bodies, intracytoplasmic eosinophilic inclusions, may be seen in neurons. Mononuclear perivascular cuffing and neuronal necrosis may also be present.

Diagnosis: Rabies diagnosis should be performed in accordance with the established national standardized protocol for postmortem rabies testing by a qualified laboratory that has been designated by the local or state health department. Euthanasia should be accomplished in such a way as to maintain the integrity of the brain so that the laboratory can recognize the anatomical parts. Rabies viral antigen is typically widespread in the brain of rabid animals, though may spread unilateral. It is therefore critical to examine a complete cross section of the brainstem. Rabies diagnosis in animals is accomplished through the direct fluorescent antibody test. Brain tissues examined must include medulla oblongata and cerebellum.

Serological tests are used to monitor antibody titers in response to rabies vaccination.

Human antemortem testing requires a minimum of four samples to rule out rabies. Samples required include saliva, nuchal skin biopsy, serum and cerebral spinal fluid and brain biopsy. Nuchal skin biopsy for immunofluorescent antibody staining is the most reliable test of rabies infection during the first week. Reverse transcription polymerase chain reaction immunofluorescent staining for viral antigen, virus neutralization assays and isolation of infectious virus in cell culture can be performed.

Material Required for Laboratory Analysis: Except in the case of very small animals, such as bats, in which whole animals should be collected, only the head or brain (including brain stem) should be submitted to the laboratory. (<https://www.cdc.gov/rabies/resources/specimen-submission-guidelines.html>). Brain tissues examined must include multiple regions. To facilitate laboratory processing and prevent a delay in testing, any animal specimen being submitted for testing should preferably be stored and shipped under refrigeration and not be frozen. (http://www.cdc.gov/rabies/specific_groups/laboratories/index.html). Chemical fixation of tissues should be avoided to prevent significant testing delays and because it may preclude reliable testing.

Relevant Diagnostic Laboratories:

State and Local laboratories and
Centers for Disease Control and Prevention

Rabies Laboratory

DASH, Bldg 18, Room SSB218

1600 Clifton Road, NE

Atlanta, GA 30333

(404) 639-1050

<https://www.cdc.gov/rabies/pdf/specimen-submission-guideline-508.pdf>

Treatment: No known antivirals currently effective. A few cases of human recovery have been documented following utilization of the “Milwaukee protocol”, an experimental procedure, but failures significantly outnumber successes using this protocol.

Prevention and Control: Vaccination is primary means of prevention. Rabies in humans can be prevented either by eliminating exposures to rabid animals or by providing exposed persons (without a prior history of vaccination) with prompt local treatment of wound washing for 15 minutes combined with the administration of human rabies immune globulin and a series of 4 doses of vaccine. Though not nationally notifiable, some state health departments have made animal bites and use of postexposure prophylaxis (PEP) reportable within their jurisdictions. Individuals that have been previously vaccinated and have a potential rabies exposure require prompt wound care and a series of 2 doses of vaccine. These recommendations, along with information concerning the current local and regional epidemiology of animal rabies and the availability of human rabies biologics, are available from state health departments.

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Suggested Disinfectant for Housing Facilities: Lyssaviruses are not stable in the environment and are inactivated by common disinfectants. The best disinfectants are detergents, hypochlorites, alkalis, Virkon®, and glutaraldehyde.

Notification: Rabies is rare in vaccinated animals. If such an event is suspected, it should be reported to state public health officials, the vaccine manufacturer, and USDA, Animal and Plant Health Inspection Service, Center for Veterinary Biologics at 800-752-6255 or

<https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/veterinary-biologics> .

The laboratory diagnosis should be confirmed, and the virus variant characterized by a rabies reference laboratory. A thorough epidemiologic investigation should be conducted. Because of the risk of rabies in wild animals (especially raccoons, skunks, coyotes, foxes, and bats), the AVMA, CSTE, NACA, and NASPHV strongly recommend the enactment and enforcement of state laws prohibiting their importation, distribution, translocation, and private ownership. Other biting animals which might have exposed a person to rabies should be reported immediately to the local health department. Management of animals other than dogs, cats, and ferrets depends on the species, the circumstances of the bite, epidemiology of rabies in the area, and biting animal's history, current health status, and potential exposure to rabies. Prior vaccination of these animals may not preclude the necessity for euthanasia and testing, merely quarantining.

Measures Required under the Animal Disease Surveillance Plan: The National Association of State Public Health Veterinarians (NAS-PHV) Guidelines for dogs and the Compendium of Animal Rabies Control Guidelines are updated regularly by the NASPHV and provide recommendations

(<http://www.nasphv.org/documentsCompendia.html>). However these guidelines do not supersede state and local laws.

Measures Required for Introducing Animals to Infected Animal: See below.

Conditions for Restoring Disease-Free Status after an Outbreak: Unvaccinated animals exposed to a rabid animal should be euthanized immediately. If the owner is unwilling, the animal should be placed in strict isolation for 6 months. Rabies vaccine should be administered upon entry into isolation or 1 month prior to release to comply with pre-exposure vaccination recommendations. Animals maintained in USDA-licensed research facilities or accredited zoological parks should be evaluated on a case-by-case basis. Rabies virus may be excreted in the saliva of infected animals during illness and/or for only a few days prior to illness or death. A healthy animal which was previously vaccinated that bites a person should be confined and observed daily for 10 days; administration of rabies vaccine is not recommended during the observation period to avoid confusing signs of rabies with possible side effects of vaccine administration. Animals should be evaluated by a veterinarian at the first sign of illness during confinement. If signs suggestive of rabies develop, the animal should be euthanized and the head submitted for testing. Any stray that bites a person may be euthanized immediately and the head submitted for rabies examination. Other biting animals which might have exposed a person to rabies should be reported immediately to the local health department. Management of animals other than dogs, cats, and ferrets depends on the species, the circumstances of the bite, epidemiology of rabies in the area, and the biting animal's history, current health status, and potential for exposure to rabies. Prior vaccination of these animals may not preclude the necessity for euthanasia and testing.

Experts Who May Be Consulted: Public Health Veterinarians can be found at:

Other state and local rabies consultations can be found at:

<http://www.nasphv.org/Documents/StatePublicHealthVeterinariansByState.pdf> (last updated 12 Sept 2018)

<https://www.cdc.gov/rabies/resources/contacts.html> (last updated 31 July 2018)

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- <https://www.avma.org/KB/Policies/Pages/Ownership-and-or-Possession-and-Appropriate-Disposition-of-Wild-and-Exotic-Pet-Species-or-Their-Hybrids.aspx>
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RANAVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Amphibians, especially larvae and metamorphs, fish, and reptiles.	Transmission can occur through direct contact with infected animals; contact with contaminated water or substrates; ingestion of infected tissues or fomites.	Large-scale die offs, especially of larval stages of amphibians. Infection can be sub-clinical. Subtle to severe hemorrhages in the ventral skin, especially at the base of the hind limbs and around the vent opening; fluid accumulation under the skin or within the coelom; hemorrhages within serosa of heart, stomach and liver. Chelonians show swollen eyelids, oral plaques, ulcers on feet.	Infection with <i>Ranavirus</i> is an important cause of mortality in wild amphibians, and chelonia; only occasional reports of this infection in captive animals.	None.	Quarantine any infected animals, Screen incoming amphibians for history of clinical signs consistent with disease. Disinfect all equipment and effluent water.	No.

Fact Sheet compiled by: Ann E. Duncan

Sheet completed on: 15 January 2011; updated 19 August 2013, updated 2018

Fact Sheet Reviewed by: Allan P. Pessier; Amanda Duffus

Susceptible animal groups: All types of amphibians including urodeles (salamanders and newts), and anurans (frogs and toads). Larvae and metamorphs are most often associated with morbidity and mortality. Adult morbidity and mortality occurs less often. Some species may have covert infections and be able to shed and transmit virus to other susceptible animals without ever exhibiting clinical signs. Ranaviruses are also found in other poikilothermic vertebrates including reptiles and fish. Has been associated with mortality events in wild and captive chelonia. Sporadic mortality in captive snakes and lizards. Amphibians may serve as a reservoir

Causative organism: *Ranaviruses* are members of the Iridoviridae, a group of double stranded DNA viruses. Numerous strains are identified; however, viruses related to the *Ambystoma tigrinum* virus (ATV) and Frog virus 3 (FV3) appear to be the most important in North America. The Bohle iridovirus (BIV) from Australia also is of concern as it has recently been identified in a zoological collection in the USA. Some ranaviruses are able to infect animals from more than one class (e.g. amphibians and reptiles or amphibians and fish).

Zoonotic potential: None

Distribution: Worldwide although hotspots have been identified in recurrent mortality events. Ranaviruses are considered to be globally emerging infections.

Incubation period: Variable: Less than 5 days to several weeks. Incubation is affected by ambient temperatures, dose of virus exposure, immunosuppression, developmental stage, and species differences in susceptibility to different *Ranavirus* strains.

Clinical signs: In amphibians, subtle to severe hemorrhages in the ventral skin, especially at the base of the hind limbs and around the vent opening; fluid accumulation under the skin or within the coelom; hemorrhages within serosa of heart, stomach and liver. Skin ulceration and/or epithelial proliferation may be seen. Infection does not always cause clinical disease. Long-term nonclinical carriers have been identified. In chelonian, nasal discharge, conjunctivitis, caseous plaques in the oral cavity and subcutaneous edema of the palpebra and neck have been seen.

Post mortem, gross, or histologic findings: In amphibians, necrosis and/or hemorrhage is present in multiple tissues, especially skin, liver, kidney, spleen/ hematopoietic tissue and gastrointestinal tract. In chelonians, necrotizing and fibrinous stomatitis/esophagitis, splenitis and vasculitis are seen. Histologically intracytoplasmic inclusion bodies may be seen; however, they are difficult to identify, are not always due to the virus, and may be absent or inconspicuous in many cases.

Diagnosis: PCR is the most useful test and is becoming more widely available. Real-time PCR techniques

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allow detection of smaller amounts of virus, but to identify the group type (ATV or FV3 virus-like) of *Ranavirus* present conventional PCR with DNA sequencing is required. Determining the specific species of *Ranavirus* usually requires cell culture, virus isolation, and molecular characterization. These techniques are not widely available outside of research laboratories. Conventional PCR may not detect low level infections and can provide false-positive results if confirmatory DNA sequencing or Southern blot analysis is not performed. Histopathology is helpful to screen for lesions in sick animals, but lesions tend to be nonspecific unless intracytoplasmic inclusion bodies are seen. Virus isolation, immunohistochemistry, transmission electron microscopy, cell culture, and serology (not widely available or validated for most species) have also been used to identify infected animals.

Material required for laboratory analysis: The best choice is tissue samples collected at necropsy, especially liver, kidney and, if lesions are present, skin. Frozen tissues are required for virus isolation and are generally best for molecular analysis; however, freezing is not acceptable for histology. For histology, tissues should be submitted fresh or fixed in 70% ethanol or 10% neutral buffered formalin. Ethanol-preserved tissues may be used for some molecular testing. Formalin-fixed tissues may also be used for some molecular testing if the length of time in formalin is minimal at days to weeks but it is possible to perform PCR on paraffin embedded tissues. Samples can also be collected from clinically ill living animals such as cloacal or pharyngeal swabs, tissue biopsy (tail clips) or blood. Contact the laboratory to determine the best swab choice for testing, as some can inhibit detection. If living animals are tested, results should be interpreted with caution recognizing test limitations (e.g., a positive test result is more meaningful than a negative test result). Test sensitivity for antemortem PCR increases with time post-exposure and development of clinical signs of illness. Contact individual laboratories for more information regarding screening.

Relevant diagnostic laboratories:

For an overall list <https://www.ranavirus.org/resources/testing-labs/>

Amphibian Disease Laboratory; Taqman PCR for Ranavirus; Conventional PCR and MCP sequencing
15600 San Pasqual Valley Road
Escondido, CA 92027
(760) 747-8702 x 5471
http://www.sandiegozooglobal.org/News/Amphibian_Disease_Laboratory/

Diagnostic or research: Conventional PCR, qPCR, virus culture, MCP sequencing, histopathology:
University of Tennessee Center for Wildlife Health
274 Ellington Plant Sciences Building
2431 Joe Johnson Drive
Knoxville, Tennessee 37996-4563
(865) 974-7948
dmill42@utk.edu or mgray11@utk.edu

qPCR, cell culture, genomic sequencing and speciation:
Zoo Medicine Infectious Disease Lab
c/o April Childress
University of Florida
2015 SW 16th Ave
Building 1017 Room V2-186
Gainesville, FL 32608
Phone 352-294-4420
ChildressA@ufl.edu
<http://labs.vetmed.ufl.edu/sample-requirements/microbiology-parasitology-serology/zoo-med-infections/>

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qPCR:

Zoologix
9811 Owensmouth Avenue, Suite 4
Chatsworth, CA 91311-3800
Phone: 818-717-8880
Fax: 818-717-8881
Email: info@zoologix.com

Treatment: None in amphibians is available. Antiviral therapy and supportive care have been attempted in reptiles.

Prevention and control: The major concerns in captive programs are that mortality will occur in a valuable species or population or that subclinically infected animals will expose naïve wild populations. The prevalence of infection in captive animals is not yet known. Disease has likely gone unrecognized due to clinical and pathological similarities to other diseases in amphibians. Captive amphibian populations can be surveyed continuously for disease by histopathology testing of samples collected at necropsy and PCR. Once a population or individual has been found positive by PCR the disposition of these animals will depend on careful risk assessment. A positive test does not distinguish between a lethal infection and a subclinical carrier. Factors to be considered include their importance to the survival of the species, the presence or absence of pre-existing infection in captive and wild populations and results of follow-up histologic and PCR testing. In some cases, the animals or a population may be managed in permanent isolation from the general amphibian population. Further prevention measures include quarantining all incoming animals. The health history of animals being brought into a population needs to be reviewed- if there have been deaths or illness due to confirmed or suspected *Ranavirus* in the prior 6 months the risk of disease transmission with introduction is considered higher. Animals dying during quarantine can be screened using PCR and histopathology. Strict biosecurity measures must be followed to avoid transmission of infection to other amphibians or susceptible classes of animals (fish, turtles, tortoises).

Suggested disinfectant for housing facilities: 1% Potassium peroxydisulfate (Virkon®), 3% sodium hypochlorite and 1% chlorhexidine have been reported to be effective at inactivating *Ranavirus* after 1 min. contact duration. Some ranaviruses were found to remain viable for 113 days on dry surfaces and up to 2 weeks in water. Amphibians are sensitive to disinfectant residues- thorough rinsing is required after use. Biosecurity measures must include treatment of waste and effluent from *Ranavirus* infected animals.

Notification: Infection by a *Ranavirus* is classified as a reportable disease by the OIE requiring proof of *Ranavirus*-negative results before commercial shipment of amphibians (OIE 2008).
http://www.oie.int/eng/normes/fcode/fcode2008/en_chapitre_2.4.2.htm. A reporting mechanism (e.g. via USDA-APHIS) has not been announced for the US at this time.

Measures required under the Animal Disease Surveillance Plan: Currently none. See http://www.oie.int/eng/normes/fcode/fcode2008/en_chapitre_2.4.2.htm as Article 2.4.2.10. states that importation of live aquatic animals intended for use in zoos from a country not declared free from *Ranavirus* should be followed by lifelong holding of the animals in biosecure facilities for continuous isolation from the local environment and treatment of all effluent and waste materials in a manner that inactivates *Ranavirus*.

Measures required for introducing animals to infected animals: Animals should not be introduced to those showing clinical signs of disease or with exposure to known infected animals.

Conditions for restoring disease-free status after an outbreak: None established.
See: http://www.oie.int/eng/normes/fcode/fcode2008/en_chapitre_2.4.2.htm

Experts who may be consulted:

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American Association of Zoo Veterinarians Infectious Disease Manual
CHIMPANZEE CORYZA/RESPIRATORY SYNCYTIAL VIRUS (RSV)

masks, gloves, and hand washing. Highly transmissible. No vaccine is available.

Suggested disinfectant for housing facilities: Virus is readily inactivated by most disinfectants (i.e., quaternary ammonium compounds, phenols). It usually lasts only hours in environment, although can persist longer in cool, shady areas or in serum or tissue debris, transmission via fomites (i.e., enrichment items, cage furniture)

Notification: Reportable in humans in many states, check individual state regulations.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: No long term immunity, and no carriers. Introduction after clinical signs have resolved and area is disinfected would be optimal.

Conditions for restoring disease-free status after an outbreak: Resolution of clinical signs; some immunocompromised humans can shed for up to 4 weeks, though usual time of shedding is 3-8 days.

Experts who may be consulted: CDC

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RICKETTSIOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals	Vector-borne, primarily ticks but some species are transmitted by fleas	Non-specific	Non-clinical or mild to severe including death.	Doxycycline	Avoid contact with ticks and other ectoparasites. No vaccine available.	Many species are zoonotic.

Fact Sheet compiled by: Michael J. Yabsley

Sheet completed on: 1 August 2013, updated 2018

Fact Sheet Reviewed by: Edward B. Breitschwerdt; Kristina M. Delaski; Gail Miriam Moraru

Susceptible animal groups: For those *Rickettsia* species that are tick-borne, ticks serve as the definitive and reservoir hosts for these bacteria, but numerous vertebrate hosts are important as they serve as blood-meals for ectoparasites and some can serve as amplifying hosts for *Rickettsia* spp. Antibodies to *Rickettsia* spp. have been reported in a wide range of wildlife and domestic animal species.

Causative organism: The four main *Rickettsia* species that are known to cause disease in people and/or animals in the United States are:

Rickettsia rickettsii, the causative agent of Rocky Mountain Spotted Fever, is transmitted by ticks (primarily *Dermacentor* spp. and rarely by *Amblyomma americanum*). In Arizona (USA), transmission to dogs and people has been documented by *Rhipicephalus sanguineus*.

Rickettsia parkeri, causative agent of Parkeri Rickettsiosis or American Boutonneuse Fever, is transmitted by ticks (primarily *Amblyomma maculatum* and rarely *A. americanum*) and frequently causes an eschar.

Rickettsia philipii (*Rickettsia* 364D), causative agent of Pacific Coast tick fever, an eschar-associated febrile disease in people, is transmitted by *Dermacentor occidentalis*.

Rickettsia typhi (endemic or murine typhus) is transmitted by *Xenopsylla cheopis* usually infesting rats.

Rickettsia felis, (commonly referred to as cat flea typhus) which is transmitted by *Ctenocephalides felis*, is endemic to all continents except Antarctica.

Other species of *Rickettsia* have been detected in the US but most are considered endosymbionts of ticks (i.e., these species aren't known to induce disease in vertebrate hosts). However, in recent years, some of these endosymbionts (e.g., *Rickettsia amblyommatis*) have been associated with mild disease in people. Outside of the US, numerous of *Rickettsia* species exist, many of which are zoonotic.

Zoonotic potential: Many species, but not all, are zoonotic.

Distribution: *Rickettsia* spp. have been reported world-wide. *R. rickettsii* and *R. felis* are distributed throughout the Americas while *R. parkeri* is found in the southeastern US and *R. philipii* occurs in California. *R. typhi* and *R. felis* are widely distributed throughout the world.

Incubation period: Typically 3-14 days.

Clinical signs:

People: Wide range of symptoms from asymptomatic to severe potentially fatal disease. Mild or asymptomatic cases rarely diagnosed. Some individuals develop a fever, muscle pain, headache, and rash (due to damage of vascular endothelial cells), but, importantly, a rash is not always observed with rickettsioses. Multi-organ disease results in high mortality rate if not treated. Infections with *R. parkeri* and *R. philipii* tend to be less severe than *R. rickettsii* and often present with an eschar at the site of tick attachment. Neurologic signs may develop in people infected with *R. typhi* or *R. felis*.

Canines: Canines are susceptible to *R. rickettsii* and can develop severe disease rapidly, although most infections are asymptomatic or mild. Dogs can develop similar clinical signs as people. The most common clinical signs include fever, lethargy, anorexia, ataxia, rash, swollen lymph nodes, and localized edema.

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Other animals: Most other animals only have short-term infections with no associated disease. These animals as well as others that don't become ill develop antibodies that can be detected by serologic testing.

Clinical pathological, gross, and histopathological findings: Thrombocytopenia is common. Leukopenia followed by a leukocytosis and mild anemia may develop. Petechiae and ecchymoses are common due to damage to endothelial cells.

Diagnosis: These diseases can be difficult to diagnose but diagnosis is based on clinical signs, exposure to ectoparasites (ticks/fleas), and supporting data from laboratory findings, serology, and/or molecular assays. Ideally, acute and convalescent serum samples are tested for antibodies. Molecular testing of petechial skin biopsies (or blood, although this sample is less rewarding) can be used. Fluorescent antibody (FA) or molecular testing of tissues can be used to diagnose cases post-mortem. Because clinical signs may develop quickly, lack of a serologic response doesn't preclude infection. PCR testing has not been widely used to document active infection in wildlife species.

Material required for laboratory analysis: Serum, EDTA blood for PCR, skin biopsy, and/or tissue samples.

Relevant diagnostic laboratories:

Humans: Many state diagnostic labs have testing capabilities.

Animals:

North Carolina State University
College of Veterinary Medicine
Vector Borne Disease Diagnostic Laboratory
1060 William Moore Drive
Room 462A
Raleigh, NC 27607
919-513-8279
<http://www.cvm.ncsu.edu/vhc/csds/ticklab.html> (serology and PCR)

Antech Diagnostics

Corporate Headquarters:
17672-B Cowan Avenue
Irvine, CA 92614

ANTECH West	1-800-745-4725
ANTECH East	1-800-872-1001
ANTECH Canada	1-800-341-3440
ANTECH Test Express	1-888-397-8378

(serology)

Zoologix Inc.

9811 Owensmouth Avenue
Suite 4
Chatsworth, CA 91311-3800
Phone: 818-717-8880
Fax: 818-717-8881
Email: info@zoologix.com

(This PCR does not differentiate among *Rickettsia* spp.)

Treatment: The most common treatment is doxycycline, usually 10 - 20 mg/kg every 12 hours for 7 days. A lower dose (5 mg/kg every 12 hours) can also be given for 14 days. Chloramphenicol can also be used.

Prevention and control: Because *Rickettsia* spp. are vector-borne, limiting exposure to vectors is necessary to prevent transmission. Transmission doesn't occur from animal to animal, but can occur through blood

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inoculation of wounds. Habitat modification to limit ticks in areas where animal frequent. Some birds are known hosts for certain tick species, and while they may not be competent hosts of the rickettsial pathogens, they can aid in distribution of vectors.

Suggested disinfectant for housing facilities: *Rickettsia* spp. are not viable outside of the host. Prevent vector exposure. Application of acaricides and removal of leaf litter can decrease tick abundance.

Notification: CDC Reportable Disease for human cases in US

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: These bacteria are vector-borne so direct contact between animals is not a risk factor for infection. However, ectoparasite prevention should be implemented.

Conditions for restoring disease-free status after an outbreak: n/a

Experts who may be consulted:

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RIFT VALLEY FEVER

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Domestic ruminants, some wild ruminants, humans, some primates, gray squirrels, some rodents, newborn cats and dogs.	Vector-borne; direct contamination in humans. Virus harbored in mosquito eggs, remains dormant. Periodic heavy rains and flooding cause mosquito hatching and virus transmission and amplification in mammalian hosts.	Epizootic, abortion storms in pregnant ewes, high fever, weakness, gastro-intestinal signs, mortality.	May be inapparent, mild or fatal. Abortions may reach 100%. High mortality in susceptible young and pregnant animals.	None, supportive care.	Animal movement, remove infected animals and vector control. Vaccination in endemic areas. Barrier protocols for exposed humans.	Yes

Fact Sheet compiled by: Thomas W deMaar; updated by Mark. W. Atkinson

Sheet completed on: 21 January 2011; updated 26 August 2013

Fact Sheet Reviewed by: John C. Morrill, Pierre Rollin

Susceptible animal groups: Sheep, cattle, goats, African buffalo, water buffalo, Asian monkeys and humans can be infected. Susceptibility of cervids is not known. Death in wild African ruminants is rare but there are recent reports of abortion and/or deaths with virus isolation in African buffalo, wildebeest, waterbuck, giraffe, sable, springbok and impala. Camels, African monkeys, baboons, equids, pigs and domestic carnivores are considered resistant experiencing only asymptomatic viremia. Gray squirrels, mice, hamsters and newborn dogs and cats can be experimentally infected but don't usually play a role in the transmission.

Causative organism: RVF virus is an RNA *Phlebovirus* of the family Bunyaviridae. Only one serotype is recognized but strains of variable virulence exist. Virus circulates in endemic areas among wild ruminants and hematophagous mosquitoes; certain *Aedes* species act as reservoirs during inter-epizootic periods and increased precipitation in dry areas leads to an explosive hatching of eggs. Precipitation cycles of 5-25 years produce RVF-immuno naïve animal populations, and introduction of virus can lead to explosive outbreaks. Virus can be transmitted by many species of mosquitoes and other biting insects during viremic phase in mammalian hosts.

Zoonotic potential: Humans infected via contact with nasal discharge and blood from viremic animals as well as aborted fetuses and vaginal secretions following abortion in animals, mosquitoes, and by aerosols and possibly, though unproven, by consumption of raw milk. It is possible that humans can act as amplifying hosts. Generally, raw meat is not a source although it can contain viremic blood, and for humans, it is usually cooked.

Distribution: Serologic or virologic evidence over most of Africa. Considered endemic in sub-Saharan Africa but recently it has made incursions into some Middle Eastern countries and Madagascar.

Incubation period: 1-6 days; 12-36 hrs in lambs.

Clinical signs: Abortion storms occur in domestic livestock at any stage of pregnancy. Biphasic fever up to 106° F (up to 104° F in humans, to 107° F in sheep.) Young animals more severely affected showing high fever, listlessness and unwillingness to move; up to 90% mortality in newborn and young animals after very short incubation period. Affected animals die within 24-36 hours and are often just found dead without exhibiting clinical signs. Older susceptible animals (> 2 weeks of age) show high fever,

RIFT VALLEY FEVER

listlessness, anorexia and weakness and often develop a high titered viremia. Gastrointestinal signs are common: abdominal pain, regurgitation, foul smelling bloody diarrhea, and icterus. Abortion maybe the only sign (40-100% in sheep). Adults may have inapparent infections with abortion being the only sign. Mortality in adult sheep ranges from 20 to 70% and approximately 10% in adult cattle. Camels present either hyperacute form, with sudden death in <24 hours; or and an acute form with fever, ataxia, dyspnea, blood-tinged nasal discharge, icterus, severe conjunctivitis, hemorrhages of gums and tongue, foot lesions, nervous symptoms, and abortions. Humans experience a febrile disease that is usually mild and transient but in rare cases can be fatal with hemorrhagic fever, ocular disease (retinal vasculitis), liver disease and meningoencephalitis.

Post mortem, gross, or histologic findings: Focal or generalized hepatic necrosis; enlarged, discolored, soft, friable liver with irregular congestion and white necrotic foci (~1 mm diameter). Lesions are most severe in aborted fetus and young animals. Widespread cutaneous hemorrhages, petechiae and ecchymoses on serosal membranes. Gall bladder wall edematous with possible hemorrhage. Spleen and lymph nodes are edematous, enlarged and may show petechiae. Hemorrhagic enteritis, intestinal contents dark chocolate-brown.

Diagnosis: It is suspected in endemic areas when presented with abortions and relevant signs combined with febrile disease in humans after heavy rains and/or flooding. Histopathology of liver is relevant. Most tissues will contain virus and can be used for detection (virus isolation, PCR, ELISA antigen detection) and numerous serologic tests exist: VN, ELISA, IgG and IgM.

Material required for laboratory analysis: Blood, liver, spleen, brain and aborted fetuses are tissues of choice.

Relevant diagnostic laboratories:

Centers for Disease Control and Prevention
 Viral Special Pathogens Branch
 1600 Clifton Road NE
 Atlanta, GA 30333
 Phone: (404) 639-1115 or (404) 639-1510
Contact prior to specimen submission

Treatment: No specific treatment is available but supportive care can be provided.

Prevention and control: Vector control and prevent movement of livestock are important measures for managing this disease. General barrier measures (gloves, masks, goggles, etc) should be used when handling suspected materials. Attenuated (Smithburn strain) and inactivated virus vaccines available for use in Africa. No licensed vaccine for use in US but several live-attenuated mutant vaccines are undergoing experimental analysis. The Smithburn vaccine strain is known to cause abortion and birth defects so immunization of pregnant animals is not advised.

Suggested disinfectant for housing facilities: While this is not usually performed, virus is susceptible to acidic solutions, lipid solvents and hypochlorite solutions.

Notification: Reportable to USDA National Animal Health Reporting System (A080)

Measures required under the Animal Disease Surveillance Plan: None described but response would be massive.

Measures required for introducing animals to infected animal: Unadvised

Conditions for restoring disease-free status after an outbreak: A disease free period with active surveillance longer than 4 years. Recovery probably confers lifelong immunity.

Experts who may be consulted:

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 Dept. of Microbiology & Immunology

RIFT VALLEY FEVER

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ROTAVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, including humans; and certain avian species	Fecal-oral	Diarrhea and other signs of enteritis, including inappetence, and lethargy	Self-limiting to severe	Supportive, correcting dehydration, acid-base imbalance; antibiotics to prevent secondary infection	Vaccines available for humans, cattle, horses, and pigs, poultry	Yes, potentially

Fact Sheet compiled by: Meredith M. Clancy

Sheet completed on: 15 October 2018

Fact Sheet Reviewed by: Cara Field

Susceptible animal groups: Ruminants, including non-domestic bovids, antilocaprids (pronghorn), cervids, and giraffids. Rotaviral enteritis also documented in poultry and wild birds, ferrets, rabbits, guinea pigs, felids, canids, camelids, equids, and domestic pigs.

Rotaviruses, especially of Group A, are the most common cause of severe diarrhea in children under 5 years of age. Rotavirus infections are considered species-specific, but re-assortment of the virus between species may occur.

Causative organism: Rotaviruses (family Reoviridae) are generally named after the species where it was first found: Bovine Rotavirus, Porcine Rotavirus, Feline Rotavirus, Canine Rotavirus, etc. Much diversity exists in these viruses due to their genomes' ability to mutate, reassert and rearrange. In human medicine and virology, rotaviral isolates are grouped according to antigens present using A – E, with Group A being the most prevalent cause of illness in humans, but Group C can also cause outbreaks.

Zoonotic potential: Animal rotaviruses are reservoirs for genetic exchange with human rotaviruses, and animal rotaviruses can infect humans, both naturally and experimentally.

Distribution: Worldwide

Incubation period: Variable – from 15h to 5d

Clinical signs: Enteritis resulting in diarrhea is nearly always the presenting sign, sometimes pale yellow or mucoid in character. Lactose-intolerance may be present due to the lack of lactase secretion by enterocytes—an important sign in nursing animals. Other clinical signs may include fever, inappetence, dullness, and progressive dehydration causing metabolic acidosis, which if severe enough can lead to death.

Post mortem, gross, or histologic findings: Gross lesions include thinning of the intestinal walls with sequestration of fluid into the small intestine leading to marked distention of the intestines and abdomen. In young animals, non-digested milk may be present in the intestine. Depending on the strain's virulence, lesions may present in only localized areas of the jejunum, or may be throughout the small intestine and into the large intestine. Rotaviruses infect mature enterocytes on the villi surface in the small intestine, leading to villous atrophy and blunting with club-shaped, stumpy villi that are often fused. Crypt epithelium is often hyperplastic while trying to recover the lost villous enterocytes. Columnar epithelium is lost and replaced with cuboidal or squamous epithelium.

Diagnosis: Electron microscopy (EM) can be used as a screening tool to identify virus in the feces. EM alone is not sufficient to diagnose rotavirus as the cause of diarrhea; comparative levels with nonclinical animals are used in cattle to support diagnosis. Antigen detection can be performed via enzyme-linked immunosorbent assay (ELISA), commonly used to diagnose rotavirus.

Enzyme immunoassays (EIA) point-of-care tests exist for human medicine that have been validated in detecting bovine rotavirus. Latex agglutination testing can also be used to detect Group A rotavirus antigen. Polymerase chain reaction (PCR), including reverse-transcriptase qPCR can both detect rotavirus and differentiate between species. Indirect fluorescent assay (IFA) can detect antigen in tissue, generally using

ROTAVIRUS

post-mortem samples. Serology is generally noncontributory, as rotavirus exposure is often widespread and results are nonspecific.

Material required for laboratory analysis:

PCR has become the most widely available test
 Feces for ELISA, EIA, latex agglutination, PCR
 Fresh tissue (small intestine) for IFA

Relevant diagnostic laboratories:

Michigan State University Diagnostic Center for Population and Animal Health

PCR: Bovine, Equine, Ferret, Porcine

Clinical Pathology Laboratory

A215 Veterinary Medical Center

Michigan State University

East Lansing, MI 48824-1314

(517) 353-1683

<https://www.animalhealth.msu.edu/>

Texas A&M Veterinary Diagnostic Laboratory

Electron microscopy, PCR: bovine

College Station Laboratory

PO Box Drawer 3040

College Station, TX 77841-3040

Phone: (979) 845-3414

Fax: (979) 845-1794

<http://tvmdl.tamu.edu/>

Point of care testing: ImmunoCard STAT!® Rotavirus test is available through numerous suppliers, produced by Meridian Bioscience <http://www.meridianbioscience.com/diagnostic-products/rotavirus-and-adenovirus/immunocard/immunocard-stat-rotavirus.aspx>

Treatment: Treatment relies on correction of dehydration and metabolic acidosis, using IV fluid resuscitation or oral rehydration solutions and bicarbonate given orally or IV to address acidosis. Antibiotics are often used to prevent secondary bacterial infections via the compromised gastrointestinal tract. Zinc is used adjunctively in management of human rotavirus.

Prevention and control: In ruminants, colostrum often contains antibodies (IgA) to rotavirus in herds where rotavirus is naturally circulating, but the calf's antibody concentrations decline sharply after one week. Vaccination of the dam 1-3m prior to calving increases circulating antibodies in the milk and helps reduce rotavirus in calves. Vaccination strategies differ among practitioners for nondomestic hoofstock. In species without viable vaccine, prevention and control are best achieved by reducing fecal contamination of the environment through routine cleaning and removal of feces, disinfection of enclosures and all material the animal contacts. Isolation of sick individuals and quarantine of new animals is important to reduce exposure of naïve animals to shed virus. In production animals, the all-in/all-out technique is used to reduce exposure and contamination.

Suggested disinfectant for housing facilities: Rotaviruses are hardier than coronaviruses and other diarrheal viruses. Disinfectants that are reported to be effective include formaldehyde (0.25%), phenol (2%), sodium hypochlorite (1%), quaternary ammonium compounds, and iodophores. Cleaning, steaming, and disinfecting of housing facilities is recommended.

Notification: Not reportable to USDA or OIE

Measures required under the Animal Disease Surveillance Plan: N/A

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Measures required for introducing animals to infected animal: Not recommended. Animals that have been naturally infected may have short-lived immunity via mucosal and cell-mediated immunity, however, so can be reintroduced once convalesced.

Conditions for restoring disease-free status after an outbreak: N/A

Experts who may be consulted:

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SALMONELLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Most vertebrates	Fecal-oral	Mild: gastroenteritis with vomiting, diarrhea. Severe: additionally anorexia, lethargy, pyrexia, dehydration. Severe acute septicemia: systemic infection possible	Varies from subclinical carriage to septicemia and death. Septicemic form often is fatal without prior or unobserved clinical signs.	Antibiotics essential for septic salmonellosis; controversial for enteric infection	Biosecurity essential Pest control Sanitation Vaccination with autogenous bacterin available	Yes

Fact Sheet compiled by: Cornelia J. Ketz-Riley; updated by Meredith M. Clancy

Sheet completed on: 18 December 2018

Fact Sheet Reviewed by: Lana Krol

Susceptible animal groups: Non-typhoidal salmonellosis causes natural infection in all taxa of vertebrates. Reptiles are important carriers, but multiple exotic pet species have been implicated in human disease outbreaks. Only humans are susceptible to *S. typhi*, the causative agent of typhoid.

Causative organism: Family: Enterobacteriaceae; Genus: *Salmonella*

-*Salmonella enterica* has 6 subspecies, but common language can abbreviate the serotype. *Salmonella enterica* subsp. *enterica* serotype Typhimurium can be abbreviated to *Salmonella* Typhimurium.

- *S. enterica* subspecies *enterica* (I) with common serovars
 - S. enteritidis
 - S. paratyphi
 - S. typhimurium
 - S. typhi
 - S. pullorum
 - S. gallinarum
- *S. enterica* subspecies *salamae* (II)
- *S. enterica* subspecies *arizonae* (IIIa)
- *S. enterica* subspecies *diarizonae* (IIIb)
- *S. enterica* subspecies *houtenae* (IV)
- *S. enterica* subspecies *indica* (VI)

There are over 2600 extant serovars recognized determined by phenotyping of the O (somatic) and H (flagellar) antigens. Nomenclature for this genus has constantly evolved, leading to some inconsistencies in the literature, particularly for serovars from subspecies *arizonae* (IIIa) and *diarizonae* (IIIb) which were once listed in their own genus (*Arizona*) or species (*Salmonella arizona*), and some laboratories fail to differentiate between these 2 during initial biochemical testing.

- *S. bongori*, formerly *S. enterica* subspecies V is of less veterinary importance.

For nomenclature clarify, WHO, CDC, and Institut Pasteur use the Kauffman-White scheme for naming serovars, the most recent of which is found at: https://www.pasteur.fr/sites/default/files/veng_0.pdf

Zoonotic potential: High

Distribution: Worldwide

Incubation period: Generally 1-4 days.

SALMONELLOSIS

Clinical signs:

Acute: gastroenteritis (including vomiting and diarrhea), pyrexia, and anorexia.

Severe/septicemic: lethargy, polydipsia, dehydration, petechial hemorrhages on cutaneous and mucosal surfaces, joint pain (polyarthritis), abdominal pain, respiratory signs, neurological signs; possibly death

Chronic: reduced productivity such as egg and milk production, suppressed growth, decreased fertility, decreased hatchability, and abortion.

Post mortem, gross, or histologic findings: Most common findings during gross necropsy include signs of dehydration, gastroenteritis, hepatomegaly with or without miliary white foci, splenomegaly, and mesenteric lymphadenopathy. Pneumonia can be observed more often in birds and calves. In cases of septicemia, petechial hemorrhages can occur in multiple organs, with muscular necrosis typically involving myocardial and gizzard (in avian species) muscle, nephropathy, polyserositis, and synovitis commonly found. Histopathological findings include multifocal necrotic hepatitis, necrosis of cryptic or surface enterocytes in lower small intestines, cecum and colon.

Diagnosis: Culture of fresh fecal material is still the most commonly used diagnostic tool to detect *Salmonella* shedding. PCR can be used to evaluate shedding with a quicker turn-around time than culture.

Historically, serotyping was performed on isolates to elucidate course of disease in individuals and epidemiology in populations. Molecular techniques such as pulsed-field gel electrophoresis now allow for more exact epidemiologic tracing.

Serological examinations can be used to establish presence of *Salmonella* on herd basis, but are not reliable for individual animal status identification, although have been used to evaluate vaccination response and flock exposure.

Material required for laboratory analysis: For culture or PCR, feces, organ tissue, whole blood, milk or other environmental material are recommended. Serum is best used for serology such as ELISA.

Relevant diagnostic laboratories: Any laboratory that is set up for culture methods can be used for first screening for *Salmonella*. Serotyping is most commonly sent to the National Veterinary Services Laboratories.

Treatment: Mild infections are self-limiting and are only treated with supportive care, such as rehydration, electrolytes, and analgesics. Antibiotic therapy is controversial as elimination is rare, re-infection common, and creation of a carrier state a likely outcome. Animals treated with antibiotics have shown prolonged bacterial shedding post-treatment. Antibiotics are generally used for suspected sepsis or in immunocompromised or young animals where sepsis is likely. While ideally antibiotics are based on antimicrobial resistance patterns, commonly used antibiotics include trimethoprim-sulfonamide combinations, ampicillin, fluoroquinolones, and third-generation cephalosporins, although resistance to nearly all classes have been reported in some isolates.

Prevention and control: Eradication is difficult due to asymptomatic carriers. Preventive control programs should consist of a good biosecurity protocols. Multiple non-pharmaceutical therapeutic measures, including food and water additives such as probiotics, have been tried to increase intestinal immunity. Vaccination is not possible for most taxa of animals, although vaccines exist for production animals (poultry, cattle, and swine), and autogenous vaccines may be produced for local use.

Suggested disinfectant for housing facilities: Most commonly used disinfectants, such as diluted hypochlorite, quaternary ammonium based products are effective against *Salmonella* sp.

Notification:

- Fowl Typhoid (*Salmonella enterica* subsp. *enterica* Gallinarum) and serovar Pullorum (*Salmonella* Pullorum), reportable to USDA and OIE
- *Salmonella* Abortusovis, reportable to USDA and OIE

Positive laboratory tests are often reportable and various serovars may be reportable in particular states or jurisdictions.

Measures required under the Animal Disease Surveillance Plan: Salmonellosis is part of the National Animal Disease Surveillance Plan, due to its importance as a foodborne bacterial illness. Specific measures required depend on the animal species and nature of the outbreak.

SALMONELLOSIS

Measures required for introducing animals to infected animal: Regular quarantine in a clean environment; reduce access to host animals; separate tools and personnel for quarantined animals

Conditions for restoring disease-free status after an outbreak: Quarantine of whole collection; isolation of sick and potentially infected animals; testing of any potentially contaminated feed, water, surface and also healthy animals, as well as personnel, before giving access to previously contaminated area. Multiple cultures of potentially infected animals necessary due to inconsistent shedding of bacteria.

Experts who may be consulted:

Centers for Disease Control and Prevention
Division of Foodborne, Waterborne, and Environmental Diseases
1600 Clifton Rd
Atlanta, GA 30333
800-CDC-INFO

USDA–APHIS–VS–Center for Epidemiology and Animal Health
NRRC Building B, M.S. 2E7
2150 Centre Avenue
Fort Collins, CO 80526-8117
970-494-7000
E-mail: NAHMS@aphis.usda.gov
<http://nahms.aphis.usda.gov>

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SCHISTOSOMIASIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Old World monkeys, great apes, humans, dogs, cows, rats, water buffaloes, pigs	Percutaneous in contaminated water	<i>S. mansoni</i> , <i>S. japonicum</i> : fever, nausea, cough, diarrhea, abdominal pain, gastroesophageal bleeding, CNS signs; <i>S. haematobium</i> : hematuria, dysuria, SCC of the bladder	Sometimes fatal; more often a chronic disease	Praziquantel	Snail control, good sanitation, access to clean water	Yes, via snail vector

Fact Sheet compiled by: Christine Fiorello

Sheet completed on: January 25, 2011; updated 1 November 2012

Fact Sheet Reviewed by: Sara Childs Sanford, Walter Boyce

Susceptible animal groups Natural infections of *S. japonicum* have been reported in nearly 50 mammalian species, including humans, rhesus macaques, dogs, cats, rats, pigs, water buffalo, cows, horses, donkeys, goats, rabbits, wild carnivores, wild pigs, wild rodents, shrews, hedgehogs. Many more primates and other species have been experimentally infected. The most important species thought to maintain the disease in natural transmission cycles include humans, dogs, cows, and pigs. *S. haematobium* infects humans, and hybridizes with *S. bovis* to infect cattle.

Causative organism *Schistosoma japonicum*, *S. mansoni*, *S. haematobium*

Zoonotic potential Yes, via a snail vector. Humans are the most common host for *S. mansoni* and *S. haematobium*, but *S. japonicum* infects many domestic and wild mammals that can serve as reservoirs of the fluke. Old World monkeys, including baboons and vervet monkeys, are hosts for *S. mansoni*. These host species are commonly found around human settlements and share water sources with humans.

Distribution *S. mansoni*: Africa, Arabian peninsula, South America; *S. japonicum*: China, Phillipines, Indonesia; *S. haematobium*: African, Arabian peninsula

Incubation period 4-6 weeks (although signs due to the acute phase of infection may be immediate)

Clinical symptoms *S. japonicum* and *S. mansoni* (acute phase): fever, nausea, cough, diarrhea (chronic phase): anemia, bloody diarrhea, gastro-esophageal bleeding, hepatomegaly, splenomegaly, cirrhosis, cachexia, ascites, portal hypertension, pulmonary hypertension. *S. haematobium*: hematuria, dysuria, ureteral obstruction, hydronephrosis, squamous cell carcinoma of the bladder

Post mortem, gross, or histologic findings Portal and periportal hepatic fibrosis, hepatosplenomegaly, gastroesophageal varices, granulomatous hepatic inflammation, mesenteric lymphadenopathy, colonic ulceration, urinary bladder and ureteral fibrosis, hydronephrosis

Diagnosis Fecal sedimentation or centrifugation, Falcon assay screening test (FAST) ELISA, IgG-ELISA, PCR. Urine centrifugation (*S. haematobium*)

Material required for laboratory analysis Feces, serum, urine

Relevant diagnostic laboratories: Any commercial lab should be able to find ova in feces or urine; ARUP Laboratories in Salt Lake City, UT can perform antibody testing (800 522-2787; aruplab.com)

Treatment Praziquantel is the treatment of choice; it should be repeated in 4-6 weeks. Recently, resistance to praziquantel is being recognized in some areas.

SCHISTOSOMIASIS

Prevention and control Snail control, improved sanitation, access to clean water
Suggested disinfectant for housing facilities Niclosamide 10 mg/l to kill snails
Notification: none
Measures required under the Animal Disease Surveillance Plan: none
Measures required for introducing animals to infected animal N/A (requires vector for transmission)
Conditions for restoring disease-free status after an outbreak: N/A (not in USA)
<p>Experts who may be consulted: Dr. Patrick Skelly Molecular Helminthology Lab Cummings School of Veterinary Medicine Tufts University http://vet.tufts.edu/mhl/ Phone: 508-887-4348 Fax: 508-839-7911 Email: Patrick.Skelly@tufts.edu</p>
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SCHISTOSOMIASIS

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NEW WORLD SCREWWORM (*Cochliomyia hominivorax*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All warm-blooded animals but most cases occur in cattle, goats and sheep. In Oct. 2016 cases were confirmed in Key deer (<i>O. virginianus clavium</i>) in Florida, USA.	Gravid female flies deposit eggs either in wounds or directly onto intact mucous membranes.	Discomfort, decreased appetite, wounds with malodorous, reddish/brown fluid with larvae; slight movement inside a closed wound. Upon closer observation of wound a mass of clear-colored eggs can be observed.	Untreated animals could die. Mortality rates in Texas when disease was endemic in the USA was 20 –80% in fawns. However, no cases of myiasis in newborn Key deer fawns were observed in 2016. Mortality in the 2016 outbreak varied between 7-98 animals/month)	Treatment of wounds with organo-phosphates (spray, foam, dip, dust) (e.g. coumaphos, ronnel), or lindane. Carbamate and pyrethroid compounds are also effective vs larvae, immature forms and flies.	Monitoring wounds and treating infested wounds with insecticides Doramectin injection.	Yes

Fact Sheet compiled by: Carlos R. Sanchez

Sheet completed on: updated December 2017

Fact Sheet Reviewed by: Heather Robertson

Susceptible animal groups: Mammals with most cases occurring in cattle, goats, sheep and wildlife; however, dogs, and cats may be affected. Birds are rarely affected.

Causative organism: *Cochliomyia hominivorax*

Zoonotic potential Yes, with the young, elderly or infirm higher risk of infection.

Distribution: Current distribution includes: Caribbean islands (eradicated in Curacao, Virgin Islands and Puerto Rico) and northern countries of South America to Uruguay, northern Chile and northern Argentina. Panama was recognized free of NWS in 2006 and a permanent barrier zone was established in the Darien province of Eastern Panama. New World screwworm had been eradicated from the United States more than three decades ago, On October 3, 2016 USDA declared the confirmation of New World screwworm (*Cochliomyia hominivorax*) in Key deer from the National Key Deer Refuge in Big Pine Key, Florida. In March 2017, the USDA’s APHIS announced the successful eradication of the New World Screwworm from Florida

Incubation period: After 12-24 hrs, eggs are deposited in wounds or mucous membranes have larvae emerge which burrow into the wound. After 7 days, the larvae exit from the wound and fall to the ground. Pupal period ranges from 7d-2mo (depending on temperatures). Complete cycle takes between 3 weeks and 3 months.

Clinical signs: Animals with screwworm infestation often display discomfort and appear unthrifty and depressed. Other non-specific clinical signs include: separation from group, anorexia, and reduced milk production in dairy cattle. Typically, an open wound is present with malodorous reddish/brown fluid that has either eggs or larvae. Egg masses are found around the wound as “shingle-like” raft of whitish or cream-colored egg. The larvae can be visible or deep inside the wound; closed wounds may have slight movement inside. Larvae can also be observed on intact mucous membranes of body orifices (nose, anus, vaginal area). The wound can enlarge due to multiple infestations and if not treated animal could die within 2 weeks.

Post mortem, gross, or histologic findings: Screwworms do not feed on dead tissue or carrion so larvae are unlikely to be found on post-mortem examination unless the animal died recently. Larvae of different ages are normally found on wounds or natural opening mucous membranes. Other fly larvae may be present in lesion making gross diagnostic difficult. Microscopic lesions are not useful for the definitive diagnosis of screwworm.

Diagnosis: Screwworm is a reportable disease in US. Before collecting or sending any samples from animals with suspected screwworm, federal and state authorities should be contacted. Identification of the eggs and flies

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are best to left to an entomologist. However, specifically for larvae, they should be removed from the deepest part of the wound and examined grossly by dissecting microscope. Larvae grow from 2mm to fully grown larvae that can reach 1.5cm in length. Larvae are identified by their “wood screw” shape. Screwworm larvae have whitish bodies, and can be differentiated from other larvae by the darkly pigmented tracheal tubes on the dorsal aspect of the posterior end of 3rd stage larvae.

Material required for laboratory analysis: Larvae, eggs or flies can be conserved in vials containing 80% ethanol or isopropyl alcohol; formalin should not be used. Larvae should be removed from the deepest part of the wound to reduce the possibility of collecting non-screwworm species; optimal preservation of larvae, in their natural extended state, can be made by killing them in boiling water (15–30 seconds immersion) before storage in 80% ethanol. Suspected screwworm eggs or flies may also be submitted for diagnosis; eggs may be collected using a scalpel as scraper.

Before collecting or sending any samples from animals with suspected screwworm infections, federal and state authorities should be contacted. In the US, screwworm is a reportable disease and should be reported within 24 hours. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. Screwworms can infest humans; samples should be collected and handled with all appropriate precautions.

Relevant diagnostic laboratories:

USDA-APHIS-VS-NVSL

1920 Dayton Ave. (for parasite specimen submission) – Use VS Form 5-38 available on the APHIS website:
(http://www.aphis.usda.gov/library/forms/pdf/VS_Form5_38.pdf)

P.O. Box 844 (for letters)

Ames, IA 50010

(515) 337-7266

Fax: (515) 337-7397

For detailed information concerning the handling and shipping of diagnostic specimens as well as overall guidance on FAD investigations please see APHIS Veterinary Services (VS) Guidance Document 12001 (previously VS Memorandum 580.4) and the FAD Investigation Manual (Manual 4-0), available at <http://www.aphis.usda.gov/fadprep>.

Treatment: Before any treatment is implemented federal and local authorities must be notified. Organophosphate insecticides (coumaphos, ronnel) and lindane are effective against newly hatched larvae, immature forms and adult flies. Carbamates and pyrethroids may also be used as are effective against larvae and adult flies. In a recent study, nitenpyram showed 100% efficacy on the treatment of myiasis by *C. hominivorax* in naturally infested dogs. Screwworms in wounds are killed by direct application of aerosol, dust or foam that contain any of these products. Removal of necrotic tissue may be necessary and antibiotics may be given when secondary bacterial contamination is present.

Prevention and control: In areas where NWS is found, measures should be implemented to prevent wounds and avoid myiasis. For example, eliminate wounding procedures, handle livestock with care, and inspect pens for sharp objects.

In 1966, US was declared officially free of indigenous screwworms therefore any presumptive case must be reported. The OIE International Animal Health Code stipulates that is necessary to follow strict observation of the requirements for international trade.

When importing domestic and wild mammals from countries considered infested with New World or Old World screwworm, veterinary administrations should require the presentation of an international veterinary certificate attesting that:

- 1) Immediately prior to loading, the animals have been inspected on the premises by an official veterinarian and that any infested animal has been rejected for export;
- 2) Immediately prior to entering the quarantine pens in the exporting country:

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- a) each animal has been thoroughly examined for infested wounds by an official veterinarian and that no infestation has been found in any animal; and
 - b) any wounds have been treated prophylactically with an officially approved only larvicide at the recommended dose; and
 - c) all animals have been dipped, sprayed, or otherwise treated, immediately after inspection, with a product officially approved by the importing and exporting countries for the control of New World or Old World screwworm, under the supervision of an official veterinarian and in conformity with the manufacturer's recommendations;
- 3) at the end of the quarantine and immediately prior to shipment for export:
- a) all animals have been re-examined for the presence of infestation and all animals have been found free of infestation;
 - b) all wounds have been prophylactically treated with an approved only larvicide under the supervision of an Official Veterinarian;
 - c) all animals have been prophylactically treated again by dipping or spraying as in point 2) above.

The floor of the quarantine area and transport vehicles must be thoroughly sprayed with an officially approved larvicide before and after each use. The transit route must be the most direct, with no stopover without prior permission of the importing country. On arrival at the importation point, all animals must be thoroughly inspected for wounds and possible new world or old world screwworm infestation under the supervision of an Official Veterinarian. The bedding material of the vehicle and the quarantine area should immediately be gathered and burned following each consignment.

In addition: any imported animals from areas where screwworms are endemic must be thoroughly inspected for wound and infestations before they are allowed to enter premises. Wounds that do not appear to be infested are treated with an insecticide as preventative measure. Any infestations that become apparent after an animal enters the country must be treated promptly.

APHIS began releasing sterile flies in October 2016, as part of aggressive eradication effort undertaken in collaboration with the U.S. Fish and Wildlife Service, Florida Department of Agriculture and Consumer Services, and local partners.

Suggested disinfectant for housing facilities: Facilities where screwworm was diagnosed and vehicles that may contain adults or immature screwworms should be sprayed with insecticides; any bedding material used in the area where animal was quarantined should immediately be gathered and burned.

Notification: Any presumptive screwworm infestation must be reported to both state and federal (Area Veterinarian In Charge -AVIC) authorities. Residents who have warm-blooded animals (pets, livestock, etc.) should watch their animals carefully. Florida residents should report any potential cases to 1-800-HELP-FLA (1-800-435-7352) or non-Florida residents should call (850) 410-3800.

Measures required under the Animal Disease Surveillance Plan: Because New World screwworm has been recently eradicated from the US, the National Animal Health Surveillance System (NAHSS) does not have a program for active surveillance at this time. However, APHIS and Florida Department of Agriculture and Consumer Services (FDACS) will continue passive surveillance to ensure any new findings are quickly identified. This surveillance includes veterinarians reporting any suspicious cases, wildlife surveillance, concerned citizens that see suspicious wounds on animals or even on a person, and continued communication with the parks and the National Key Deer Refuge. Because this is a reportable disease, state and federal (AVIC) authorities should be notified of any presumptive screwworm infestation.

Measures required for introducing animals to infected animal: In non-endemic regions, any infected animal is quarantined until treatment is complete and the wounds have healed. Treatment of the environment, as explained above, may also be necessary.

Conditions for restoring disease-free status after an outbreak: Areas must be sprayed with approved larvicide; the disease has been eradicated in the US by the Sterile Male Release Technique (SMRT) program and therefore if there is indication of infection in the U.S.A the USDA-APHIS must be involved on any discussion about free-status of a premise.

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Experts who may be consulted:

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National Preparedness and Incident Coordination

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OLD WORLD SCREWORM (*Chrysomya bezziana*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All warm-blooded animals, including birds.	Flies are attracted to open superficial wounds as small as a tick bite. Occasionally, Old World screwworms also lay their eggs on unbroken soft skin, particularly if it has blood or mucous on its surface. Gravid female flies deposit eggs either into wounds or directly onto intact mucous membranes.	Severe myiasis in open wounds; associated discomfort and decreased appetite.	Severe infestations that remain untreated may result in the death of the host in a short time (7-14 days).	Removal and killing of the larvae in lesion. Treatment of the wound with approved insecticide. Treatment is normally repeated until the wound has healed. Removal of necrotic tissue is necessary. Ivermectin 200-300 mcg/kg.	Monitoring wounds and treating infested wounds with insecticides.	Yes

Fact Sheet compiled by: Carlos R. Sanchez

Sheet completed on: last update December 2017

Fact Sheet Reviewed by: Sarrah Kaye

Susceptible animal groups: All mammals (domestic and many species of wildlife) are affected potentially; problem is rare in birds.

Causative organism: *Chrysomya bezziana*

Zoonotic potential: Yes, humans can be hosts for screwworm larvae but it is primarily a veterinary pest.

Distribution: The distribution of Old World screwworm is confined to the Old World. *Chrysomya bezziana* is widely distributed throughout tropical areas. It is most prevalent in Southeast Asia, and throughout much of Africa (from Ethiopia and sub-Saharan countries to northern South Africa), some countries in the Middle East (reports confirmed from Iran, Iraq and recently Yemen), India, the Malay Peninsula, the Indonesian and Philippine Islands, and Papua New Guinea. *C. bezziana* has never become established in Europe, Australia, New Zealand or the Western Hemisphere. Because of its distribution, the most likely potential port of entry into the US is Hawaii.

Incubation period: Eggs hatch within 8-24 hrs after being laid. Once the larvae emerge, they immediately begin to feed on the wound fluids and underlying tissues, burrowing as a group, head-downwards into the wound. The entire larval stage lasts 5-8 days, followed by larvae leaving the wound and pupating in the soil. Maturation of pupae to adult is temperature dependent and ranges from 7 days at 28°C to 60 days at temperatures of 10–15°C. Female flies mate usually only once, but can lay more than one batch of eggs at intervals of a few days.

Clinical signs: Animals with screwworm infestations often display discomfort and appear unthrifty and depressed; other non-specific clinical signs include: separation from group and anorexia. Screwworms can infest a wide variety of wounds, from tick bites to cuts and dehorning or branding wounds. Infestations are very common in the navels of newborns (fawns with screwworms in their navels may stand in water up to their abdomen), and the perivulvar and perineal regions of their dams. If a screwworm deposits its eggs on mucous membranes, the larvae may enter any orifice including the nostrils, sinuses, mouth, orbits of the eye, ears or genitalia. Infested wounds often have a serosanguineous discharge and sometimes a distinctive odor. By the third day, the larvae may be easily found; secondary bacterial contamination is also common. The wound can enlarge due to multiple infestations and if not treated animal could die within 2 weeks.

Post mortem, gross, or histologic findings: The larvae of *C. bezziana* are obligatory wound parasites that never develop in carcasses or decomposing organic material. Larvae are unlikely to be found on post-mortem

OLD WORLD SCREWORM (*Chrysomya bezziana*)

examination unless the animal died recently. Larvae of different ages are normally found on wounds or natural openings and mucous membranes in live animals. Other fly larvae may be present in lesions making gross diagnosis difficult. Microscopic lesions are not useful for the diagnosis of screwworm.

Diagnosis: Diagnosis is by identification of the parasite under the microscope; however, before collecting or sending any samples from animals with suspected screwworm infections, federal and state authorities should be contacted. Clinical presentation of screwworm is always associated with a variety of pre-established wounds and should be considered in the event of any myiasis. Definitive diagnosis can be made after observation, extraction and identification of typical larvae along with history of travel to an area endemic for *C. bezziana*. Larvae must be removed from the deeper areas as well as superficial regions to be sure all species present are examined. Larva should be placed in 70% alcohol and not in formalin for future identification. Fully mature larvae develop a reddish-pink tinge over the creamy white color of younger larvae. Screwworm species have prominent rings of spines around the body and these spines appear large and conspicuous under a microscope when compared with most non-screwworm species. If a wound is considered to be infested with Old World screwworms samples should be collected and sent to eradication officials. Adult screwworms are uncommonly seen. They are also difficult to distinguish from other flies. Other techniques used mostly in research laboratories include cuticular hydrocarbon analysis, analysis of mitochondrial DNA, and random amplified polymorphic DNA polymerase chain reaction (RAPD-PCR) assays.

Material required for laboratory analysis: Before collecting or sending any samples from animals with suspected screwworm infections, federal and state authorities should be contacted. Screwworms can infest humans; samples should be collected and handled with all appropriate precautions. Larvae, eggs or flies can be conserved in vials containing 70-80% ethanol or isopropyl alcohol; formalin should not be used. Different larval stages should be collected; larvae should be removed from the deepest part of the wound to reduce the possibility of collecting non-screwworm species. Optimal preservation of larvae, in their natural extended state, can be made by killing them in boiling water (15–30 seconds immersion) before storage in 80% ethanol. Suspect screwworm eggs or flies may also be submitted for diagnosis; eggs are best collected using a scalpel as a scraper.

Relevant diagnostic laboratories:

USDA-APHIS-VS-NVSL

1920 Dayton Ave. (for packages)

P.O. Box 844 (for letters)

Ames, IA 50010

(515) 337-7266

Fax: (515) 337-7397

http://www.aphis.usda.gov/services/report_pest_disease/report_pest_disease.shtml

USDA-APHIS-VS-NVSL-FADDL

40550 Route 25 (for packages)

Orient Point, NY 11957

P.O. Box 848 (for letters)

Greenport, NY 11944-0848

(631) 323-3256

Fax: (631) 323-3366

Treatment: Removal and killing of the larvae present in any wound or lesion. Immediate treatment of all detected wounds with an approved insecticide (organophosphate insecticides, carbamates and pyrethroids) should be followed by a precautionary spraying or dipping of the animals before transport. For residual protection against re-infestation, insecticides must be applied at 2–3-day intervals until the wound has healed; animals with screwworm-suspect wounds should be quarantined until treated and wounds have clearly healed.

OLD WORLD SCREWORM (*Chrysomya bezziana*)

A single subcutaneous injection of ivermectin (200 mcg/kg) has been effective against OWS in preventing navel strike of newborn calves and scrotal strike of castrated calves and also prevented re-strike of treated wounds of adult cattle.

Prevention and control: The OIE International Animal Health Code stipulates that it is necessary to follow strict observation of the requirements for international trade:

When importing domestic and wild mammals from countries considered infested with New World or Old World screwworm, veterinary administrations should require presentation of an international veterinary certificate attesting that:

- 1) Immediately prior to loading, the animals have been inspected on the premises by an official veterinarian and that any infested animal has been rejected for export;
- 2) Immediately prior to entering the quarantine pens in the exporting country:
 - a) each animal has been thoroughly examined for infested wounds by an official veterinarian and that no infestation has been found in any animal; and
 - b) any wounds have been treated prophylactically with an officially approved larvicide at the recommended dose; and
 - c) all animals have been dipped, sprayed, or otherwise treated, immediately after inspection, with a product officially approved by the importing and exporting countries for the control of New World or Old World screwworm, under the supervision of an official veterinarian and in conformity with the manufacturer's recommendations;
- 3) At the end of the quarantine and immediately prior to shipment for export:
 - a) all animals have been re-examined for the presence of infestation and all animals have been found free of infestation;
 - b) all wounds have been prophylactically treated with an approved larvicide under the supervision of an official veterinarian;
 - c) all animals have been prophylactically treated again by dipping or spraying as in point 2) above.

The floor of the quarantine area and transport vehicles must be thoroughly sprayed with an officially approved larvicide before and after each use. The transit route must be the most direct, with no stopover without prior permission of the importing country. On arrival at the importation point, all animals must be thoroughly inspected for wounds and possible New World or Old World screwworm infestation under the supervision of an official veterinarian. The bedding material of the vehicle and the quarantine area should immediately be gathered and burned following each consignment.

In addition: any imported animals from areas where screwworms are endemic must be thoroughly inspected for wounds and infestations before they are allowed to enter premises. Wounds that do not appear to be infested are treated with an insecticide as preventative measure. Any infestations that become apparent after an animal enters the country must be treated promptly.

Suggested disinfectant for housing facilities: Facilities where screwworm was diagnosed and vehicles that may contain adults or immature screwworms should be sprayed with insecticides; any bedding material used in the area where the animal was quarantined should immediately be gathered and burned.

Notification: Any presumptive screwworm infestation must be reported to both state and federal (Area Veterinarian In Charge - AVIC) authorities.

Measures required under the Animal Disease Surveillance Plan: Because Old World screwworm has never been reported in the US, the National Animal Health Surveillance System (NAHSS) does not have a program for active surveillance. However, as this is a reportable disease, state and federal (AVIC) authorities should be notified of any presumptive screwworm infestation.

Measures required for introducing animals to infested animal: In non-endemic regions, any infected animal is quarantined until treatment is complete and the wounds have healed. Treatment of the environment, as explained above, may also be necessary.

OLD WORLD SCREWORM (*Chrysomya bezziana*)

Conditions for restoring disease-free status after an outbreak: Old World screwworm has never been reported in the US and therefore if there is any indication of any screwworm infection in the US, the USDA-APHIS must be notified immediately.

Experts who may be consulted:

Steven R Skoda
Research entomologist
USDA/Agricultural Research Service
2700 Fredericksburg Road
Kerrville, TX, 78028
(830) 792-0334
Fax: (830) 792-0314
steve.skoda@ars.usda.gov

Project Manager, Old World screwworm fly

Animal Health Australia

(02) 6203 3912

aha@animalhealthaustralia.com.au or call or call the Emergency Animal Disease Watch Hotline 1800 675 888.

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SEALPOX VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Many pinniped species, especially seals and sea lions.	Direct contact (i.e., rubbing, bites, scratches, saliva, bodily fluids), and fomites (i.e., rubber gavage tubing, feeding apparatus, gloves, needle puncture)	Most often seen in juveniles, animals in distress or newly housed. Firm skin nodules (1-3cm) will appear on head, neck, and thorax and can spread to abdomen, flippers, and mucosa. Infected area can become inflamed or necrotic	Often mild severity with low mortality. Those individuals with immune-suppressive conditions are at risk for a more severe infection	Lesions usually heal within a few weeks without treatment and leave a slightly raised gray scar without fur	Restrict movement of animals between enclosures; replace or disinfect gloves when handling animals; drain and scrub pens with 10% bleach solution regularly; wear proper personal protective equipment	Yes

Fact Sheet compiled by: Nadia F Gallardo-Romero, Benjamin P Monroe

Sheet updated on: 20 August 2013

Fact Sheet Reviewed by: William Van Bonn; Ginny Emerson

Susceptible animal groups: Harbor seals, grey seals, Northern fur seals, Northern elephant seals, California sea lions, Steller's sea lions and South American sea lions.

Causative organism: Sealpox virus, a member of the *Parapoxvirus* genus

Zoonotic potential: Yes

Distribution: The geographic range of sealpox virus is considered worldwide, and infection has been confirmed in free-ranging pinnipeds in the Atlantic and Pacific Oceans (including America, Europe, and Siberia), and Antarctica. Sealpox infection has been identified in captive pinnipeds and humans at marine rehabilitation centers in North America and Europe.

Incubation period: Clinical signs can appear within 1-5 weeks post exposure in captive animals. Human clinical signs have reportedly developed one week after exposure.

Clinical signs:

Animals: Sealpox infection is highly contagious in confined spaces with low mortality rates but very high morbidity. Juveniles, distressed, and newly-housed animals are the most likely to have active disease. The skin will present 1-3 cm firm skin nodules or lumps on head, neck and thorax, and may spread to abdomen, flippers, and mucosa. Lesions can present as solitary, in clusters, or generalized and progress from inflamed skin to necrotic.

Humans: Persons who handle sick animals may come into contact with the virus and may get infected if they have small open cuts or breaks in the skin. Rare cases develop painful, swollen sores that may evolve into a bullous lesion. Infection may be more severe in persons with skin or immune-deficient medical conditions.

SEALPOX VIRUS

Post mortem, gross, or histologic findings: Firm cutaneous nodules 1-3 cm diameter are the characteristic lesions of the disease. They can be congested and focally ulcerated, solitary, in clusters, or generalized along the animal body. Histologically, the lesions are characterized by epithelial hyperplasia and acanthosis. The dermis may present intense inflammatory infiltrate and necrosis, the epidermis may demonstrate edema, vacuolization and ballooning degeneration of keratinocytes. Eosinophilic cytoplasmic inclusions are also typical findings.

Diagnosis: Classic clinical presentation is used predominantly, especially in rehabilitation settings where it is observed seasonally. Molecular assays for viral DNA detection are most commonly used including PCR, RFLPs, and sequencing. Observation of typical cytoplasmic effect (CPE) in cell culture, histology, viral isolation, and virion visualization by electron microscopy also are used as confirmation of findings. Differential diagnosis with “seal finger” (caused by a *Mycoplasma*), anthrax, and fungal infections should be performed.

Material required for laboratory analysis: Swabs of swelling, mucosal or other lesions are the preferred sample. Place swab in a dry, sterile micro tube, store at -20°C. Skin biopsies containing a margin of normal tissue around the affected area. Place the half of the sample in 10% formalin, and the other half in a dry, sterile micro tube, store at -20°C. CDC laboratories can provide specimen collection guidance. Contact the reference laboratory prior to shipping to inquire about necessary permits.

Relevant diagnostic laboratories:

Centers for Disease Control and Prevention
Poxvirus and Rabies Branch
CDC Poxvirus Inquiry line: 404-639-4129
1600 Clifton Rd NE, Atlanta GA 30333
Nzr6@cdc.gov

University of Florida
College of Veterinary Medicine,
Marine Mammal Health Program
Fax: (352) 392-5464
PO Box 100126, Gainesville, FL 32610
NollensH@mail.vetmed.ufl.edu

Treatment: Lesions usually resolve within a few weeks without treatment and may leave a scar. Palliative treatment is recommended for human infection to control secondary infections, inflammation, and pain. However, the literature has previously reported *in vitro* susceptibility of sealpox virus to cidofovir.

Prevention and control: Quarantine newly admitted animals, restrict movement of animals between enclosures, and decrease the number of animals per pen. Replace or disinfect gloves and equipment when handling sick animals and between enclosures; drain and scrub pens with 10% bleach solution or other disinfectant regularly. Wear proper personal protective equipment (PPE) including rubber or latex gloves, rain pants, overalls or suits, goggles and/or masks. Frequent hand washing is encouraged after handling animals, enclosures, or equipment.

Suggested disinfectant for housing facilities: 10% bleach solution, chlorhexidine gluconate based solutions, and other anti-viral solutions.

Notifications: Sealpox virus infection is not a reportable disease. However, state or local health departments should be notified of suspected human infections.

Measures required under the Animal Disease Surveillance Plan: The disease is not currently listed under the USDA National Animal Health Surveillance and/or Reporting systems.

Measures required for introducing animals to an infected animal(s): Sealpox is highly contagious among pinnipeds and will spread easily between animals in direct contact. Introduction of healthy animals

SEALPOX VIRUS

to sick animals is not recommended until skin lesions have completely healed.

Conditions for restoring disease-free status after an outbreak: No specific standards exist at this time. However, it is recommended to test the animals for viral DNA presence once the lesions are completely healed. If all animals from the center are negative, disease-free status can be restored and recommendation of quarantine and testing of new individuals should be applied.

Experts who may be consulted:

Mary Reynolds, PhD, MPH
Poxvirus and Rabies Branch
CDC Poxvirus Inquiry line: 404-639-4129
1600 Clifton Rd NE, Atlanta GA 30333
Nzr6@cdc.gov

References:

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SHEEP AND GOAT POX

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Sheep and goats	Airborne; direct contact with infected animals and fomites; biting insects are possible source	Inappetence; fever; skin lesions of maculae, papules and scabs; dyspnea; nasal discharge; conjunctivitis	Mild to severe depending on age, breed and immunity	None although can administer antibiotics to prevent secondary bacterial infections	Quarantine incoming and cull infected animals; isolate recovering animals; properly clean infected area and utensils. Outside US, prophylactic vaccination is used commonly.	No

Fact Sheet compiled by: Kevin Leiske; revised by Alfonso Torres

Sheet completed on: 20 January 2011; updated 2 August 2013

Fact Sheet Reviewed by: James Rasmussen; Charles Lamien

Susceptible animal groups: All breeds of domestic and wild sheep and goats

Causative organism Sheep pox and Goat pox viruses, Family Poxviridae, Genus Capripoxvirus. While it is recognized that sheep pox virus and goat pox virus are different, but related viruses, both agents have the ability to infect either sheep or goats causing a disease that is clinically and pathologically identical.

Zoonotic potential: None

Distribution: Africa (north of Equator), Middle East, Turkey, Greece, Central Asia, South East Asia, several countries in East Asia including parts of China, Russia, and Mongolia. This disease is one of the most actively spreading diseases affecting small ruminants during the last 5-10 years.

Incubation period: 4-21 days but usually 1-2 weeks.

Clinical signs: Fever usually precedes the skin lesions that start as erythematous macules and progress to hard papules. The center of the papules become depressed and turns a whitish grey color. The area then becomes necrotic and is surrounded by an area of hyperemia. Necrotic skin lesions culminate in scabs that leave a scar after scab loss. Lesions are usually easier to find in areas of the body that have sparse hair (i.e., axillary and inguinal areas and under the tail). Mucous membranes can develop similar lesions that may become necrotic. Dyspnea, nasal discharge and conjunctivitis also can occur. In endemic areas, the disease can be mild or the infection inapparent.

Post mortem, gross, or histologic findings: Typical “pox” skin lesions, ulceration of the mucous membranes, firm nodules in the lungs (grey or white), papules and ulcerations can also be seen in the abomasal mucosa as well as the rumen, large intestines, pharynx, trachea and esophagus. Lymph nodes are enlarged and edematous and the liver and kidney may have pale, discrete subcapsular foci on the surface.

Diagnosis: Virus detection by electron microscopy on dry skin scabs. Nucleic acid detection by PCR in tissue samples, or virus isolation on cell culture. AGIDs or ELISAs can detect viral antigens. Serology (AGID, IFA, ELISA, VN, Western blotting) is available, but is not that reliable given that capripoxvirus immunity is mostly cell-associated. Nasal swab can be sampled for molecular diagnostics.

SHEEP AND GOAT POX

Material required for laboratory analysis: In live animals biopsies of skin lesions, scraping of skin lesions as well as lymph node aspirates and blood. Nasal swabs can be utilized for PCR. At necropsy, samples from skin lesions, lymph nodes and lung lesions should be collected. Lesions in other organs can also be submitted based on postmortem findings.

Relevant diagnostic laboratories:

Foreign Animal Disease Diagnostic Laboratory

USDA-APHIS-VS-NVSL-FADDL

40550 Route 25 (for packages)

Orient Point, NY 11957

P.O. Box 848 (for letters)

Greenport, NY 11944-0848

Director: Dr. Fernando Torres-Velez

Phone: (631) 323-3256

Fax: (631) 323-3366

Email: Fernando.J.Torres-Velez@aphis.usda.gov

Treatment: None although antibiotics could be used to prevent secondary bacterial infections.

Prevention and control: Quarantine incoming and cull infected animals. Recovering animals should be isolated for 45 days after clinical signs are no longer present. Infected areas and utensils should be cleaned properly. Viable virus may be found in shaded areas of the environment for up to 6 months after an outbreak. Vaccination after 6 months of age has helped decrease morbidity and control spread in other countries. MLV products tend to provide best protection, but they are not 100% protective. These MLV products are not allowed for use in the US.

Suggested disinfectant for housing facilities: Approved disinfectants for sheep/goat pox include 4% sodium carbonate solutions; 2% sodium hydroxide solution; or up to 12.5% sodium hypochlorite.

Notification: Any suspected case should be notified to State and federal authorities within 24 hours for proper investigation and diagnosis by trained State or Federal Foreign Animal Disease Diagnosticians.

Measures required under the Animal Disease Surveillance Plan: Early stages of sheep/goat pox can be similar to some cases of Contagious Ectyema (orf).

Measures required for introducing animals to infected animal: Animals that are infected and survive have very good immunity. However, they should be isolated for 45 days after clinical signs are no longer present.

Conditions for restoring disease-free status after an outbreak: Culling infected herd may be required. Isolation for 45 days after no more clinical signs seen and properly disinfected. However, since this disease has never been reported in the Western Hemisphere, disease-free status after a confirmed outbreak will require a comprehensive surveillance program conducted by state and federal authorities.

Experts who may be consulted:

Foreign Animal Disease Diagnostic Laboratory

USDA-APHIS-VS-NVSL-FADDL

40550 Route 25 (for packages)

Orient Point, NY 11957

P.O. Box 848 (for letters)

Greenport, NY 11944-0848

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Email: Fernando.J.Torres-Velez@aphis.usda.gov

SHEEP AND GOAT POX

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http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.07.14_S_POX_G_POX.pdf
Accessed 10 September 2013.
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http://www.cfsph.iastate.edu/Factsheets/pdfs/sheep_and_goat_pox.pdf. Accessed 10 September 2013.

SHIGELLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans and non-human primates; ruminants; occasionally dogs.	Fecal-oral; via direct contact with infected animals; or indirectly via food, water, flies or inanimate objects contaminated and contact with shedding animals Food borne disease; sexual contact	Diarrhea or dysentery with potentially blood and/or mucus; abdominal cramps; tenesmus; and pyrexia. Asymptomatic carriers are possible.	Generally self-limiting disease. Complication due to bacteremia is possible, mainly in immuno-compromised individuals, that result in arthritis, neuritis, vulvovaginitis, chronic colitis, conjunctivitis; eventually death.	Oral rehydration and antibiotics.	Proper sanitation; reduction of stress; and isolation of potential carriers; fly control.	High zoonotic potential.

Fact Sheet compiled by: Cornelia J. Ketz-Riley

Sheet completed on: updated 8 February, 2018

Fact Sheet Reviewed by: David Miller

Susceptible animal groups: Primates (humans and non-human) are natural hosts. Reports of infection in cattle and dogs have been made.

Causative organism: Family: Enterobacteriaceae; genus: *Shigella*; four species: *Shigella dysenteriae* – serogroup A; *Shigella flexneri* – serogroup B; *Shigella boydii* – serogroup C; *Shigella sonnei* – serogroup D. Infection and transmission occurs mainly via fecal-oral route through contaminated food, water or direct contact; in humans, person-to-person transmission is the most common route. Arthropods, such as houseflies can function as mechanical vectors. Serovars are of antigenetic difference; serotyping and subtyping via pulsed-field gel electrophoresis is important in epidemiologic investigations. *Shigella* bacteria are able to invade intestinal mucosa cells, but this varies by strain; cytotoxins (Shiga -toxin) may also be produced. While *Shigella dysenteriae* is mostly responsible to larger outbreaks in humans, mainly children, there seems to be an epidemiological shift towards other serogroups, mainly *Shigella sonnei*. This will have additional consequences for treatment and vaccine production.

Zoonotic potential: High.

Distribution: Worldwide. Originally, a common problem encountered mostly in under-developed regions, but with higher tourist travel activity and movements of refugees in more recent times, infections more often seen in other parts of the worlds.

Incubation period: 1-6 days.

Clinical signs: Pyrexia, headache, abdominal cramps, and severe painful diarrhea that is watery, and potentially with mucus, pus or blood. The presentation is usually self-limiting within 10 days. However, in its more severe form, other signs can present such as dehydration and neurological signs. Bacteremia has potential complications of arthritis, neuritis, vulvovaginitis, chronic colitis, conjunctivitis, iritis, hemolytic uremic syndrome, or death. *Shigella* infection is affecting T-lymphocyte activity and therefore alters immune response. It also stimulates protective local IgA secretion supporting the integrity of intestinal epithelial cells. Gingivitis has been reported in macaques.

Post mortem, gross, or histologic findings: Most common findings during gross necropsy; signs of dehydration, gastroenteritis, enteritis, hepatomegaly, splenomegaly, military white foci in the liver, and mesenteric lymphadenopathy. After development of septicemia, submucosal and subserosal petechial

SHIGELLOSIS

hemorrhages in multiple organs, muscular necrosis - typically involving myocardial and gizzard muscle, nephropathy, polyserositis, synovitis are commonly found. Histopathologic findings include multifocal necrotic hepatitis, necrosis of cryptic or surface enterocytes in lower small intestines, sometimes in cecum and colon, depending on bacterial species involved.

Diagnosis: Culture of fresh fecal material or use of a transport medium, due to limited viability, is still the most commonly used diagnostic tool. Selective media are used for identification of *Shigella* sp. Such media are: MacConkey, *Salmonella-Shigella* Agar (S-S), Xylose-Lysin-Desoxycholate (XLD), Lysine iron agar. In cases of small samples and bacterial overgrowth, transfer of cultured sample to enrichment media, such as Gram-negative broth, is recommended. Serological and immunohistochemical methods can be used to identify *Shigella* species and serotypes involved in disease process. These methods are essential when a *Shigella* infection is suspected, and when isolation of live organisms by culturing is not possible. ELISA and similar modified assays for antibody reactions against *Shigella* types in individuals. Serological examinations valid for identification of acute or subacute infected individuals, but chronic carriers are often seronegative. A variety of PCR assays is researched and used to recover *Shigella* DNA in live material or dead surfaces. PCR is also used for further classification of *Shigella* serovars. A multiplex PCR assay was recently optimized for simultaneous detection and differentiation of three pathogenic *Shigella* species by using amplified target genes of the bacteria. Also, a flow cytometry method and other molecular methods have been investigated as rapid methods for detection of *Shigella* bacteria.

Material required for laboratory analysis: For culture, feces, organ tissue, and whole blood are recommended. For ELISA and other serologic assays, feces, organ tissue, serum, food, milk, and water may be used. Tissue, feces, whole blood, soil, or processed food can be used for PCR testing.

Relevant diagnostic laboratories: Any laboratory that is set up for culture methods can be used for first screening for *Shigella*.

Treatment: Mild infections are self-limiting and are only treated with supportive care, such as rehydration, electrolyte and analgesic treatment. Antibiotics should be used only in cases of severe acute and life-threatening infection, when a subsequent bacteremia is anticipated, mainly in immunocompromised and young individuals. The choice of antibiotics should be based on an antibiogram of the culture; however, recommended antibiotics are quinolones (nalidixic acid, norfloxacin, enrofloxacin, ciprofloxacin, danofloxacin), beta-lactams (ampicillin, amoxicillin, 1st 2nd cephalosporin), macrolids (azithromycin, erythromycin, clindamycin), aminoglycosides (gentamicin, streptomycin), others (tetracyclines, sulfonamides, cotrimoxazole and furazolidon. Antibiotic and chemotherapeutic use can reduce severity of the disease, as well as the period of convalescent carriage of *Shigella* organisms. Multi-drug resistance against the commonly recommended antibiotics and chemotherapeutics, including the more recently advocated drugs like azithromycin, ciprofloxacin, marbofloxacin, is developing rapidly worldwide. Therefore, newer drugs and alternative treatment methods are constantly researched for their efficacy and safety in the treatment of shigellosis.

Prevention and control: Asymptomatic carriers make eradication and control of shigellosis difficult. Preventative control programs should include a good sanitation protocol and animal collection management. Feeding additives to introduce competitive bacteria through food or to influence the local pH values and mucosal integrity, such as probiotics, plant extracts and essential oils with antimicrobial activity seems to be beneficial in controlling *Shigella* infections. Bacteria and yeast found in kefir yogurt have shown to effectively inhibit the invasion of intestinal tissue by *Shigella* bacteria and the subsequent local inflammation. The existence of multiple *Shigella* serotypes and their growing resistance to antibiotics stress the urgent need for the development of a low-cost vaccine that is protective across all serotypes. No vaccine is yet officially available, but human and animal challenge-rechallenge trials with virulent *Shigella* as well as observational studies in *Shigella*-endemic areas have shown that the incidence of disease decreases following *Shigella* infection, pointing to biological feasibility of a vaccine. A variety of *Shigella* vaccine constructs are under development, including live attenuated, formalin-killed whole-cell, glycoconjugate, subunit, and novel antigen vaccines (e.g., Type III secretion system and outer membrane proteins). All persons involved in

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animal care, dealing with and processing and preparing food and feed need to be properly educated in sanitation and potential risks of contamination of the animal collection or the food chain with *Shigella*. High sanitation standards and low-stress impact to the animals are key elements in the control of *Shigella* infections.

Suggested disinfectant for housing facilities: Most commonly used disinfectants, such as diluted hydrochlorite, quaternary ammonium-based products are effective against *Shigella*.

Notification: Reportable disease; Most states require that local health departments report outbreaks to their state health department. States report voluntarily to CDC.

Measures required under the Animal Disease Surveillance Plan: Culture and serotyping of *Shigella* of any animals potentially in contact with infected animals and asymptomatic carrier in a collection with shigellosis outbreak. Any potential sources, such as introduced animals, care personnel, feed and water sources and any potentially contaminated dead surfaces need to be cultured and potentially serologically and immunohistochemically investigated.

Measures required for introducing animals to infected animal: Regular quarantine in a clean environment; reduce access to potential vectors, and host animals; separate tools and personnel for quarantined animals; fecal examination and culture as preshipment evaluation and quarantine examination before introduction.

Conditions for restoring disease-free status after an outbreak: Quarantine of whole collection. Isolation of sick and potentially infected animals. Testing of any potentially contaminated feed, water, surface and also healthy animals before giving access to previously contaminated area. Multiple cultures of potentially infected animals necessary due to inconsistent shedding of bacteria.

Experts who may be consulted:

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SIMIAN FOAMY VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Found in most non-human primates	Horizontal through contact with saliva	Not known to cause disease	Not known to cause disease	None	Life-long infection	Yes
Fact Sheet compiled by: Natalie Mylniczenko						
Sheet completed on: Jan2019						
Fact Sheet Reviewed by: Donna Ialeggio						
Susceptible animal groups: All species of non-human primates are susceptible.						
Causative organism: Simian foamy virus (SFV) in Genus <i>Spumavirus</i> .						
Zoonotic potential: Yes						
Distribution: Worldwide distribution in wild and captive non-human primate populations.						
Incubation period: Unknown						
Clinical signs: None reported, considered to be 'medically insignificant'.						
Post mortem, gross, or histologic findings: No known pathology is associated with SFV infection. Co-infection with SIV may increase SIV-related disease progression. At least in NHP co-infection with species-specific SFV (eg chimp + colobus, see ref. 14, below) is documented.						
Diagnosis: Serology (ELISA and WB for confirmation), PCR, and virus isolation can be used.						
Material required for laboratory analysis: Whole blood, serum/plasma, body fluids, and tissues.						
Relevant diagnostic laboratories:						
<p>Primate Assay Laboratory (PAL) <i>Formerly PDL</i> California National Primate Research Center University of California, Davis Phone: 530-752-8242 E-mail: cnprc-pdl@ucdavis.edu http://www.cnprc.ucdavis.edu/primate-assay-laboratory-core/</p> <p>Virus Reference Laboratories, Inc. VRL-San Antonio, USA P.O. Box 40100 7540 Louis Pasteur, Suite 200 San Antonio, Texas 78229 Office: 877-615-7275 Fax: 210-615-7771 http://www.vrlsat.com/</p> <p>Zoologix Inc. 9811 Owensmouth Avenue, Suite 4 Chatsworth, California 91311-3800 818-717-8880 Fax: 818-717-8881 Email: info@zoologix.com http://www.zoologix.com (accessed 15Jan19)</p>						
Treatment: None is reported.						
Prevention and control: Infection is ubiquitous in non-human primates.						
Suggested disinfectant for housing facilities: 70% ethanol, formalin, 10% household bleach (sodium hypochlorite), Lysol, and most lipophylic detergents.						
Notification: None at this time.						

SIMIAN FOAMY VIRUS

Measures required under the Animal Disease Surveillance Plan: None at this time.

Measures required for introducing animals to infected animal: Seroconversion of naïve animals is possible with exposure.

Conditions for restoring disease-free status after an outbreak: Life-long infection so no changes can be made to restore disease-free status.

Experts who may be consulted:

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SIMIEN HEMORRHAGIC FEVER

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Natural host: Patas monkey (<i>Erythrocebus patas</i>) and other African primates	Direct and indirect contact with infected animals, secretions or fomites.	Anorexia, lethargy, fever, diarrhea or melena with frank blood, facial edema, petechia, DIC	Fatal in macaques but no clinical disease in natural hosts	Isolation of unaffected animals No successful clinical treatment	Testing of African primates Separation of African primates and macaques in captivity	No
Aberrant host: Asian macaques	Iatrogenic transmission					

Fact Sheet compiled by: Thomas P. Meehan; updated by Dawn Zimmerman

Sheet completed on: 30 June 2011; updated 15 August 2013; updated 27 December 2017

Fact Sheet Reviewed by: Kimberlee B. Wojick; Meredith M. Clancy

Susceptible animal groups: Captive rhesus macaques (*Macaca mulatta*) were affected during an “explosive” outbreak in the index case in 1964. This and subsequent outbreaks in macaques have apparently resulted from contact with, or iatrogenic transmission from, asymptomatic captive African monkeys. African monkey species including Patas monkey (*Erythrocebus patas*), vervet monkeys (*Cercopithecus aethiops*) and baboons (*Papio* spp.) are suspected to be the natural reservoirs. Red colobus monkeys and red-tailed guenons also have been identified as natural hosts for SHFV variants. Currently, SHFV is thought only to affect Asian macaques of diverse species, including: rhesus macaque (*Macaca mulatta*), bonnet macaque (*M. radiata*), cynomolgus macaque (*M. fascicularis*), stump-tailed macaque (*M. arctoides*), Assam macaque (*M. assamensis*) and Southern pig-tailed macaque (*M. nemestrina*).

Causative organism: SHF is caused by at least three *Arteriviruses* (family *Arteriviridae*): simian hemorrhagic fever virus (SHFV), simian hemorrhagic encephalitis virus (SHEV), and Pهبjah virus (PBJV). Since 2011, nine additional distant relatives of these three viruses were discovered in apparently healthy African cercopithecoid primates, and are thought to also be potential causes of SHF.

Zoonotic potential: None; however, the virus is being researched for species jump potential due to the presence of highly divergent SHFV variants.

Distribution: Natural hosts in Africa but consideration for captive animals worldwide.

Incubation period: 2-9 days.

Clinical signs:

Natural hosts: asymptomatic

Macaques: Although these aberrant hosts can be asymptomatic, in clinical animals, fever, depression, facial edema, anorexia, adipsia, dehydration, proteinuria, cyanosis, skin petechiae, melena, epistaxis, DIC, and retrobulbar hemorrhages can present. Mortality ranges widely at 11-100% (64% in recent study) but death occurs in 10-15 days.

Post mortem, gross, or histologic findings: Petechial hemorrhages on mucosal and serosal surfaces, hemorrhage of proximal duodenum, splenomegaly, splenic lymphoid follicles ringed with zone of hemorrhage, multi-organ necrosis, vasculitis and hemorrhage, intravascular fibrin, fibrin in spleen, lymphohistiocytic meningoencephalitis.

Diagnosis: Real time RT- PCR, ELISA, DIA.

Material required for laboratory analysis: blood, serum.

Relevant diagnostic laboratories:

Zoologix
9811 Owensmouth Ave, Suite 4
Chatsworth CA 91311
818-717-8880

SIMIAN HEMORRHAGIC FEVER

BioReliance Corp.
14920 Broschart Rd.
Rockville, MD 20850-3349
301-738-1000

VRL-San Antonio, USA
P.O. Box 40100/ 7540 Louis Pasteur, Suite 200
San Antonio, Texas 78229
877-615-7275
<http://www.vrlsat.com/catalog/specimen/45>

Treatment: None.

Prevention and control: Separation of African primates and macaques in captive settings; Testing of African primate species for antibodies. Due to the indication that SHF may be caused by a number of distinct simian arteriviruses, screening procedures for SHFV in primate-holding facilities should allow for detection of all known simian arteriviruses.

Suggested disinfectant for housing facilities: Disinfectants effective against *Arteriviridae*: quaternary ammonium and glutaraldehyde mixture (Synergize™, Preserve International) 0.8%, potassium monopersulfate (Virkon-S™, DuPont Animal Health) 1.0%.

Notification: None.

Measures required under the Animal Disease Surveillance Plan: Currently none.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Depopulation of affected macaque group and premise disinfection.

Experts who may be consulted

While this disease is not zoonotic, similarities to other hemorrhagic diseases of primates should indicate consultation with:

Centers for Disease Control and Prevention (CDC) Division of Global Migration and Quarantine May be contacted 24 hours/day through the CDC emergency operations center (770-488-7100).

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SIMIEN HEMORRHAGIC FEVER

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SIMIAN IMMUNODEFICIENCY VIRUSES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Found in many African non-human primates. Macaques susceptible.	Mainly horizontal through sexual contact and bite wounds. Vertical transmission reported by virus-infected milk.	Clinical disease occurs in only a minority of infected individuals. When pathogenic, disease depends on the nature of the organ and opportunistic infections.	Severe and fatal in non-natural host	None specific although same treatment options for HIV could be used	Test collection and determine risk to benefit of introductions to naïve animals.	Infection should be considered a zoonotic disease since many SIV species can grow in human cell lines <i>in vitro</i> .

Fact Sheet compiled by: Sam Rivera; updated by Natalie Mylniczenko

Sheet completed on: 1 June 2011; updated 10 September 2013, April 2018

Fact Sheet Reviewed by: Lana Krol

Susceptible animal groups: Natural host can be susceptible to disease and older animals may succumb to AIDS-related disease. Non-natural host infections can be fatal. Asian macaques are highly susceptible to fatal infection.

Causative organism: Identified in 45 species, including: SIV_{agm}, SIV_{asc}, SIV_{bkm}, SIV_{blu}, SIV_{col}, SIV_{cpz}, SIV_{deb}, SIV_{den}, SIV_{drl}, SIV_{gor}, SIV_{gsn}, SIV_{l'hoest}, SIV_{mnd} 1 and 2 (possibly 3), SIV_{mon}, SIV_{mus}, SIV_{olc}, SIV_{rcm}, SIV_{schm}, SIV_{smm}, SIV_{stm}, SIV_{sun}, SIV_{syk}, SIV_{tal}, SIV_{wrc}.

Zoonotic potential: The virus should be considered a zoonotic disease. Many SIV species can grow in human cell lines *in vitro*. HIV-1 originated from SIV_{cpz} and SIV_{gor}; HIV-2 from SIV_{smm}.

Distribution: Natural infections occur in Africa. Infection in captive non-human primates occurs worldwide. Cross species viral 'jumping' has been reported but appears relatively rare.

Incubation period: Strain and host dependent. Can be as short as a few weeks in non-natural host or as long as several decades in natural host.

Clinical signs: Clinical disease does not usually present in natural hosts. However, when disease occurs, common findings are lymphadenopathy and diarrhea. Other signs may include wasting, malabsorption, and weight loss. Cardiac disease, arteriopathies, transient cutaneous erythematous maculopapular rash, and CNS involvement can be observed. Secondary infections can be due to immunodeficiency and hypergammaglobulinemia can be observed.

Post mortem, gross, or histologic findings: Lymphoid organs may be hypertrophied. Other findings depend on affected organ systems: encephalitis, cardiac necrosis, myocarditis, coronary or systemic arteriopathy, glomerulosclerosis, pneumonia, follicular hyperplasia and fragmentation in lymphoid tissues, extramedullary hematopoiesis in lymph nodes and follicular and paracortical hyperplasia, epididymitis, prostatitis, urethritis, malignant lymphomas.

Diagnosis: Serology (ELISA, Western blot), PCR, virus isolation. If positive on serology, SIV genotyping is recommended to identify natural reservoirs that are often African non-human primates. Screening is typical with ELISA testing, but confirmation should be completed with Western blot or PCR. It should be noted that highly divergent SIVs may not react completely with HIV and SIV_{mac} antigens used in commercial assays. Viral isolation efficiency is highly variable.

Material required for laboratory analysis: Whole blood, serum/plasma, body fluids, tissues

Relevant diagnostic laboratories:

Primate Assay Laboratory (PAL) *Formerly PDL*
 California National Primate Research Center
 University of California, Davis
 Phone: 530-752-8242 E-mail: cnprc-pdl@ucdavis.edu
<http://www.cnprc.ucdavis.edu/primate-assay-laboratory-core/>

SIMIAN IMMUNODEFICIENCY VIRUSES

Virus Reference Laboratories, Inc.
 VRL-San Antonio, USA
 P.O. Box 40100
 7540 Louis Pasteur, Suite 200
 San Antonio, Texas 78229
 Office: 877-615-7275
 Fax: 210-615-7771
<http://www.vrlsat.com/>

Zoologix Inc.
 9811 Owensmouth Avenue, Suite 4
 Chatsworth, California 91311-3800
 818-717-8880
 Fax: 818-717-8881
info@zoologix.com
<http://www.zoologix.com/>

Treatment: None

Prevention and control: Identify status of animals in collection. Determine risk to benefit of maintaining a closed population in the face of population needs.

Suggested disinfectant for housing facilities: 70% ethanol, formalin, 10% household bleach (sodium hypochlorite), most lipophilic detergents, quaternary ammonium chloride, bezalkonium chloride.

Notification: None at this time.

Measures required under the Animal Disease Surveillance Plan: None at this time.

Measures required for introducing animals to infected animal: Determine current status of both groups of these animals then determine risk to benefit of introducing negative individuals to positive individuals. It is important to remember that natural reservoirs of particular SIV variants exist.

Conditions for restoring disease-free status after an outbreak: Life-long infection results in inability to restore disease free status.

Experts who may be consulted:

William M. Switzer, MPH
 Retrovirus Surveillance Activity Leader, Laboratory Branch
 Division of HIV/AIDS Prevention, NCHSTP
 Centers for Disease Control and Prevention
 1600 Clifton Rd.
 Atlanta, Georgia 30333
bis3@cdc.gov

References:

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SIMIEN IMMUNODEFICIENCY VIRUSES

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Type D SIMIAN RETROVIRUSES (SRV)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Macaques are natural host; langurs, squirrel monkeys, baboons, talapoins	Direct contact; transplacentally	Diarrhea, weight loss, fever, splenomegaly, lymphadenopathy, anemia, neutropenia, lymphopenia, cutaneous fibrosarcoma, and malignant lymphomas	Fatal disease	None	Test and remove/isolate positive animals	Yes

Fact Sheet compiled by: Sam Rivera; updated by Natalie Mylniczenko

Sheet completed on: 1 June 2011, updated September 2013, April 2018

Fact Sheet Reviewed by: Sam Rivera

Susceptible animal groups: Macaques are natural hosts; langurs, squirrel monkeys, baboons, and talapoins also susceptible. Largely, this disease is one of laboratory colonies.

Causative organism: Simian type D retroviruses (SRV); seven genotypes recognized; SRV-1 – SRV-7; Genus (Betaretroviruses)

Zoonotic potential: Zoonotic infection, serologic evidence of human infection in one study.

Distribution: Mostly in Asian macaques and langurs

Incubation period: Unknown, life-long infection

Clinical signs: Immunosuppressive disease (neutropenia and lymphopenia), generalized lymphadenopathy, diarrhea, weight loss, anemia, opportunistic infections. SRV-1 causes malignant lymphomas and SRV-2 abdominal fibromatosis, and subcutaneous fibrosarcomas.

Post mortem, gross, or histologic findings: Splenomegaly, hyperplastic lymphoid follicles, follicular atrophy, fibrosarcomas, polymyositis, nonsuppurative enteritis, sialoadenitis, bone marrow hyperplasia; SRV2: retroperitoneal fibromatosis, subcutaneous fibrosarcomas.

Diagnosis: Serology (ELISA, Western blot for confirmation), PCR, virus isolation. Some animals can have latent infection and be antibody negative. High false positive rate with standard ELISA but newer microbead-based immunoassays have improved specificity.

Material required for laboratory analysis: Whole blood, serum/plasma, saliva, urine, tissues.

Relevant diagnostic laboratories:

Primate Assay Laboratory (PAL) *Formerly PDL*

California National Primate Research Center

University of California, Davis

Phone: 530-752-8242 E-mail: cnprc-pdl@ucdavis.edu

<http://www.cnprc.ucdavis.edu/primate-assay-laboratory-core/>

Virus Reference Laboratories, Inc.

VRL-San Antonio, USA

P.O. Box 40100

7540 Louis Pasteur, Suite 200

San Antonio, Texas 78229

Office: 877-615-7275 Fax: 210-615-7771 <http://www.vrlsat.com/>

Zoologix Inc.

9811 Owensmouth Avenue, Suite 4

Chatsworth, California 91311-3800

818-717-8880 Fax: 818-717-8881 Email: info@zoologix.com

<http://www.zoologix.com/>

Type D SIMIAN RETROVIRUSES (SRV)

Treatment: None
Prevention and control: Test and remove/isolate positive animals.
Suggested disinfectant for housing facilities: 70% ethanol, formalin, 10% household bleach (sodium hypochlorite), Lysol, and most lipophylic detergents can be used.
Notification: None at this time.
Measures required under the Animal Disease Surveillance Plan: None at this time.
Measures required for introducing animals to infected animal: Not recommended.
Conditions for restoring disease-free status after an outbreak: Life-long infection so disease-free status cannot be restored.
<p>Experts who may be consulted: William M. Switzer, MPH Retrovirus Surveillance Activity Leader, Laboratory Branch Division of HIV/AIDS Prevention, NCHSTP Centers for Disease Control and Prevention 1600 Clifton Rd. Atlanta, Georgia 30333 bis3@cdc.gov</p>
<p>References:</p> <ol style="list-style-type: none"> Engel GA, Besnard F. Human–Nonhuman Primate Disease Transmission. In: Fuentes A (ed.). The International Encyclopedia of Primatology. Chester (UK): John Wiley & Sons Ltd.; 2017. Lerche NW. Common viral infections of laboratory primates. <i>In:</i> Wolfe-Coote, S. (ed). The Handbook of Experimental Animals; The Laboratory Primate. San Diego (CA): Elsevier Academic Press; 2005/ p. 75-89. Lerche NW, Cotterman RF, Dobson MD, Yee JL, Rosenthal AN, Heneine WM. Screening for simian type – D retrovirus infection in macaques using nested polymerase chain reaction. <i>Lab Anim Sci.</i> 1997;47:263 -268. Lerche NW. Emerging viral diseases of nonhuman primates in the wild. <i>In:</i> Fowler, M.E. (ed). <i>Zoo & Wild Animal Medicine, Current Therapy 3.</i> Philadelphia (PA): W.B. Saunders Co.; 1993. p. 340-344. Lerche NW, Switzer WM, Yee JL, Shanmugam V, Rosenthal AN, Chapman LE, Folks TM, Heneine W. Evidence of infection with simian Type D retrovirus in persons occupationally exposed to nonhuman primates. <i>J. Virol.</i> 2001;75:1783-1789. Liao Q, Guo H, Tang M, Touzjian N, Lerche NW, Lu Y, Yee JL. Simultaneous detection of antibodies to five simian viruses in nonhuman primates using recombinant viral protein based multiplex microbead immunoassays. <i>J Virol Meth.</i> 2011;178:143-52. Lowestine LJ, Lerche NW. 2003. Nonhuman primate retroviruses and simian acquired immunodeficiency syndrome. In: Fowler ME (ed). <i>Zoo & Wild Animal Medicine, Current Therapy 3.</i> Philadelphia (PA): W.B. Saunders Co.; 2003. p. 373- 378. Montiel NA. An updated review of simian betaretrovirus (SRV) in macaque hosts. <i>J Med Primatol.</i> 2010;39(5):303-314. Voevodin AF, Marx PA. <i>Simian Virology.</i> Ames (IA): Wiley Blackwell; 2009. P. 163-181. Weston-Murphy H, Switzer WM. Occupational exposure to zoonotic simian retroviruses: health and safety implications for persons working with nonhuman primates. In: Fowler ME, Miller RE (eds). <i>Zoo & Wild Animal Medicine, Current Therapy 6.</i> St. Louis (MO): Saunders/Elsevier; 2008. p 251-264.

SIMIAN T-LYMPHOTROPIC VIRUSES

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Old World non-human primates.	Direct contact: bite wounds, sexual contact, sharps puncture (IV, or transdermal). Transmission rate is low.	STLV-1 has been associated with lymphoma, leukemia and wasting disease in several non-human primates; non-Hodgkin's lymphomas and lymphosarcomas	Can be fatal; affects a small percentage of the population	None reported	Test collection and determine risk to benefit of introductions to naïve animals.	Yes, it can infect humans

Fact Sheet compiled by: Sam Rivera; updated by Natalie Mylniczenko

Sheet completed on: 1 June 2011; 10 September 2013; 19 April 2018

Fact Sheet Reviewed by: Sam Rivera

Susceptible animal groups: *Bonobo*, *Cercocebus* sp., *Cercopithecus* sp., *Erthrocebus patas*, *Gorilla gorilla*, macaques, *Mandrillus* sp., *Pan troglodytes*, *Pan paniscus*, *Papio* sp., *Pongo pygmaeus*, *Symphalangus syndactylus*, colobines, and others.

Causative organism: STLV-1 (most likely), STLV-2, STLV-3, STLV-4 that are in genus *Deltaretrovirus*.

Zoonotic potential: STLV can infect humans and causes disease in up to 5% of infected persons. HTLV-1, -2, -3, and -4 originated from STLV-1, -2, -3, and -4 respectively.

Distribution: Africa and Asia naturally, and captive non-human primates worldwide.

Incubation period: Long incubation period has been reported of at least four years; however, it can be shorter in persons receiving blood transfusions from persons with HTLV-1-induced leukemia. Cases are generally spontaneous.

Clinical signs: Mostly reported in laboratory animals in isolated 'outbreaks' where the virus jumped species. Most immunocompetent infected animals are healthy. Disease occurs in a few percent of the positive carriers. Leukemia/ lymphoma syndrome (enlarged lymph nodes, persistent lymphocytosis and abnormal T-cells, T-cell lymphomas and leukemia, lymphadenopathy, and splenomegaly, and non-Hodgkin's lymphomas). Lymphoma without presence of virus is more common in NHPs and in humans disease includes leukemia, lymphoma, inflammatory disorders, and neurologic disease. STLV-4 recently described in wild gorillas.

Post mortem, gross, or histologic findings: Generalized enlarged neoplastic lymph nodes are seen in affected animals. Malignant lymphomas sometimes metastasize, with pale foci or larger nodules found in various organs such as spleen, kidney, and liver. In some individuals, lymph nodes are depleted. Other findings are more variable.

Diagnosis: Serology (IFA and EIA and WB for confirmation) and PCR. Rarely, virus isolation is performed. Care must be taken in interpreting seropositive animals with associated disease manifestations. Lymph node and bone marrow PCR are used to determine disease presence. Dual STLV-1 and STLV-3 infections have been reported in naturally infected simians.

Material required for laboratory analysis: Whole blood, serum/plasma, lymph nodes, bone marrow, and urine.

Relevant diagnostic laboratories:

Primate Assay Laboratory (PAL) *Formerly PDL*

California National Primate Research Center

University of California, Davis

530-752-8242 cnprc-pdl@ucdavis.edu <http://www.cnprc.ucdavis.edu/primate-assay-laboratory-core/>

SIMIAN T-LYMPHOTROPIC VIRUSES

<p>Virus Reference Laboratories, Inc. VRL-San Antonio, USA P.O. Box 40100 7540 Louis Pasteur, Suite 200 San Antonio, Texas 78229 Office: 877-615-7275 Fax: 210-615-7771 http://www.vrlsat.com/</p> <p>Zoologix Inc. 9811 Owensmouth Avenue, Suite 4 Chatsworth, California 91311-3800 818-717-8880 Fax: 818-717-8881 info@zoologix.com http://www.zoologix.com/</p>
<p>Treatment: None</p>
<p>Prevention and control: Identify status of animals in collection. Determine risk to benefit of maintaining a closed population in the face of population needs.</p>
<p>Suggested disinfectant for housing facilities: 70% ethanol, formalin, 10% household bleach (sodium hypochlorite), benzalkonium chloride, and most lipophylic detergents.</p>
<p>Notification: None</p>
<p>Measures required under the Animal Disease Surveillance Plan: None at this time.</p>
<p>Measures required for introducing animals to infected animal: Determine current status of both animal sets, determine risk to benefit of introducing negative individuals to positive individuals.</p>
<p>Conditions for restoring disease-free status after an outbreak: Life-long infection so disease-free status cannot be restored.</p>
<p>Experts who may be consulted: William M. Switzer, MPH Retrovirus Surveillance Activity Leader, Laboratory Branch Division of HIV/AIDS Prevention, NCHSTP Centers for Disease Control and Prevention 1600 Clifton Rd. Atlanta, Georgia 30333 bis3@cdc.gov</p>
<p>References:</p> <ol style="list-style-type: none"> 1. Cournaud V, Van Dooren S, Liegeois F, Pourrut X, Abela B, Loul S, Mpoudi-Ngole E, Vandamme A, Delaporte E, Peeters M. Simian T-cell leukemia virus (STLV) infection in wild primate populations in Cameroon: evidence for dual STLV type 1 and type 3 infection in agile mangabeys (<i>Cercocebus agilis</i>). <i>J Virol.</i> 2004;78:4700-4709. 2. Lerche NW. Common viral infections of laboratory primates. In: Wolfe-Coote, S. (ed). <i>The Handbook of Experimental Animals; The Laboratory Primate.</i> San Diego (CA): Elsevier Academic Press; 2005. p. 75-89. 3. Lowestine LJ, Lerche NW. Nonhuman primate retroviruses and simian acquired immunodeficiency syndrome. In: Fowler, M.E. (ed). <i>Zoo & Wild Animal Medicine, Current Therapy 3.</i> Philadelphia (PA): W.B. Saunders Co.; 2003. p. 373-378. 4. Masters N, Niphuis H, Verschoor E, Breuer J, Quinlivan M, Wawrzynczyk T, Stidworthy M. Debilitating clinical disease in a wild-born captive western lowland gorilla (<i>Gorilla gorilla gorilla</i>) co-infected with varicella zoster virus (VZV) and simian T-lymphotropic virus (STLV). <i>J Zoo Wildl Med.</i> 2010;41:713-716.

SIMIAN T-LYMPHOTROPIC VIRUSES

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SPIRURIDOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Non-human primates, equids, cervids, bovids, camelids, canids, felids, insectivores, birds, and reptiles	Ingestion of intermediate (invertebrate or vertebrate) or paratenic host	Variable, but may include chronic gastritis, vomiting, hemoptysis, anemia, anorexia, weight loss, conjunctivitis, keratitis, and sudden death	Inapparent to severe; many are subclinical	Levamisole, albendazole, mebendazole, ivermectin and other anthelmintics	Control of arthropod intermediate and paratenic hosts; quarantine of shedding animals	Rare, although food-borne infection or vector-borne can occur

Fact Sheet compiled by: Inga F. Sidor; updated by Christopher S. Hanley

Sheet completed on: 31 January 2011; updated 24 August 2013

Fact Sheet Reviewed by: Guilherme G. Verocai; Inga Sidor

Susceptible animal groups: Many vertebrates are susceptible to members of this order of nematode parasites, including wild and captive primates, equids, cervids, bovids, camelids, suids, canids, felids, insectivores, marsupials, rodents, birds, amphibians, and reptiles

Causative organism: Commonly encountered pathogenic spirurids of zoo and wildlife species include nematodes of the genera *Habronema*, *Draschia* (equids, camelids), *Parabronema* (primates), *Thelazia* (mammals, birds), *Spirocerca* (canids, felids, ruminants), *Gongylonema* (primates, ruminants, equids, suids, birds), *Trichospirura* (primates, reptiles, amphibians), *Tetrameres*, *Oxyspirura* (birds), *Physaloptera* (small carnivores, primates, insectivores, rodents), and *Gnathostoma* (carnivores, suids, primates, marsupials).

Zoonotic potential: Most species are not known to cause human disease, although some zoonotic spirurids exist. *Gnathostoma* spp. may be acquired by ingestion of uncooked infected paratenic hosts (fish, frogs, crustaceans), and cause cutaneous, visceral or ocular larva migrans. *Thelazia* spp. Also can affect human eyes and it is transmitted by flies directly into the eyes.

Distribution: Global, more common in warm climates

Incubation period: Variable, typically weeks to months; the life cycle includes an obligate arthropod intermediate host, including house or stable flies, cockroaches, coprophagous beetles, and crickets. Paratenic hosts (rodents and other small mammals, amphibians, reptiles, small birds) may also be involved. *Gnathostoma* are aquatic, with a secondary fish or amphibian intermediate host.

Clinical signs: Most species of spirurids live in the lumen or walls of the upper gastrointestinal tract (oral cavity, esophagus, stomach, proventriculus, or ventriculus); cutaneous or conjunctival infections are also seen (*Habronema* and *Thelazia*, respectively). Signs vary according to site of parasitism and infections are often inapparent, but signs can include esophagitis with aneurysms, chronic gastritis, vomiting, hemoptysis, anemia, anorexia, weight loss, aortic stenosis or aneurysm, or may induce tumors such as sarcoma (*Spirocerca*), acute or chronic pancreatitis (*Trichospirura*), cutaneous ulceration or nodules, ocular discharge, keratitis/conjunctivitis, and/or sudden death.

Post mortem, gross, or histologic findings: Superficial epithelial infections (*Gongylonema*) may result in esophageal epithelial hypertrophy and cornification. With more invasive infections (*Spirocerca*, *Habronema*, and *Tetrameres*), granulomatous or ulcerative lesions of organs develop surrounding necrotic nematodes and caseous debris, including gastritis, esophagitis, and aortitis. Granulomas may be large and

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coalescent, appearing neoplastic. Larval migration may cause focal tissue hemorrhage and necrosis. Nodular granulomatous dermatitis can be seen with cutaneous infections due to erratic larval migration when life-cycle is not completed. Conjunctivitis and progressive keratitis are typical of *Thelazia*.

Diagnosis: Morphological identification of larvae, eggs or adult nematodes. Adults may be recovered from ocular conjunctiva (e.g. *Thelazia*, *Oxyspirura*), including surgical removal, or during necropsy. Because of encysting or encapsulation, for some species, of the adult nematodes in granulomas, fecal shedding of eggs may be intermittent. Imaging techniques such as endoscopy may assist in some cases (e.g. granulomas by *Spirocerca*). Eggs of different species may be difficult to separate morphologically (e.g. *Spirocerca* and *Physaloptera*) and may require larvae to make a definitive identification. Confirmation of infection in biopsies or necropsy tissues may be desired by histopathology. Oral and lingual scraping has been used to identify *Gongylonema* in callitrichids, but results are inconsistent. Molecular techniques, including EM are available for identification of some parasites.

Material required for laboratory analysis: Feces, vomitus, surgical/postmortem lesions

Relevant diagnostic laboratories: Any diagnostic laboratory with routine parasitologic capabilities should be able to diagnose this infection.

Treatment: A variety of anthelmintics have been used to treat these infections, with variable efficacy, including mebendazole, albendazole, levamisole, fenbendazole, ivermectin, doramectin, moxidectin, and milbemycin oxime, but controlled studies are uncommon. Surgical removal of nematodes (*Thelazia*) or granulomas (e.g. *Spirocerca*) may apply.

Prevention and control: Removal of arthropod intermediate hosts (terrestrial and aquatic) or paratenic hosts from enclosures is key to controlling infections. Prophylactic treatment of animals with endectocides or insecticides may prevent contact of arthropod intermediate hosts. In endemic regions, preventative treatment may be possible for some spirurid species. Animals with active fecal shedding or vomiting should be separated from uninfected animals. Quarantine, routine parasitological diagnostics, and prophylactic treatment of new arrivals.

Suggested disinfectant for housing facilities: General measures for cleaning and disinfection should reduce environmental parasite contamination. Bleach or ethanol treatment may reduce viability of spirurid eggs, which are believed not to be very resistant in the environment.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Direct infection is not observed; the indirect life cycle of these parasites makes control of intermediate hosts the most important measure.

Conditions for restoring disease-free status after an outbreak: Undefined as ante mortem testing may be unreliable (due to the low sensitivity of certain techniques) and return to disease-free status may be difficult to ascertain.

Experts who may be consulted:

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Fishes, specifically members of the Family Cyprinidae. Salmonids and a percid have been experimentally infected.	Horizontal transmission (direct, vectors, fomites). Most cases occur in the spring or early summer when the water begins to warm but remains below 15°C. One report of virus isolation from ovarian fluid in carp.	Multiple and varied, including lethargy, pale gills, fecal cast, and branchial hemorrhage.	Mortality with SVCv in carp may reach 100% but is frequently much less.	None	Strict biosecurity and quarantine protocols should be followed based on information available through the OIE and USDA. A DNA vaccine has potential as a method of prevention and control.	No

Fact Sheet compiled by: Gregory A. Lewbart

Sheet completed on: updated 20 May 2018; updated 12 January 2019

Fact Sheet Reviewed by: Kathryn Tuxbury and Elsburgh “Tres” Clarke

Susceptible animal groups: Fishes of the family Cyprinidae. Some notable examples include: carp/koi (*Cyprinus carpio*), golden orfe (*Leuciscus idus*), goldfish (*Carassius auratus*), tench (*Tinca tinca*), *Percocypris pingi*, and sheatfish (*Silurus glanis*). Documented in Chinese firebelly newts (*Cynops orientalis*) in 2016.

Causative organism: Spring Viremia of Carp Virus (SVCv); *Rhabdovirus carpio*

Zoonotic potential: None

Distribution: Global, especially in temperate geographical areas.

Incubation period: Varies depending on water temperature. Latent infections can likely persist for months or even years. Arthropods such as the fish louse (*Argulus* sp.) are likely vectors.

Clinical signs: Infected fish may present with a variety of clinical signs including, but not limited to, abdominal distention, exophthalmia, lethargy, pale gills, darkening of the body surface, fecal casts, skin and branchial hemorrhage, and distention or protrusion of the vent.

Post mortem, gross, or histologic findings: On necropsy, affected fish may have generalized edema which may be sanguineous, organ hemorrhage, intestinal inflammation, and the gastrointestinal tract may contain mucus and no ingesta. Histopathologic examination may reveal multifocal necrosis in liver and pancreas, pericarditis, and renal tubular degeneration.

Diagnosis: Diagnosis is usually made with viral isolation from spleen and/or caudal kidney and/or serum antibody titers and confirmed with virus neutralization. It is important to note that SVCv infected fish also may present with opportunistic Gram-negative bacterial infections.

Material required for laboratory analysis: A minimum of 10 moribund fish or 10 fish exhibiting clinical signs of SVCv must be collected. Fish should be sent live to the laboratory or sacrificed and packed separately in sealed aseptic refrigerated containers or on ice. Depending on the size of fish, whole fish (body length 0-4 cm) or the entire viscera including kidney and encephalon (body length 4-6 cm) should be collected. If the fish is larger, liver, kidney, spleen and encephalon should be collected aseptically. Samples should be combined to form pools of a maximum of five fish per pool that should not exceed 1.5g. Tissues should be placed in sterile vials and stored at 4°C until virus extraction is performed at the laboratory, which is recommended to begin within 24 hours of sample collection.

For detecting asymptomatic carriers, tissue samples of kidney, spleen, gill and encephalon should be collected. Depending on the population size, fish collection must encompass a statistically significant number of specimens. The sampling should be designed in order to enable detection, at a 95% confidence level, of infected animals. Ultra-filtration using large volumes of water can be used to concentrate and isolate the virus.

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<p>Relevant diagnostic laboratories: Various approved state and federal laboratories. Information is available through the USDA web site.</p>
<p>Treatment: None.</p>
<p>Prevention and control: Facilities holding and importing high risk cyprinid fishes should be diligent in following standard quarantine protocols and adhere to appropriate and periodic screening as prescribed by the OIE and USDA. A DNA vaccine utilizing the SVCv glycoprotein gene has proved promising in challenge trials using koi. USDA placing restrictions on import of SVCv susceptible species - gametes, fertilized eggs and live fish.</p>
<p>Suggested disinfectant for housing facilities: The disinfection protocol depends on the size, type and nature of the materials and sites to be disinfected. When an active outbreak of SVCv has occurred, the infected stocks should be depopulated and all areas that held the infected fish must be disinfected. The virus may be inactivated by formalin, ozone, sodium hypochlorite, organic iodophors, gamma and ultraviolet radiation, pH extremes of < 4.0 or > 10.00, and heating at 60⁰ C for 15 minutes. All equipment and tanks, raceways and ponds should be disinfected. The USDA APHIS also recommends if surface water – rather than municipal water source - is used as incoming water to the farms it be treated with sand filtration and UV.</p>
<p>Notification: All suspect cases should be necropsied and the United States Department of Agriculture (USDA) contacted for proper routing of diagnostic samples. Confirmed cases must be reported to the USDA and state veterinarian.</p>
<p>Measures required under the Animal Disease Surveillance Plan: Once an infection is reported, a facility has to follow the recommendations described in the International Aquatic Animal Health Code and the Diagnostic Manual for Aquatic Animal Diseases by OIE to be declared free of SVCv. In the United States, the USDA recommendations must be followed.</p>
<p>Measures required for introducing animals to infected animal: Not applicable.</p>
<p>Conditions for restoring disease-free status after an outbreak: See the OIE web site for most current information. Facilities must be disease free for at least 2 years before disease-free status can be granted. Periodic testing with negative results is required to maintain this status.</p>
<p>Experts who may be consulted: A complete summary of the disease and diagnostic procedures are available through the Office International des Epizooties (OIE) (http://www.oie.int/).</p>
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ST. LOUIS ENCEPHALITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Humans (clinical disease) Other mammals (inapparent infection or clinical illness may be possible) Birds (usually subclinical reservoirs, amplifying hosts, possible illness)	Bite of infected mosquito; theoretical risk of direct contact with infected tissues at necropsy.	Fever, stiff neck, seizures, coma.	In people, mild to severe, can be fatal although most infections are asymptomatic. Illness in animals is not completely understood.	Supportive care - fluids, anticonvulsants; anti-inflammatories.	Prevention of mosquito exposure and bites.	Yes, primarily via mosquito vector.

Fact Sheet compiled by: Rose Borkowski

Sheet completed on: 2 February 2011; updated 13 August 2013, August 1, 2018

Fact Sheet Reviewed by: Michael McBride

Susceptible animal groups: This disease is primarily a concern for humans, especially the elderly, but it may be a concern for other mammals, including nonhuman primates and birds. SLE was isolated from a domestic horse with neurologic disease, raising concern for other equid species. Several zoo animal taxa (mammals and birds) were found to have positive serologic tests for St. Louis encephalitis virus (SLEV) during arbovirus surveillance studies. The relationship of positive SLEV serology to clinical disease in these species was not completely understood. Usually, wild birds are subclinical reservoirs.

Causative organism: A single stranded RNA virus in the genus *Flavivirus*, family Flaviviridae; it is closely related to West Nile virus.

Zoonotic potential Yes – Primarily via mosquito vector, usually *Culex* sp.

Distribution: Human SLEV infections are known from Canada to Argentina. The illness occurs throughout the US, particularly eastern and central states, and an SLE outbreak recently occurred in Arizona

Incubation period: 5-15 days in people

Clinical signs: Wild birds serving as viral reservoirs and amplifying hosts generally do not show signs of illness. Signs of SLE illness in other animal species are incompletely understood, yet the virus was isolated from a horse that succumbed to neurologic disease. In humans, fever, headache, and fatigue are common clinical signs. More serious clinical signs, including stiff neck, altered mental status, seizures, and coma or death, are more likely to occur in the elderly. The case-fatality ratio in humans has been reported as 5-15%. May be a concern for non-human primates.

Post mortem, gross, or histologic findings: In humans, evidence of meningitis and/or encephalitis may be found. Diffuse inflammation of the brain and edema of the substantia nigra have been described.

Diagnosis: Care must be taken to perform virus-specific testing as SLEV cross-reacts with West Nile virus on many diagnostic tests.

Animals: Serology – Plaque Reduction Neutralization Test (PRNT) or virus isolation from various tissues.
Humans: Isolation of virus from, or demonstration of specific SLEV antigen or nucleic acid in tissue, blood, CSF, or other body fluid; four-fold or greater change in SLEV-specific quantitative antibody titers in paired sera; SLEV-specific IgM antibodies in serum with confirmatory PRNT antibodies in the same or a later specimen; SLEV-specific IgM antibodies in CSF and a negative result for other IgM antibodies in CSF for arboviruses endemic to the region where exposure occurred.

Material required for laboratory analysis: Serum, cerebrospinal fluid, and tissues.

ST. LOUIS ENCEPHALITIS

Relevant diagnostic laboratories:

Veterinary samples:

New York State Veterinary Diagnostic Lab (Serology, Plaque Reduction Neutralization Test)

Cornell University

PO Box 5786 (for letters)

240 Farrier Rd (for packages)

Ithaca, NY 14852-5786

Phone: 607-253-3900

Fax: 607-253-3943

diagcenter@cornell.edu

<http://ahdc.vet.cornell.edu>

Louisiana Animal Disease Diagnostic Laboratory (Virus Isolation from Tissues) School

of Veterinary Medicine

1909 Skip Bertman Drive, Room 1519

Baton Rouge, LA 70803

laddl_info@vetmed.lsu.edu

Human Samples:

Contact CDC, state or county public health departments for appropriate laboratories

Treatment: Supportive care is based on clinical signs at presentation. No specific treatment regime is available for SLEV infection and illness

Prevention and control: No vaccine is available. Prevention of mosquito bites is important by use of repellants, protective clothing, screens, and fans. Reduction in mosquito presence includes elimination of standing water in containers that can support mosquito breeding and modification of animal enclosures to reduce areas for mosquito access and breeding. Efforts to limit exposure of animals and humans to insect vector should be taken (e.g. indoors housing at night to avoid exposure during times of peak mosquito feeding activity or repellent application). Use personal protective equipment and proper sharps handling when working with infected animals or their tissues. Prevent aerosolization of virus and contact of infected tissues and fluids with skin and mucous membranes. Do not use mechanical saws to obtain spinal cord samples due to risk of aerosolization. Additional recommendations for handling of potentially infected tissues include use of 3 pairs of gloves (inner layer disposable, middle layer waterproof, and outer layer of metal or Kevlar gloves), face shield or goggles plus a disposable "half mask" high efficiency particle arresting (HEPA) respirator.

Suggested disinfectant for housing facilities: Sodium hypochlorite 500 – 5000 ppm, 2% glutaraldehyde, 2-3% hydrogen peroxide, 1% iodine, and ethanol can be used. The virus may be inactivated by UV light.

Notification: SLE in humans is notifiable to State Public Health Departments. If the disease occurs in an animal, state veterinary regulations should be queried for requirements to report the disease.

Measures required under the Animal Disease Surveillance Plan: None at this time.

Measures required for introducing animals to infected animal: SLE is not known to be transmissible

between mammals, birds, reptiles, or people except via the bite of an infected mosquito. A newly diagnosed SLEV-positive animal indicates that SLEV-infected mosquitoes have been active in the area. In people, infection with SLE is believed to confer lifelong immunity to subsequent SLE infection.

Conditions for restoring disease-free status after an outbreak: If an outbreak of SLE among zoo animals is verified, information-gathering regarding SLEV serologic status of sentinel animals managed by public health officials may be prudent. Thorough evaluation and enhancement of mosquito control efforts would also contribute to restoration of disease-free status.

ST. LOUIS ENCEPHALITIS

Experts who may be consulted:

Division of Vector-Borne Diseases
Centers for Disease Control and Prevention
3156 Rampart Road
Ft. Collins, Colorado 80521
800-CDC-INFO

References

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STAPHYLOCOCCUS SPP. INFECTIONS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals, including humans; birds; reptiles	Opportunistic pathogens often involving breaks in the skin. Ubiquitous, and live free in the environment and commensal parasites of skin and upper respiratory tract. Droplet, direct/indirect contact transmission can occur.	Can affect every organ system and clinical signs depend upon organ affected. Common cause of dermatitis. Fever, anorexia, pain, abscesses and infections of the skin, eyes, ears, respiratory system, mammary glands, genito-urinary tract, skeleton, joints. Toxins may produce signs of food poisoning.	Depends upon organ(s) affected and immune status of host.	Antibiotics: First-choice antibiotics (pending culture and sensitivity testing) include cephalosporins and fluoroquinolones. Antibiotic resistance is common so sensitivity testing is recommended.	Appropriate wound care. Frequent hand washing. Sanitation of environment. Avoid abrasions or injury to skin. Isolate animals under treatment. Appropriate PPE for animal care-takers.	Yes

Fact Sheet compiled by: June Olds

Sheet completed on: 5 April 2011; updated 7 March 2013; updated February 21, 2018

Fact Sheet Reviewed by: Leah Greer

Susceptible animal groups: All mammals, including humans; birds; reptiles.

Causative organism: *Staphylococcus* spp. of various species, but not MRSA. *Staphylococcus* spp. are Gram positive, facultative anaerobic cocci occurring typically in clusters, although pairs and short chains do occur.

Zoonotic potential: Yes

Distribution: Worldwide, ubiquitous. Staphylococcal species have been identified as part of the normal microflora in the nasal mucosa and intestinal tract of wild, freshly shot agouti from various areas of Trinidad and are expected to be part of the normal flora of most mammals, humans, birds and reptiles.

Incubation period: Interval of 2-10 days although signs of poisoning from food contaminated with toxins may occur within 30 minutes and up to 6 hours following ingestion.

Clinical signs: Members of the genus *Staphylococcus* are among the most common pyogenic or pus-inducing bacteria, causing local abscesses and generalized infections in a wide variety of species. Depending upon organ system affected, and if bacteremia and septicemia occur, clinical signs may include: pneumonia, endocarditis, meningitis, metritis, peritonitis, osteomyelitis – all organs are susceptible. Dermatitis and local abscesses are common. The organisms are opportunistic pathogens that require some damage to skin or mucous membranes to become established in underlying tissues. Staphylococcal infections in wild rabbits may result in severe and sometimes fatal disease. Clinical signs of the disease are non-specific. Infected lagomorphs may be listless, emaciated, and lame if joints or tendons are involved. Large subcutaneous abscesses may be visible externally, as well as swelling and draining tracts, resulting in crusting of the hair. Infected areas of the skin are usually crusted with exudate. Staphylococcal organisms have also been identified as part of mixed infections with fusobacterium spp. and actinomyces spp. in mandibular osteomyelitis in wallabies and kangaroos.

Post mortem, gross, or histologic findings: Purulent inflammation of any organ can be produced. Skin: (*Staph* pyoderma), abscesses, cellulitis, necrotizing dermatitis. It is also a common cause of pneumonia, endocarditis, osteomyelitis, urinary tract infection, septicemia, mastitis, and meningitis.

Diagnosis: Clinical picture and/or gross presentation of skin lesions (pyoderma) observed. Inflammatory leukogram is often present. Large Gram-positive cocci, arranged in clusters, are readily found in smears of exudate from lesions. Culture and sensitivity testing of affected tissues should be performed for definitive diagnosis.

STAPHYLOCOCCUS SPP. INFECTIONS

<p>Material required for laboratory analysis: Culture (aerobic) of affected tissues with media designed for facultative anaerobe. The organism grows well on 5% blood agar media.</p>
<p>Relevant diagnostic laboratories: Any lab capable to perform microbiology culture/sensitivity testing should be able to identify this organism.</p>
<p>Treatment: Appropriate antibiotics can be guided by culture and sensitivity testing.</p>
<p>Prevention and control: Appropriate antibacterial disinfectants that list efficacy against <i>Staphylococcus</i> should be used. Clean environments reduce skin contamination and decreasing risk of skin trauma reduces entry point. Clean environment with dilute bleach solution to the extent possible.</p>
<p>Suggested disinfectant for housing facilities: Use disinfectants that list efficacy against <i>Staphylococcus</i>. Clean environment with dilute bleach solution to the extent possible.</p>
<p>Notification: None</p>
<p>Measures required under the Animal Disease Surveillance Plan: None</p>
<p>Measures required for introducing animals to infected animal: Clear infection and sanitize environment to the extent possible prior to introductions.</p>
<p>Conditions for restoring disease-free status after an outbreak: Resolution of infection in affected animals, sanitation of environment.</p>
<p>Experts who may be consulted: These are common bacterial pathogens, most diagnostic laboratories and bacteriologists should be familiar with the diseases associated with these bacteria.</p>
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STREPTOCOCCUS GROUP C

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Equines as “Strangles”; pneumonias, or reproductive disease. Swine as pneumonias and polyarthritis. Ruminants as mastitis and polyarthritis. Marine mammals, birds, and salmon as septicemias. Fish as ulcers.	Inhalation; ingestion; during breeding; transplacental. Indirectly via hands and/or fomites. Direct contact with infectious exudates. Undercooked horsemeat.	Variable based on organ system affected. Abscesses; pharyngitis; cellulitis; septicemias; rhinitis; ocular discharge; coughing; sneezing; draining tracts. Abortions. Mastitis.	Severity can range from mild to severe or fatal, depending on age, species, and immune status of the individual.	1 st choice: Procaine penicillin and Ampicillin. 2 nd choice: Cephalosporins, Chloramphenicol, macrolides, Rifampin, and Trimethoprim-sulfas.	Vaccination and isolation.	Yes, although rarely and mostly in immunocompromised individuals. Death has occurred. <i>Strep. zooepidemicus</i> has been the main isolate in those cases. <i>Strep. dysgalactiae</i> is also zoonotic.

Fact Sheet compiled by: Christie Hicks

Sheet completed on: 30 April 2011; updated 7 August 2013; updated 14 February 2018.

Fact Sheet Reviewed by: Ryan Colburn

Susceptible animal groups: Virtually all mammals, including humans, can be susceptible. Equine, swine, ruminants, and marine mammals are at risk and published reports include pyometra in a spotted seal. Birds, salmon, and other fish species also can be affected. 3 separate outbreaks in shelters have led to the death of many dogs with *Streptococcus zooepidemicus* as the cause.

Causative organism: *Streptococcus* species classified into Lancefield Group C, which are Gram positive cocci occurring in pairs and chains.

Zoonotic potential: *Streptococcus zooepidemicus* has been reported as the cause of zoonosis in several individuals with some cases leading to death. Possible routes of zoonotic infection are: consumption of infected milk and milk products, exposure to bodily fluids or contaminated fomites, or occupationally during the care of infected individuals. *Streptococcus dysgalactiae* is now being seen more frequently in people.

Distribution: Widely distributed worldwide.

Incubation period: *Streptococcus equi* subsp *equi*: 3 to 14 days. In humans it can be significantly shorter.

Clinical signs: An abscess filled with purulent material especially around the head and neck. Fever, nasal discharge, pharyngitis, rhinitis, ocular discharge, coughing, sneezing, draining tracts, and more rarely cellulitis and septicemia can be seen.

Streptococcus equi subsp *equi* is highly infectious. Retropharyngeal and submandibular lymph node swelling and abscesses can progress to affect other organs such as the mesentery, liver, spleen, kidney, brain and less commonly the thorax. Classic “Strangles” is typically limited to the head and neck regions. But when the disease progresses past these areas it is known as metastatic strangles or “Bastard Strangles” and can result in colic like symptoms, fever, and/or weight loss. Purpura hemorrhagica can develop secondary, a Type III hypersensitivity presenting with ventral and limb edema, petechia and ecchymoses, and result in renal and muscle disease. This infection has been considered a possible link in Idiopathic Hemorrhagic Vasculopathy Syndrome in black rhinos.

Streptococcus equi subsp *zooepidemicus* causes mastitis, abortions and infertility in adults, and pneumonias in adults and foals. Purulent rhinitis and bronchitis in weanling foals. Cases of fibrinous pleuritis and pneumonia in sheep, mastitis in goats, and hemorrhagic pneumonia in dogs have been reported.

Streptococcus dysgalactiae is seen in cattle as mastitis. Piglets, lambs, goats, and calves as polyarthritis. Acute death in puppies. And recently it has been stated to cause ulcers in fish.

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Post mortem, gross, or histologic findings: Abscesses tend to be fluid filled with *Streptococcus equi* subsp *equi*. Empyema with or without chondroids may be found in the guttural pouches. Metastasis is most commonly to the mesenteric lymph nodes. *Streptococcus equi* subsp *zooepidemicus* is associated with consolidation and adhesions of the lungs with debris in the airways. Reproductive disease is associated with placentitis especially around the cervical star. *S. dysgalactiae* causes endocarditis with yellow or white vegetations of varying sizes, fibrous and multifocal abscesses of tissues, and hypertrophy of synovial villi.

Diagnosis: On CBC: anemia, neutrophilic leukocytosis, and hyperfibrinogenemia are present while the chemistry panel remains unremarkable. Polymerase chain reaction is the most sensitive and efficient. Growth of the organism on cow or sheep blood agar at 37°C in 3 – 5% CO₂ or using the CAMP phenomenon. Ultrasound, endoscopy (particularly of the guttural pouches) and/or radiographs may be helpful to determine the extent of the abscesses and infection.

Material required for laboratory analysis: Aspirates from unopened abscesses collected in a sterile manner and/or milk collected under sterile conditions can be cultured. Aspirates and washes from the nasopharyngeal and guttural pouches can be submitted for *Streptococcus equi* subsp *equi* PCR and culture.

Relevant diagnostic laboratories: Any laboratory that performs cultures and sensitivities on a routine basis can complete testing for this organism. *Streptococcus equi* subsp *equi* PCR can be found at many major commercial and veterinary diagnostic laboratories.

Treatment: Antibiotics that are found to be effective against *Streptococcus* Group C on culture: penicillins, cephalosporins, macrolides, Chloramphenicol, Rifampin, and Trimethoprim-sulfonamides have proven to be effective. Procaine Penicillin is the antibiotic of choice. However, the use of antibiotics is controversial in *Streptococcus equi* subsp *equi* unless given in the early stages of disease because many clinicians feel that treatment at later stages only prolongs the course of disease. Antibiotic use should therefore be dependent on the severity of disease and can help limit shedding. The use of antibiotics may prevent the development of natural immunity therefore, re-infection is possible. Encouraging assessable lymph nodes and abscesses to drain via warm compresses, aspiration, and/or lancing may help to speed up the recovery process. Guttural pouch flushes with a gelatin/benzyl penicillin mixture may also help. Administer NSAIDS or corticosteroids to help decrease fever and provide analgesia.

Prevention and control: *Streptococcus equi* subsp *equi*: Intramuscular vaccination has proven to not be completely protective but can help decrease the severity of disease. Injection site reactions are possible. An intranasal product with a live attenuated strain of *Streptococcus equi* subsp *equi* is available and used commonly, except in Europe. It is recommended to vaccinate with 2 initial boosters separated by 2 weeks and then annually. Vaccination of any kind is not recommended for exposed horses at a facility with an ongoing outbreak and for 2 years afterwards due to the increased risk of purpura hemorrhagica. Vaccine titers can be performed. It is important to monitor temperatures and isolate febrile animals as minimal to no shedding occurs within the first 48 hours. Isolate the infected individuals as recovered individuals because they can still shed the bacteria for months. Examination of guttural pouches can identify carriers. Prophylactic treatment of exposed animals may be considered.

Foals should be appropriately vaccinated for respiratory viruses to help prevent secondary bacterial infections. Limit crowding when housing foals.

Suggested disinfectant for housing facilities: Clean with detergents and then disinfect either with; chlorhexidine gluconate or glutaraldehyde.

Notification: Not federally reportable but *Streptococcus equi* subsp *equi* is reportable in some states.

Measures required under the Animal Disease Surveillance Plan: Currently none.

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Measures required for introducing animals to infected animal: *Streptococcus equi* subsp *equi* can live outside the host for several weeks and can be shed for at least 4 weeks, so all facilities should not accept any new individuals for at least 1 month after an outbreak has resolved. Specifically, for *S. equi* subsp *equi*, all new animals should observe a 21-day quarantine period with three negative nasopharyngeal wash PCRs or one guttural pouch wash PCR obtained before entry into the group.

Conditions for restoring disease-free status after an outbreak: Careful monitoring of those that are infected for a resolution of clinical signs and all blood parameters returning to normal. Following clinical resolution, three negative nasopharyngeal PCRs separated by 4 to 7 days should be performed before recovered individuals are allowed back into the group with a minimum of 1 month of isolation. It should be noted that some individuals can become prolonged shedders for months with the source being within the guttural pouch therefore, PCR of the guttural pouch may prove beneficial.

Experts who may be consulted: Any laboratory that routinely tests for this bacterium, as well as large animal internists and equine veterinarians.

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STRONGYLOIDIASIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primates, especially young, captive orangutan. Various <i>Strongyloides</i> spp. can affect humans, canids, felids, suids, equids, ruminants, rodents, birds.	Larvae or ova shed in feces, develop into free-living adults or infectious larvae that can penetrate skin. They then migrate to intestines, (many species go through lungs on the way to gut); also some species are transmitted transmammary. Some species can autoinfect within intestines and produce pulmonary hyperinfection.	Often insidious; acute lethargy or sudden death, diarrhea, abdominal distension and discomfort, nausea, anorexia, cough, shortness of breath.	Can cause severe disease and death from hyperinfection in young animals; typically subclinical in immunocompetent adults.	Usually unsuccessful in severe symptomatic cases. However, can attempt to treat with ivermectin and/or benzimidazoles	Ivermectin, also benzimidazoles; improve hygiene to reduce fecal contamination	Yes

Fact Sheet compiled by: Ellen Bronson, med. vet., DACZM

Sheet completed on: 31 January 2011; updated 12 March 2013; updated 24 December 2017

Fact Sheet Reviewed by: Tom Nolan, PhD; Christy Rettenmund, DVM, DACZM

Susceptible animal groups: All vertebrates. Primates, especially captive orangutans < 5 yr; also canids, felids, suids, ruminants, equids, rodents.

Causative organism: *Strongyloides stercoralis* in primates, domestic dog; *S. fuelleborni* in Old World primates; *S. cebus* in New World primates. Other *Strongyloides* spp. reported in other primates, suids, felids, equids, ruminants, rodents, birds, and reptiles.

Zoonotic potential: Yes (*S. stercoralis* and *S. fuelleborni*); infective larvae can penetrate intact skin, also fecal-oral transmission possible. Common parasite of humans in subtropical and tropical climates. Reported in human caretakers in orangutan rehabilitation facilities.

Distribution: Worldwide with different geographic strains and species. It is most prevalent in tropics and subtropics, also endemic in Southeastern US.

Incubation period: 1-2 weeks in most species; individuals can be chronically affected.

Clinical signs: *Strongyloides* spp. infections are usually subclinical in adult immunocompetent animals. In young or immunocompromised primates with disseminated hyperinfections due to autoinfection, sudden death without premonitory signs is seen. Other clinical signs include abdominal pain, diarrhea, paralytic ileus, constipation, cough, shortness of breath, urticaria, and rash.

Post mortem, gross, or histologic findings: Petechiae and ecchymoses in lungs, pulmonary hemorrhage, erosive or ulcerative enterocolitis. Adult parasites, larvae, and eggs in pulmonary (very rare) and intestinal mucosal tissue on histologic examination and can also be found in other tissues (lymph nodes, liver, etc) in disseminated infections. In hyperinfections, secondary bacterial septicemia, pneumonia, and meningitis are common.

Diagnosis: With *S. stercoralis* infection, rhabditiform (or less frequently filariform) larvae can be seen in feces with Baermann fecal exam or direct wet mount or with charcoal or Horadi-Mori fecal culture, but diagnosis often challenging due to infrequent shedding in feces. In infants, severe tissue destruction and death can occur before fecal shedding begins. Eggs can be seen in feces with *S. fuelleborni* infections. In hyperinfections, may be able to detect larvae in sputum or respiratory tract mucus. Eosinophilia possible during acute and chronic stages. *S. stercoralis* ELISA and other serology available for humans; levels shown to decrease after treatment in humans. ELISA has been used in orangutans but has not been validated, and usefulness in non-humans is unknown.

Material required for laboratory analysis: Fresh feces for fecal exam; serum for ELISA antibody testing.

STRONGYLOIDIASIS

Relevant diagnostic laboratories:

Most reference laboratories can perform Baermann fecal exams.

ELISA antibody test available through CDC for human cases, but not currently commercially available for non-human primates or other species.

Treatment: Difficult in symptomatic or chronically infected animals but can reduce burdens with ivermectin, albendazole, other benzimidazoles. Ivermectin treatment is the preferred treatment and is usually 2 doses one week apart resulting in rapid amelioration of clinical signs. Treatment with benzimidazoles usually is daily for two weeks with no change in clinical signs expected before 3 to 7 days. Aggressive combination treatment recommended for hyperinfection cases. A second dosing of antiparasitic a week after the end of the first treatment is usually needed to kill adults developing from larvae that were migrating in the tissues during the first treatment. Treatments should always be performed in combination with control measures to prevent reinfection during treatment since the larvae are not killed by the same dosages as the adults.

Prevention and control: Daily removal of feces to break cycle; if animals on soil will not be able to break cycle. Can keep burdens low with regular anthelmintics. Due to high morbidity/mortality in captive orangutans, monthly anthelmintic strongly recommended for all members of orangutan groups with infants, juveniles, or pregnant adults. Monthly ivermectin is the most commonly used preventative regimen for orangutans.

Suggested disinfectant for housing facilities: Mechanical removal of feces most important, cleaning with soap and water and complete drying is recommended. Quaternary ammonium products containing N-alkyl dimethyl benzyl ammonium chloride or didecyl dimethyl ammonium chloride will kill infective larvae very rapidly and are suitable for hard solid surfaces. Steam cleaning also effective for disinfecting housing areas.

Notification: None

Measures required under the Animal Disease Surveillance Plan: N/A

Measures required for introducing animals to infected animal: Consider prophylactic deworming regimen during introduction phase and frequent fecal examinations, but transmission likely difficult to avoid; goal should be to avoid clinical signs, especially in groups with young primates.

Conditions for restoring disease-free status after an outbreak: If animals have access to soil it will be impossible to eliminate parasite. If area can be completely disinfected, can attempt daily complete removal of feces, cleaning/drying area, but if chronically infected animal is present, will be unlikely to eliminate infection.

Experts who may be consulted:

Thomas J. Nolan, Ph.D.

Director of the Clinical Parasitology Laboratory

University of Pennsylvania School of Veterinary Medicine

Phone: 215-898-7895 Email: parasit@vet.upenn.edu

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SWINE VESICULAR DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Domestic pigs, wild boar.	Direct – not common - contact with infected pig, fecal transmission. Indirect – feeding uncooked infected pork products, fomites.	Pyrexia, anorexia, lameness, vesicles progression to erosions (coronary bands, snout, lips, oral cavity, teats).	Moderately contagious. Moderate to high morbidity. Very low mortality.	None	Rare persistent carriers. Test and quarantine animals, disinfect environment, do not feed uncooked pork products.	Possibly

Fact Sheet compiled by: Cora Singleton

Sheet completed on: 8 August 2018.

Fact Sheet Reviewed by: Marjorie Bercier

Susceptible animal groups: Domestic pigs, wild boar.

Causative organism: An enterovirus of the *Picornaviridae* family.

Zoonotic potential: Possible. More recent studies suggest that the virus has adapted to swine and has lost its ability to infect humans. SVDV is closely related to human coxsackievirus B5.

Distribution: Europe, some parts of Asia. Exotic to the United States.

Incubation period: 2-7 days

Clinical signs: Pyrexia, anorexia, lameness, vesicles progressing to erosions (coronary bands, snout, lips, oral cavity, and teats), horn of hoof occasionally shed. May present with unsteady gait with jerky leg movements due to encephalitis. Young animals are usually more severely affected. Clinically indistinguishable from foot and mouth disease, vesicular exanthema of swine, Seneca virus A, and vesicular stomatitis. Recovery within 2-3 weeks, presence of a dark horizontal line on the hoof where growth had stopped.

Post mortem, gross, or histologic findings: Vesicles on coronary bands, snout, lips, oral cavity. Hydropic degeneration and edema of stratum spinosum of the affected epidermis, followed by ballooning degeneration of keratinocytes that then float into the vesicular fluid. Stratum basale remains intact. A nonsuppurative meningoencephalitis and necrotizing myocarditis and endocarditis have been reported.

Diagnosis: Agent identification – virus culture along with electron microscopy, ELISA (method of choice), complement fixation, RT-PCR

Serology – ELISA, virus neutralization; also double immunodiffusion, radial immunodiffusion, counter-immunoelectrophoresis.

Material required for laboratory analysis: Vesicular fluid, epithelium covering a vesicle, heparinized whole blood, serum, feces, tissues in formalin.

Relevant diagnostic laboratories:

Foreign Animal Disease Diagnostic Laboratory, Plum Island

40550 Route 25 (for packages)

Orient Point, NY 11957

P.O. Box 848 (for letters)

Greenport, NY 11944-0848

(631) 323-3256

Fax: (631) 323-3366

Since vesicular diseases cannot be distinguished clinically, contact the proper authorities prior to sample collection and shipment.

Treatment: No effective treatment. Supportive care and treatment of secondary problems.

Prevention and control: Prevention should include no feeding of uncooked pork products, regulation of movement of animals and animal products, and serologic monitoring to detect infections. No vaccine is available. Control measures include notification of authorities, quarantine or depopulation of infected animals,

SWINE VESICULAR DISEASE

and disinfection of the environment.
Suggested disinfectant for housing facilities: Phenols, sodium hydroxide, formalin, sodium carbonate, ionic and non-ionic detergents, strong iodophors in phosphoric acid, chloroform. The virus is extremely persistent in the environment, thus difficult to eradicate.
Notification: Reportable to the USDA/APHIS through the State Veterinarian or the federal Area Veterinarian in Charge. The disease is also reportable to the World Organization for Animal Health (OIE).
Measures required under the Animal Disease Surveillance Plan: Reportable disease.
Measures required for introducing animals to infected animal: Not recommended.
Conditions for restoring disease-free status after an outbreak: Infections must be reported to USDA/APHIS for management.
Experts who may be consulted: USDA State Veterinarians or federal Area Veterinarians in Charge.
<p>References:</p> <ol style="list-style-type: none"> Alexandersen S, Knowles NJ, Belsham GJ, Dekker A, Nfon C, Zhang Z, Koenen F. Picornaviruses. <i>In:</i> Zimmerman JJ, Karriker LA, Ramirez A, Schwartz KJ, Stevenson GW, Zhang J (eds.). Diseases of Swine, 11th ed. Ames (IA): Wiley-Blackwell; 2018. <i>in press</i>. Jackson PGG, Cockcroft PD. Handbook of Pig Medicine. London (England): Saunders Elsevier; 2007. p. 186-187. Knowles NJ, McCauley JW. Coxsackievirus B5 and the relationship to swine vesicular disease virus. <i>Curr. Top. Microbiol. Immunol.</i> 1997;223:153–167. Spickler, AR [Internet]. Swine vesicular disease; 2015 [cited 2018 August 08]. Available from http://www.cfsph.iastate.edu/Factsheets/pdfs/swine_vesicular_disease.pdf. United States Department of Agriculture, Animal Plant and Health Inspection Services [Internet]. All National Animal Health Laboratories Network (NAHLN) Laboratory List; 2018 [cited 2018 August 08]. Available from http://www.aphis.usda.gov/animal_health/nahln/downloads/all_nahln_lab_list.pdf. United States Department of Agriculture, Animal Plant and Health Inspection Services [Internet]. Diagnostic Testing at the National Veterinary Services Laboratories; 2015 [cited 2018 August 08]. Available from http://www.aphis.usda.gov/aphis/ourfocus/animalhealth/lab-info-services/sa_diagnostic_tests/ct_diagnostic_tests. World Organization for Animal Health (OIE) [Internet]. Swine vesicular disease. <i>In:</i> Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2018; 2018 [cited 2018 August 08]. Available from http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.08.08_SVD.pdf. Fernandez PJ and White WR. Swine vesicular disease. <i>In:</i> Atlas of Transboundary Animal Diseases. World Organization for Animal Health (OIE). 2010; 237-242.

TANAPOXVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primates, including humans	African species are usually source for Asian species. Humans infected via skin wounds.	Erythematous 2-3cm raised, thickened skin lesions with umbilicated centers that developing within days to weeks of contact. Lesions often on face.	Mild to moderate severity. Increased severity with immuno-compromise conditions	Supportive as lesions usually have spontaneous regression.	Avoid cohabitation of African and Asian non-human primate species. Disinfection of fomites and vector control.	Yes

Fact Sheet compiled by: E. Marie Rush

Sheet completed on: 3 December 2010; updated 15 July 2013; May 1 2018.

Fact Sheet Reviewed by: Marc Valitutto

Susceptible animal groups: Primates, human and non-human

Causative organism: Tanapoxvirus (genus *Yatapoxviridae*)- Principle reservoir is unknown, thought to be a non-human primate.

Zoonotic potential: Yes

Distribution: Sub-Saharan Africa (originated in Tana River Valley of Kenya). Cases have been reported from travelers in Tanzania and during WHO smallpox eradication in Central Africa.

Incubation period: Unknown, but clinical signs can appear within days of inoculation. Replication has been shown in owl monkey renal cells.

Clinical signs: In non-human primates, vesicles may be numerous, are often around the upper body and head region, and appear within 2-3 weeks of inoculation. In humans, often a single – occasional clusters of 10 lesions - erythematous, thick-walled dermatologic vesicle/ papule is noted often on the extremities or lower body regions, and the patient may have prostration, general body ache or headache, tender regional adenopathy, and prodromal (2-4 days) pyrexia prior to lesion onset. Lesions may reach maximal size by two weeks then typically regress spontaneously within 4-6 weeks. Pruritus may accompany lesions. This disease is clinically virtually indistinguishable from Yaba-like disease virus, which is in the same genus *Yatapoxviridae*, but is different from Yaba Monkey tumor virus, also in the same genus. In humans, risk for secondary bacterial infections in humans.

Post mortem, gross, or histologic findings: Grossly apparently epidermal lesions that when biopsied, show marked thickening and ballooning degeneration of prickle cell layer and eosinophilic viral inclusion bodies characteristic of poxviruses on histopathology and enveloped forms seen on EM.

Diagnosis: History of direct or indirect contact with non-human primates (or transport from or travel to Africa), complement fixation, serum neutralization and precipitation tests, ELISA, and PCR.

Material required for laboratory analysis: Serum, tissue for histopathology or EM

Relevant diagnostic laboratories: This is an uncommon disease, but has been noted in North American collections. Most laboratories that process non-human primate samples can either run the PCR for this virus or can direct personnel accordingly to an appropriate laboratory facility for testing of samples. Histopathology or EM can be done at most laboratories that normally process tissues and have the capabilities for these procedures.

Treatment: Supportive – spontaneous resolution usually in ~6 weeks in humans.

Prevention and control: Avoid contact with primates that have had potential exposure. Proper quarantine and testing of animals with history of exposure or recent shipment from Africa. Humans should keep all skin wounds cleaned, bandaged and covered when working with non-human primates. Thorough

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disinfection of all potential fomites in housing areas for primates in collections and protection of animal care staff through education and proper clothing and protective wear (gloves, long sleeves). Vector control. Previous exposure/immune reaction to Yaba-like disease virus may provide immunity for tanapox, but not visa-versa.

Suggested disinfectant for housing facilities: Detergents, hypochlorite, alkalis, Virkon® and glutaraldehyde.

Notification: Public health officials may need to be notified if zoonotic transmission occurs, depending on the state.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Do not introduce animals with clinical disease (active or resolving pustules/lesions) to non-infected or new animals. Allow resolution of all lesions completely prior to introduction and follow proper quarantine measures for individual facility.

Conditions for restoring disease-free status after an outbreak: Condition typically spontaneously resolves within weeks with supportive care. Treatment of any secondary infections should assist in wound healing. Immunosuppressed animals may be more susceptible to infection and secondary disease/complications. Proper disinfection of animal area and fomites should be done following an outbreak or care of an infected animal prior to housing new animals in the area.

Experts who may be consulted:

Centers for Disease Control and Prevention
Poxvirus and Rabies Branch, Division of High-Consequence Pathogens and Pathology
1600 Clifton Rd
Atlanta, GA 30333
800-CDC-INFO

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TANAPOXVIRUS

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TOXOPLASMOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All vertebrates (primarily birds and mammals)	Ingestion of oocysts from felid feces; ingestion of tissue cysts; transplacental; transmammary	Variable, depending on species and organs affected. Can range from asymptomatic to sudden death.	Variable depending on species. Causes severe disease in Australian marsupials, New World primates, and lemurs. Usually asymptomatic in most felids.	Atovaquone, clindamycin, sulfonamide.	Prevent exposure to felid feces. Control intermediate hosts in environment.	Yes

Fact Sheet compiled by: Joseph A. Smith

Sheet completed on: 30 June 2011; updated 15 July 2013

Fact Sheet Reviewed by: Charles Faulkner; Jitender P. Dubey

Susceptible animal groups: Felids are the only definitive host for *Toxoplasma gondii*. Australian marsupials, lemurs, New World primates, brown hares, southern sea otters, and pronghorn antelope are reported to be highly susceptible. Cattle, rats, horses, Old World monkeys, and turkeys are reported as relatively resistant to clinical disease. Pallas' cats are an exception to most felids in that a positive queen's immune response does not prevent congenital transmission.

Causative organism: Toxoplasmosis is caused by the obligate intracellular coccidian *Toxoplasma gondii*. Felids are the definitive host and are the only taxa known to transmit infective oocysts in feces. Other species are most frequently infected by ingestion of oocysts from felid feces, which may survive for months to years in the environment. Once ingested by an intermediate host, the organism forms tachyzoites that rapidly reproduce in host tissues. Tachyzoites are the cause of most clinical signs. Tachyzoites can then transform into thin-walled tissue cysts containing bradyzoites. The life cycle is completed when felids ingest the tissue cysts from prey species. Other non-felid carnivorous species may also become infected from ingestion of tissue cysts, but are unable to complete the life cycle and do not produce infective oocysts in feces; however, they – as prey species – can become carrier hosts which are infective, and usually termed intermediate hosts in the literature, although they are not required to complete the life cycle.

Zoonotic potential: In the US, it is estimated that 22.5% of the population has been infected with toxoplasmosis with this number approaching 95% in some other parts of the world. Transmission can occur from ingestion of oocysts passed in cat feces (e.g. cleaning pet litter boxes, gardening/contact with contaminated soil, contaminated produce), ingestion of undercooked meat, transplacentally, or rarely through blood transfusions and organ transplants. Most infections are asymptomatic or cause mild self-limiting flu-like symptoms. Infections acquired during pregnancy can cause abortion, congenital defects, or more severe disease in the child. Clinical signs in the child, including ocular disease, seizures, and mental disability, may not be present until later in life. Infections in immunocompromised persons may be severe.

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<p>Immunosuppression may also cause a recrudescence of an infection that was acquired earlier in life.</p>
<p>Distribution: Worldwide anywhere felids are present or have been introduced. Runoff water infected with oocysts can introduce the organism to ocean environments.</p>
<p>Incubation period: Infections acquired from ingestion of tissue cysts have a 3-10 day prepatent period in felids. Infections acquired from ingestion of oocysts have a 19-48 day prepatent period in felids. Oocysts passed in feces become infective after 1-5 days in the environment. Felids can shed millions of oocysts over 1-3 weeks. Tissue cysts can remain present for years.</p>
<p>Clinical signs: Infections in felids are usually subclinical, although a transient mild diarrhea may occur. In species sensitive to the disease, animals are often found dead with no clinical signs observed prior to death. If present, clinical signs may vary depending on the organs affected. Reported clinical signs include respiratory signs (dyspnea, tachypnea, coughing), gastrointestinal signs (diarrhea), general signs (depression, anorexia, behavioral changes), lymphadenopathy, muscle weakness, neurologic signs (blindness, ataxia, dysphagia), ocular disease (keratitis, uveitis, chorioretinitis, endophthalmitis, cataracts), and abortion. Serum biochemical abnormalities may include elevated muscle and liver enzymes.</p>
<p>Post mortem, gross, or histologic findings:</p> <p><u>Gross</u>--Affected animals may have no gross lesions. If present, gross lesions may include congestion, hemorrhage, organomegaly, or necrosis of any affected organs.</p> <p><u>Histologic</u>--Multifocal, multi-organ necrosis is often associated with acute toxoplasmosis. Focal necrosis of affected organs may be associated with free and intracellular tachyzoites (2µm x 6µm crescent-shaped structures with pointed anterior and rounded posterior). Brain (encephalitis with microglial nodules and perivascular cuffing), myocardium (myocarditis), and lung (interstitial pneumonia) are frequently affected. Tissue cysts measuring 5-100µm in diameter can be found in any tissue, but frequently occur in the brain, eye, and muscle. Cysts have thin (<0.5µm) elastic walls and contain up to hundreds of 7µm x 1.5µm crescent-shaped bradyzoites.</p>
<p>Diagnosis: Definitive diagnosis can be achieved by observation of tachyzoites or bradyzoites in affected tissues with cytology or histopathology. Multiple serologic testing modalities capable of detecting IgG and IgM antibodies are available including ELISA, Western blot, direct agglutination test (DAT), modified agglutination test (MAT), latex agglutination test (LAT), and indirect hemagglutination test (IHAT). A single positive serologic test indicates exposure to the organism. In young animals, transfer of maternal antibodies can produce positive serology results. Active infections are generally characterized by a high positive IgM titer with subsequent seroconversion and development of an IgG antibody titer > 32 or by a 4-fold increase in paired IgG titers taken 2-4 weeks apart. PCR and immunohistochemical staining can also be used to detect <i>Toxoplasma</i> antigen in tissues.</p>
<p>Material required for laboratory analysis: Formalin-fixed affected tissues can be used for histopathology and immunohistochemical staining. Fresh and frozen tissue can be used for PCR. DNA denatures in formalin so PCR becomes less accurate in tissues that have been fixated in formalin for long periods. Serum is needed for the serologic tests. Aqueous humour, cerebrospinal fluid, and plasma can also be assayed for IgG antibodies by the MAT.</p>
<p>Relevant diagnostic laboratories:</p> <p>Clinical Parasitology Diagnostic Service Laboratory (immunoassay by MAT) Room A233 University of Tennessee College of Veterinary Medicine 2407 River Drive Knoxville, TN 37996-4500 865-974-5645 parasitology@utk.edu</p>

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Treatment: Atovaquone has shown the most promise in treating toxoplasmosis in multiple species. Administration with a high fat meal (e.g. canola oil) has been suggested to increase absorption of the drug. However, the efficacy of this practice is unknown for foregut fermenters. Other drugs including sulfa drugs, clindamycin, spiramycin, ponazuril, and pyrimethamine have also been used alone or in combination with variable success. General supportive care is also usually needed for active cases of toxoplasmosis.

Prevention and control: Controlling exposure to cat feces is an important part of toxoplasmosis prevention. Feral cats are a common source of infective oocysts in the environment. Contamination of food and bedding materials with cat feces may be a source of infection in situations where felids are not known to be present near the affected animal. A live attenuated vaccine has been developed for livestock, but efficacy is variable in other species. Meat containing tissue cysts can be rendered non-infective by cooking to 67 °C or freezing to -12 °C for at least 24 hours. Control of intermediate hosts (e.g. rodents) in the environment can help prevent transmission to carnivores. Prophylactic treatment of queens or kittens has been recommended to reduce morbidity and mortality in Pallas' cats.

Suggested disinfectant for housing facilities: *Toxoplasma* is resistant to most disinfectants but is usually susceptible to boiling water, formalin, and iodine.

Notification: Not a reportable disease.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: Animals introduced to the environment of an infected felid are at risk of contracting toxoplasmosis. Non-felid species that are infected with toxoplasmosis do not pose a risk to other individuals in the environment unless their tissue is ingested. Vertical transmission between females and their offspring is possible in all mammalian species when the infection occurs during gestation.

Conditions for restoring disease-free status after an outbreak: Once an individual becomes infected with toxoplasmosis, it can remain infected for life. *Toxoplasma* organisms can remain dormant in tissue cysts where they are protected from the host's immune response. Episodes of immunosuppression can result in a recrudescence of clinical disease. Serologic testing and removal of positive individuals is a possible way of reaching disease-free status provided that there is not continued exposure to infective oocysts in the environment.

Experts who may be consulted:

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TRICHOSTRONGYLOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Birds; bovids; camels; cervids; equids; giraffe; suidae; rabbits; rodents; primates	Fecal-oral with a direct life cycle	Heavy burdens cause weight loss, lethargy, anorexia, watery diarrhea, weakness, anemia, and death	Low-level infections are usually asymptomatic. Young animals more severely affected	Benzimidazoles or macrocyclic lactones. Alternatives to anthelmintics have been investigated in artiodactylids including cooper oxide wire particles and nematophagous fungus (environmental control)	Pasture rotation. Consider strategic and evidence-based treatment using in vitro sensitivity testing with anthelmintic usage. Immunity develops as animals age	Some species are zoonotic

Fact Sheet compiled by: Christopher S. Hanley

Sheet completed on: 29 December 2010; updated 1 April 2013; updated 8 February 2018

Fact Sheet Reviewed by: Deidre Fontenot

Susceptible animal groups: Birds, bovids, camelids, cervids, equids, giraffe, suidae, rabbits, rodents, and, as accidental hosts, primates

Causative organism: Any of the 35+ species of nematodes of the genus *Trichostrongylus*

Zoonotic potential: Yes, although not all species are zoonotic. Most human infections are asymptomatic or associated with mild clinical signs as all primates are accidental hosts. Abdominal pain, rashes, nausea, diarrhea, anorexia, flatulence, dizziness, generalized fatigue, and malaise all possible.

Distribution: Worldwide

Incubation period: Under ideal conditions, the third stage infective larvae develop within 5-10 days. Depending on the species of *Trichostrongylus*, prepatency is generally 15-25 days but can be delayed for prolonged periods. Some species of *Trichostrongylus* (*T. colubriformis*, *T. tenuis*) can undergo winter arrest in certain geographic areas. Soil moisture, climate warming, and pasture loads can all play a role in the incubation and infectivity.

Clinical signs: Most infections are asymptomatic or only have mild signs. Weakness and death can occur with heavy worm burdens, especially in young animals. Wasting, black or watery diarrhea, depression, anorexia, swollen mucosa, eosinophilia, and anemia can all occur, especially in chronic infections. Birds may have the above as well as decreased egg production. Because of the great variability in host, and organ invaded each species of *Trichostrongylus* must be evaluated in the specific circumstance of presentation

Post mortem, gross, or histologic findings: Depending on the species of *Trichostrongylus* adult worms may be seen in the small intestine, abomasum of ruminants, stomach of monogastrics or ceca of birds. They are very fine parasites and if they are removed from the organs and placed against a dark background, they look like small hairs. Mucosal congestion, inflammation, and thickening may be present. Gastric infection may produce an edematous stomach or abomasum. Histologically, villus atrophy, enterocyte destruction, mucosal ulceration, capillary erosion, blood loss, mucosal edema, fibrinonecrotic membranes, and secondary bacterial infection may be seen. Gastric infection may produce gastric edema and hyperplasia.

Diagnosis: Eggs can be found on fecal parasite evaluation or parasites can be identified on histologic evaluation of the gastrointestinal tract.

Material required for laboratory analysis: Feces or adult worms

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Relevant diagnostic laboratories: Any laboratory that can provide endoparasite identification and quantification. Eggs of *Trichostrongylus* spp. cannot be reliably differentiated from those of most other Trichostrongyloidea or Strongyloidea. Egg quantification (fecal egg count via McMasters technique) is recommended in hoofstock species to determine whether treatment is warranted.

Treatment: Benzimidazoles or macrocyclic lactones. Alternatives to anthelmintics have been investigated in artiodactylids including cooper oxide wire particles and nematophagous fungus (environmental control).

Prevention and control: Proper sanitation, pasture rotation, strategic and evidence based anthelmintic treatment using in vitro sensitivity testing. Immunity with age develops in some species although this has not been proven in hoofstock species.

Suggested disinfectant for housing facilities: None

Notification: None

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: In artiodactylids, fecal egg counts are recommended before releasing new animals on pasture to determine current level of infection prior to introduction. Pasture infection levels can also be considered (pasture larval counts) as well as current infection levels in animals to make risk/ benefit assessment for treatment prior to pasture introduction. . *In vitro* sensitivities can be performed to determine level of resistance to classes of anthelmintics prior to treatment. If *in vitro* sensitivity testing cannot be performed, then treatment of newly infected animals with a cocktail of anthelmintics with at least two drugs in different families at full dose using accurate animal body weights may eliminate previously acquired anthelmintic resistant worms.

Conditions for restoring disease-free status after an outbreak: Directed treatment at clinically affected animals by employing quantitative fecal parasitology (McMasters counts). Low levels of parasitism are common and may aid in the development of immunity.

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References:

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CAPILLARIASIS/TRICURIASIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Wide range of vertebrates	Fecal/oral transmission via transmission of eggs with infective L2 Some capillarids use earthworms or fish as intermediate hosts	Weight loss, diarrhea	High morbidity, but low mortality	Fenbendazole and other benzimidazoles, milbemycin oxime	Sanitation; eggs are very resistant. Do not consume undercooked fish.	Yes, some species of Trichuris, Capillaria hepaticum, and C. philippinensis

Fact sheet compiled by: Janna Wynne

Sheet completed on: 15 March 2018

Fact Sheet Reviewed by: Michael McBride

Susceptible animal groups: Mammals, birds, and reptiles.

Causative organism: Capillarids of many genera (e.g. Capillaria, Eucleus, Calodium, Pearsonema) occur in mammals, birds, reptiles, amphibians, and fish. Trichuris spp. occurs in mammals, primates, ruminants, carnivores, suids, and rodents.

Zoonotic potential: Yes, some species of Trichuris, Capillaria hepatica and C. philippinensis.

Distribution: Worldwide, although parasites of concern will vary by location and species.

Incubation period: Variable, but tends to be longer than many other parasites. Prepatent period is three months in many intestinal Trichuris.

Clinical signs: Clinical cases and fatal disease are rare. Many low parasite burdens can be asymptomatic, and infections are frequently asymptomatic in hoofed stock. In clinical animals, weight loss, colitis, diarrhea, hematochezia or melena can be present. Capillaria tend to infect airways, nasal cavity, or the urinary bladder such as air sacculitis or pneumonia from Eucolies spp. Or Pearsonema spp. In the urinary system. Capillaria hepaticum causes hepatic cirrhosis and C philippenensis is found in the tissues of fish, causing an intestinal infection when affected raw fish are consumed.

Post Mortem, gross, or histologic findings: Trichuris spp. Can be observed embedded in the wall of the colon of carnivores and ungulates, and found in the neutral pH forestomach of some leaf-eating monkeys (e.g. Colobus). Capillaria hepatica induces cirrhosis of the liver. It is usually identified on post mortem. Other capillarids can cause nasal, bronchial, intestinal, hepatic, and urinary infections, and findings will vary according to parasitic and host species and site of parasitism.

Diagnosis: Centrifugation fecal floatation can be performed for identification of infection. However. The eggs (bipolar plugs) are very dense and require correct floatation solution – good choice is Sheather’s with specific gravity of 1.27 – and centrifugation to recover them. Eggs are shed intermittently, so repeated fecals may be necessary. For pulmonary species, BAL, and for urinary tract infections, urine sedimentation. Ova of different capillarids that infect the same host (e.g. carnivores) can be distinguished by structure of patterns of egg case. Biopsies of affected areas – gastrointestinal tract or liver – can be used.

Material required for laboratory analysis: Fecal sample, colon or gastric biopsy, hepatic biopsy, BAL

Relevant diagnostic laboratories: These diagnostics are readily available, as in-house fecal floatation or any laboratory performing fecal exams or histopathology.

Treatment: Fenbendazole and other benzimidazoles, milbemycin oxime, and pyrantel pamoate can be used. Variable sensitivity to ivermectin has been noted. Due to its long prepatent period, it is appropriate to treat monthly for 3 treatments.

Prevention and control: Quarantine measure and treatment before introduction is best. Chronic treatment may be required. Environmental control and preventing recontamination are critical.

Suggested disinfectant for housing facilities: Eggs are very resistant to destruction and may remain infective in the soil for long periods of time. Remove fecal material promptly from enclosures. Dirt floored

CAPILLARIASIS/TRICURIASIS

enclosures are almost impossible to disinfect. Dig out dirt or use fire to sterilize.
Notifications: None
Measures required under the Animal Disease Surveillance Plan: None
Measures required for introducing animals to infected animal: Many facilities manage chronically infected groups with varying levels of problems. Many use chronic anthelmintic treatment.
Conditions for restoring disease-free status after an outbreak: Treat for a minimum of 3-4 months. Clear animals while held in cement floored facility before introducing to a clean group in a clean environment. Continue long term monthly fecal screening and environmental sanitation.
Experts who may be consulted:
<p>References:</p> <ol style="list-style-type: none"> 1. Centers for Disease Control and Prevention [Internet]. Parasites – Capillariasis (also known as Capillaria Infection); 2018 [cited on 2018 March 15]. Available from http://www.cdc.gov/parasites/capillaria/ 2. Centers for Disease Control and Prevention [Internet]. Parasites – Trichuriasis (also known as Whipworm Infection); 2013 [cited on 2018 March 15]. Available from http://www.cdc.gov/parasites/whipworm/ 3. Bowman DD. Helminths. In: Bowman DD, Lynn RC, Eberhard ML (eds.). <i>Georgis' Parasitology for Veterinarians</i>, 8th ed. St. Louis (MO): W.B. Saunders; 2003. p. 228-231. 4. Fuehrer H, Igel P, Auer H. Capillaria hepatica in man and an overview of hepatic capillariosis and spurious infections. <i>Parasitol Res.</i> 2011;109(4):969-979. 5. Goncalves AQ, Ascaso C, Santos I. Calodium hepaticum: Household Clustering Transmission and the Finding of a Source of Human Spurious infection in a Community of the Amazon Region. <i>PLoS Negl Trop Dis.</i> 2012;6(12):e1943. 6. Limsrivilai J, Pongprasobchai S, Apisarnthanarak P. Intestinal capillariasis in the 21st century: clinical presentations and the role of endoscopy and imaging. <i>BMC Gastroenterol.</i> 2014;12:207. 7. Liu GH, Gasser RB, Nejsun P, Wang Y, Chen Q, Song HQ, Zhu XQ. Mitochondrial and nuclear ribosomal DNA evidence supports the existence of a new Trichuris species in the endangered Francois' leaf-monkey. <i>PLoS One.</i> 2013;8(6):e66249. 8. Ravasi DF, O'Riain M, Davids F, Illing N. Phylogenetic evidence that two distinct Trichuris genotypes infect both humans and non-human primates. <i>PLoS one.</i> 2012;7(8):e44187. 9. Stephenson LS, Holland CV, Cooper ES. 2000. The public health significance of Trichuris trichiura, <i>Parasitol.</i> 2000;121(S1):S73-S95. 10. Stidworthy MF, Lewis JC, Masters NJ, Boardman S. Capillaria hepatica in primates in zoological collections in the British Isles. <i>Vet Rec.</i> 2009;164(2):66. 11. Wynne J, Garner M. Elimination of trichuriasis in a group of colobus monkeys (Colobus quereza). In: <i>Proc Am Assoc Zoo Vet.</i> 2004. p. 202-203.

TRYPANOSOMA CRUZI - CHAGAS DISEASE

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p>All mammals are susceptible to infection.</p> <p>Wildlife reservoirs include woodrats, opossums, armadillos, and raccoons.</p> <p>Disease is reported in dogs, humans, and non-human primates.</p>	<p>Contamination of blood-feeding lesion or mucous membrane by feces of insect vector; ingestion of infected vector, or food or water contaminated with bug feces; trans-placental or trans-mammary; blood transfusion</p>	<p>Dogs and humans-range from asymptomatic to acute myocarditis and sudden death. Chronic disease signs are related to cardio-myopathy; and in humans, mega-esophagus and/or megacolon can be seen.</p>	<p>Dogs and humans-variable; can cause severe disease or death.</p> <p>The degree to which wildlife reservoirs present disease is unknown.</p>	<p>No FDA approved treatment is available.</p>	<p>Prevent exposure to vectors; control vector populations; minimize wildlife reservoir access; blood donor screening; prevent seropositive female dogs from breeding.</p>	<p>Yes.</p>

Fact Sheet compiled by: Sarah A. Hamer

Sheet completed on: 4 September 2013

Fact Sheet Reviewed by: Tom Sidwa; Susan Montgomery

Susceptible animal groups: All mammal species are considered to be susceptible to *Trypanosoma cruzi*, including more than 150 species of 24 families that have been reported to be infected. Disease is best described from humans and dogs; the degree to which other domestic or wild animals present disease upon infection is unknown

Causative organism: *Trypanosoma cruzi* is a flagellated protozoan parasite that maintains many life stages. The parasite is spread by triatomine bugs. Triatomines are blood-sucking vectors commonly referred to as kissing bugs or cone-nosed bugs. After ingesting trypomastigotes from the blood of a vertebrate host, the bug's hindgut contains epimastigotes which also can multiply in the vector. Metacyclic trypomastigotes appear in the insect's rectum 8-10 days after infection. These metacyclic forms pass in the feces and can enter the body of a vertebrate host through the bite, scratched skin, or mucous membranes. Trypomastigotes are the abundant blood form that circulates in the mammalian host after infection. Amastigotes develop in muscle and other tissue cells and multiply by binary fission. Amastigotes differentiate into to trypomastigotes which lyse the host cell and burst free and this stage can then attack other host cells. Pseudocysts of parasites may form in muscle cells.

Zoonotic potential: Many kinds of wild and domestic mammals serve as reservoirs for *T. cruzi*. This parasite can be bridged to humans from mammalian reservoirs through kissing bug vectors. Zoonotic potential is high in areas of Mexico and South and Central America, where kissing bugs maintain peridomestic cycles and colonize human dwellings. In contrast, the housing structures in US are generally less able to be colonized by bugs, and therefore zoonotic potential is reduced relative to areas with peridomestic cycles.

TRYPANOSOMA CRUZI - CHAGAS DISEASE

Distribution: Chagas disease in humans or animals can occur wherever there is overlap among kissing bug vectors, the *T. cruzi* parasite, and vertebrate reservoir hosts. The disease is endemic in many areas of Mexico, South and Central America, and is increasingly recognized across the southern US. In the US, 11 species of kissing bugs occur, and are distributed across the southern half of the country and range as far north as the California/Oregon border and New Jersey. In Latin America, an estimated 12-19 million people were infected in the early 1990s, with an annual incidence exceeding 500,000. Since then, control campaigns have assisted in reducing the disease burden. The disease burden in the US is largely unknown due to lack of awareness, testing, and reporting. However, CDC has estimated that more than 300,000 cases of Chagas disease are found in US among immigrants from endemic countries of Latin America. The American Association of Blood Banks maintains The Chagas Biovigilance Network for reporting of screening and confirmatory results from the testing of US blood donors for antibodies to *T. cruzi*.

Incubation period: Once the metacyclic trypomastigotes enter the host, an acute local inflammatory reaction may occur. In humans, within 1-2 weeks of infection, the parasites spread to lymph nodes and multiply within phagocytic cells. The intracellular amastigotes multiply and pseudocysts may form. Within days, some organisms may transform to trypomastigotes and burst free from the pseudocyst. A generalized parasitemia can occur, followed by parasite invasion of many tissues within body. The incubation period may be up to several months if contaminated blood from transfusion is the source of infection.

Clinical signs: Chagas disease manifests as acute and chronic phases; in the absence of treatment, the host is infected for life. The chronic phase of infection has two forms, an indeterminate form during which the host is asymptomatic followed by development of clinical disease years to decades later. In humans and dogs, the initial acute phase of infection is usually asymptomatic or undetected; regional or generalized lymphadenopathy, fever, myalgia, headache, hepatosplenomegaly, edema, rash, vomiting, diarrhea, or anorexia may occur. Humans may note a lesion (chagoma) where the parasite enters the body. Severe manifestations, such as acute myocarditis or meningoencephalitis are rare.

Chronic phase of disease may develop in a subset of human patients who survive the acute phase of infection. In chronic disease, cardiac abnormalities may be noted including right bundle branch block and left anterior hemiblock, atrio-ventricular conduction abnormalities, and arrhythmias. Megacardia may be noted on radiographs. In humans and dogs, systolic dysfunction is indistinguishable from dilated cardiomyopathy. Weakness and exercise intolerance may be noted. Humans with Chagas disease may also have complications of the digestive system, including megaesophagus and megacolon, with or without cardiac manifestations.

Post mortem, gross, or histologic findings: Gross cardiac changes may include megacardia and focal thinning of the myocardium including apical aneurysm. Dilatation and thinning of the wall of the esophagus and colon may occur. Histologically, in canines, examination of the heart may reveal unruptured pseudocysts with no inflammatory response, or ruptured pseudocysts with characteristic infiltration of lymphocytes, monocytes, and/or polymorphonuclear leukocytes.

Diagnosis: During acute infections, the trypomastigotes (blood stage of the parasite) may be identified by microscopy of a peripheral blood sample or through culture techniques; the organism has a single flagellum and a large kinetoplast at the posterior end of the cell and appears as a characteristic 'C' shape in Giemsa stains of bloodsmears. Additionally, PCR can be used to amplify the DNA of the parasite from a blood sample. Serologic tests may be of limited utility during acute infections. Because the level of circulating parasites decreases within months, parasites are undetectable in blood by most methods during the chronic phase of disease.

During chronic disease, serologic tests are used to detect antibodies to the parasite. To increase sensitivity and specificity, a standard serodiagnostic approach is to apply two or more tests that use different techniques or different antigens. Two commonly used techniques are enzyme-linked immunosorbent assay (ELISA) and immunofluorescent antibody test (IFA). Some serological tests are cross-reactive and will also detect

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antibodies to *Leishmania* species. Rapid immunochromatographic ‘dipstick’ assays have been developed for the detection of antibodies to *T. cruzi* in humans and dogs. While sensitivity and specificity meet or exceed the characteristics of other available tests, their use for Chagas disease diagnosis is considered experimental. Two tests are FDA approved for use as screening tests for human blood donations; most samples that screen positive are then subjected to a supplemental test with greater specificity. Blood donors who screen positive are notified of results, are urged to contact their physician, and are no longer able to donate blood. In chronic disease, particular ECG abnormalities combined with positive serology results can be highly indicative. The only parasitological techniques currently considered useful in the chronic phase of disease would be xenodiagnoses and hemoculture although it is no longer used in human diagnostics. In humans, PCR and IHC also are used and PCR would be considered more sensitive. Postmortem, heart or other tissues may be examined using histopathology for the amastigotes (tissue stage of the parasite) and associated inflammation.

Material required for laboratory analysis: Whole blood, plasma, serum, and/or cardiac tissue.

Relevant diagnostic laboratories:

Texas A&M Veterinary Medical Diagnostic Laboratory
 PO Box Drawer 3040
 College Station, TX 77841-3040
 (979) 845-3414
 (888) 646-5623
<http://tvmdl.tamu.edu/>

T. cruzi rapid immunoblot assay
 Primate Diagnostic Services Laboratory (PDSL)
 Washington National Primate Research Center
 University of Washington
 Seattle Washington 98195-7330
diagnostic@wanprc.org
<http://www.wanprc.org/pdsl/>

Treatment: Although two antiparasitics can be used to treat human patients with Chagas disease (nifurtimox and benznidazole), these drugs are not approved by FDA so in the US, they are available only from CDC under investigational protocols. For both drugs, side effects are fairly common, and contraindications for treatment include severe hepatic disease and renal disease. However, antiparasitic treatment is indicated for all cases of congenital, acute or reactivated Chagas disease and for chronic *T. cruzi* infection in children. Treatment is recommended for adults up to 50 years old with chronic infection who do not already have advanced Chagas cardiomyopathy. For adults older than 50 years with chronic *T. cruzi* infection, the decision to treat with antiparasitic drugs should be individualized.

Prevention and control: In the absence of a human or veterinary vaccine and given the limited treatment options, prevention and control of Chagas disease across the Americas relies heavily on vector control and community education. Improvement of housing structures combined with insecticide treatment inside homes has significantly reduced peridomestic transmission of the *T. cruzi* parasite in Central and South America. To reduce the attraction of kissing bugs to homes or kennels, outdoor lights should be eliminated, and rodent habitat immediately surrounding the home or kennel should be removed. Screening of blood donations is an important public health tool for prevention of disease transmission through blood transfusion. Early detection and treatment of acute disease, including congenital cases, can reduce the burden of disease.

Suggested disinfectant for housing facilities: The duration of time the parasite can live outside a vector or host on environmental surfaces contaminated by bug feces is unknown, but the parasite will be destroyed by direct exposure to sunlight and other harsh environments. Surfaces that have come in contact with bugs or

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bug feces should be disinfected using 10% bleach or 70% ethanol.

Notification: States are not required by federal law to report cases of Chagas disease. However, Chagas disease in humans is reportable in 4 states: Arizona, Massachusetts, Tennessee, and Texas. Chagas disease in animals is reportable in Texas.

Measures required under the Animal Disease Surveillance Plan: N/A

Measures required for introducing animals to infected animal: The risk of animal to animal direct transmission in the absence of the kissing bug vector is minimal. However, infected animals may increase the infection prevalence in vectors in a local environment. Efforts should be made to prevent seropositive female dogs from breeding due to congenital transmission.

Conditions for restoring disease-free status after an outbreak: N/A

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
All warm-blooded animals	<p>Arthropod vectors such as ticks, biting flies, and, in some areas, mosquitoes.</p> <p>Inhalation of aerosolized infectious material</p> <p>Ingestion of contaminated food or water</p> <p>Direct transmission: Skinning dead infected animals; contaminated water</p>	<p>Depends on route of infection; general: lethargy, anorexia, pyrexia</p> <p>Transdermal exposure: ulcer at site of inoculation and swollen glands or (rarely) swollen glands without ulcer; lymphadenopathy</p> <p>Oculoglandular: conjunctivitis and lymphadenopathy</p> <p>Oral exposure: lymph-adenopathy</p> <p>Inhalation: pneumonia, coughing</p>	<p>Clinical signs vary from mild to severe, depending on route of exposure, and death can result if untreated.</p> <p>Pneumonic form: severe.</p> <p>Septicemia often death occurs without prior signs</p>	Antibiotics: streptomycin, gentamicin, tetracyclines, ciprofloxacin	<p>Rodent, lagomorph, mosquito, biting fly and tick control; Sanitation (including use of gloves and masks); avoid ingestion and contact with untreated water; avoid ingestion of uncooked meat and rodent carcasses</p>	High zoonotic potential

Fact Sheet compiled by: Cornelia J. Ketz-Riley

Sheet Revised on: 2 February 2018

Fact Sheet Reviewed by: David Miller

Susceptible animal groups: Natural infection in mammals and birds.

Causative organism: *Francisella tularensis* - four subspecies: most commonly associated with disease outbreaks are *F. tularensis* subsp *tularensis* (type A)(associated with cottontail rabbit, ticks, biting flies) and *F. tularensis* subsp *holarctica* (type B)(associated with muskrat and beaver), while *F. tularensis* subsp *mediasiatica* and *novicida* are rarely associated with severe infections.

Type A is regarded as a category A biowarfare/bioterrorism agent 3 because of the diversity of its route of transmission, ease of dissemination (especially the aerosol route), high infectivity, and potentially high mortality rate.

Type A and B can be distinguished by the ability of type A to ferment glycerol and polymerase chain reaction test (PCR).

Zoonotic potential: The zoonotic potential is very high, with inhalation of only 10-50 organisms needed to cause severe infection.

Distribution: Throughout the Northern hemisphere, this disease represents one of the largest host distributions of any zoonotic disease. Type A only occurs in North America, whereas type B found throughout Northern hemisphere. In North America, geographic overlap of both subspecies is present, although type A associated with highest disease incidence and mortality rate.

Changes involving climate and animal, as well as vector distribution, seem to cause emergence or re-emergence in areas considered non-critical for appearance of *Francisella tularensis*. Flooding may be associated with increased transmission. Arthropods, such as ticks, mosquitoes and biting flies, are common vectors associated with transmission of *F. tularensis*. While ticks are believed to be the primary biological vectors, transmission by mosquitoes and biting flies is believed to be mostly mechanical through their

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mouthparts. Infection of a patient through a ringtail possum in Tasmania, Australia, indicated the emergence of *F. tularensis* type B in the Southern hemisphere.

Incubation period: generally 3-5 days, but 1-14 days possible

Clinical signs: Clinical presentation of tularemia varies with the route of infection. First development: non-specific signs such as depression, lethargy, anorexia, vomiting, diarrhea, marked pyrexia, or peracute death without prior clinical signs; Clinical disease in humans includes forms of ulceroglandular, glandular, oculoglandular, oropharyngeal, pneumonic, and typhoidal disease. First three forms occur via local infection through arthropod bites, injuries, or mechanical transfer involving skin and lymphoid tissue, and result in local or even generalized lymphadenopathy. Skin ulcers may form at the site of dermal infection. Oropharyngeal form - ingestion of contaminated food or water involving the tonsils and retropharyngeal lymph nodes. Pneumonic form as most severe clinical form of tularemia, leading to mortality if untreated that results from direct inhalation of organisms from infected tissue. Typhoidal form - systemic disease: high fever, but without lymphadenitis or cutaneous lesions. All forms can develop into secondary septicemia, pleuropneumonia, and meningitis.

F. tularensis is usually invading and replicating in vector-derived cells and hemolymph, and in macrophages within the host. Cytokines, such as interferon-gamma and tumor necrosis factor, produced by T-cells are critical for activation of macrophages and cell-mediated and protective immunity. Yet, *F. tularensis* is able to proliferate in macrophages without destroying the host cell. It also has developed good survival and adaptation strategies using surface proteins to suppress innate immune response, which makes it harder to diagnose and control it within the host. New research has discovered that *F. tularensis* is also able to invade erythrocytes. The high hemoglobin and iron content in erythrocytes could influence the virulence gene expression in *F. tularensis*. Yet, erythrocytes do not support replication of the pathogen and, therefore, do not seem to be a major contributor to the pathogenesis of tularemia.

Post mortem, gross, or histologic findings: Gross: congested organs - mostly lungs, lymph nodes, spleen, liver - with multiple light tan miliary foci on the surface, as well as in the parenchyma. Histopathology: pyogranulomatous lymphadenitis, tonsillitis, splenitis, hepatitis and pneumonia with necrotic foci.

Diagnosis: Although culture is considered the “gold standard” diagnostic tool to confirm tularemia, recovery of live organisms of *F. tularensis* from carcasses can pose a challenge. The bacterium is very slow growing and has special biochemical needs so poor competitive characteristics in the presence of other bacterial pathogens. Selective antibiotic media (CHAB-A) are needed for isolating the bacteria from contaminating environmental flora in carcasses; Western blot and microagglutination assay demonstrate the highest level of sensitivity and specificity for *F. tularensis*, higher than enzyme-linked immunosorbent assay (ELISA) or indirect immunofluorescence (IFA). A combination of at least two serological tests, such as ELISA and Western blot, was demonstrated to be a suitable diagnostic tool for laboratory confirmation of both individual cases, and larger epidemiological studies. Immunohistochemistry (IHC) has been successfully used for post mortem diagnosis in formalin-fixed tissue.

To detect serologic titers in live animals or humans, besides microagglutination, latex or tube agglutination, a novel competitive ELISA test, can be recommended. Real-time PCR, Multiplex qPCR, 16S rDNA sequencing, and molecular subtyping using differential insertion sequence amplification and regions of differences (RD), can be especially useful for samples where organisms are non-culturable or nonviable. Serology is often difficult as short-term diagnosis due to low antigenicity of the organism. Repeated serology is necessary for evaluation of titer development. Although some commercially available serologic tests are available showing good results, these should be interpreted cautiously because of the quick onset of clinical signs as compared to the development of humoral response; clinically silent cases have been reported; and antibodies in humans can persist for years. A recombinase PCR amplification assay has been developed for rapid detection of *F. tularensis*. Molecular tests provide a safer diagnostic tool, while avoiding hazardous multiplying of the pathogen. However, cultivation of *F. tularensis* will still be required for evaluation of antibiotic resistance patterns, molecular epidemiological and pathological analysis of the pathogen. Investigation into molecular level of host macrophage survival and innate immune response to

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infection with *F. tularensis* enabled the identification of newer tools for diagnosis of and immunologic prevention of tularemia in laboratory animals and humans.

Material required for laboratory analysis: The best result is achieved by immediate culturing of fresh tissue, or by immediate freezing of tissue specimens from carcasses for subsequent culture. Blood samples are often used to confirm serologic titers in live animals or humans. Molecular tests can nowadays be used for testing of any type of tissue.

Relevant diagnostic laboratories: The contagious nature of *F. tularensis* poses an additional challenge to laboratory personnel, high biohazardous risk of infection via inhalation of aerosolized bacteria. Testing for tularemia demands a laboratory setting with a minimum biological safety level 2 (BSL-2), and testing procedures performed according to BSL-3 regulations

Confirmation of results are suggested in Centers for Disease Control and Prevention, National Center for Zoonotic, Vector-Borne, and Enteric Diseases, Division of Vector-Borne Infectious Diseases, Bacterial Diseases Branch, Foothills Campus, Fort Collins, Colorado, 80522, USA

Treatment: Streptomycin is considered the treatment of choice in humans with tularemia. Other chemotherapeutics, such as gentamicin, tetracyclines, chloramphenicol, and fluoroquinolones, have been used successfully. Tetracyclines and chloramphenicol are bacteriostatic and require a longer treatment period of at least 14-21 days. Due to frequent treatment relapses or failure, combined with increase in resistance against commonly used products, alternative chemotherapeutics and newer chemotherapeutic generations, such as glycoacyclines, ketolides, and new generation fluoroquinolones are currently investigated and show promising results. Cystatin 9, a type 2 cysteine protease inhibitor with immunomodulatory properties has shown to help develop effective protection against *F. tularensis*, in vitro and in vivo, and may become a future treatment tool against tularemia.

Prevention and control: There are still no approved vaccine products available in the USA. A live attenuated vaccine strain of *F. tularensis* type B was developed in the Soviet Union for immunization of humans. Although this live vaccine serum (LVS) strain was also shown to be effective against the type A strain and oral infection, this vaccine was not fully effective against infection acquired by inhalation. Currently, newer LVS vaccine affords no better efficient protection against an aerosolization challenge by *F. tularensis*. Subunit or recombinant vaccines have been more recently researched, but any results did not show better prevention efficacy than the LVS. DNA Gold Micronanoplex used for genetic immunization seems to produce efficient levels of antibodies against *F. tularensis*. Ongoing trials were completed to develop vaccine using mutant strains or nonpathogenic *F. novicida* strain, but they have not shown improved protection efficacy over the LVS, either. Some research is concentrating on virus-vectored vaccine for better stimulation of immunity in presence of *F. tularensis*. Newest research is focusing on adding epitope to the immunogenic products to stimulate high avidity of CD4+ and CD8+ T-cells, as well as using adjuvants to help stimulate higher antibody titers against *F. tularensis*, to increase efficacy of any existing LVS. Monophosphoryl Lipid A has already been approved by the FDA as adjuvants for other vaccine. One study identified nitric oxide as a predictor of vaccine efficacy, which also has already been used in connection with other diseases. Good pest control is the best defense against development of *F. tularensis* carrying population on zoo grounds.

Suggested disinfectant for housing facilities: Diluted hypochlorite, quaternary ammonium disinfectants or any other ordinary medical disinfectants are useful. *F. tularensis* can be inactivated by heat, at least at 60 °C for 20 min.

Notification: Reportable disease at a variety of levels – city, county, state, and federal as *F. tularensis* is considered a Category A Bioterrorism agent.

Measures required under the Animal Disease Surveillance Plan: Continuous surveillance of wildlife and vector populations, as well as first-level emergency response plan after detection of tularemia cases are important to prevent or minimize outbreaks in animals and humans.

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Measures required for introducing animals to infected animal: Regular quarantine in a clean environment; reduce access to potential vectors, and host animals.

Conditions for restoring disease-free status after an outbreak: Pest and vector control are necessary to minimize exposure. Constant pathogen surveillance of wildlife populations is strongly recommended.

Experts who may be consulted:

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ULCERATIVE STOMATITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Reptiles	Opportunistic-normal flora of the oral cavity or present in the environment, but also seen with some primary pathogens such as herpesvirus	Anorexia, dysphagia, ptyalism, periodontal disease, ulceration of mucous membranes with caseous exudate, pneumonia, osteomyelitis	Severe cases can result in septicemia and death	Debridement, irrigation with antimicrobial solution, topical ointment, analgesia, and long term antibiotics or antifungal based on culture and sensitivity testing	Appropriate nutrition, hygiene, and temperature; minimize stress; prevent trauma to oral cavity	Some associated agents can be human pathogens

Fact Sheet compiled by: Genevieve Vega Weaver

Sheet completed on: 27 January 2011; updated 1 October 2012; 15 December 2017

Fact Sheet Reviewed by: Charles Innis

Susceptible animal groups: Mostly reptiles, especially snakes, chelonians, and some groups of lizards such as chameleons, bearded dragons, and monitors

Causative organisms: Gram-negative bacteria are most commonly implicated, but there are multiple possible causative agents including various aerobic and anaerobic bacteria, viruses, and fungi. **Bacteria:** *Aeromonas*, *Pseudomonas*, *Escherichia coli*, *Morganella*, *Proteus*, *Vibrio alginolyticus*, *Providencia*, *Salmonella*, *Corynebacterium*, *Flavobacterium*, *Citrobacter freundii*, *Acinetobacter*, *Micrococcus*, *Aureobacterium*, Beta-hemolytic *Staphylococcus*, *Streptococcus* group C, *Enterobacter*, *Klebsiella*, *Pasteurella*, *Bacteroides*, *Clostridium*, *Fusobacterium*, *Peptostreptococcus*, *Mycoplasma*, and *Mycobacterium*;

Virus: herpesvirus, ranavirus, adenovirus

Fungi: *Candida albicans*, *Aspergillus*, *Sporothrix schenkii*, and *Paecilomyces*.

Zoonotic potential: Yes. Immunocompromised individuals and young children are most at risk. *Aeromonas* can cause enteric disease in humans. *Pseudomonas* can cause urinary tract, respiratory tract, soft tissue, bone, joint, and gastrointestinal disease. *Salmonella*, *E. coli*, *Klebsiella*, *Enterobacter*, *Pasteurella*, *Corynebacterium*, *Mycobacterium*, *Vibrio*, *Staphylococcus*, and *Streptococcus* also can cause disease in humans.

Distribution: Worldwide in both captive reptiles and injured and immunosuppressed free-living animals.

Incubation period: Weeks to months

Clinical signs: Anorexia, dysphagia, ptyalism, tongue paralysis, gingivitis, ecchymosis, petechiation, loss of teeth, tongue sheath abscesses, ulceration of mucous membranes with caseous material accumulation, and osteomyelitis. In lizards with acrodont dentition (e.g., bearded dragons, water dragons), periodontal disease may be seen more frequently. Infection can spread from the nasolacrimal duct and involve the eyes or can descend the trachea and cause pneumonia. Septicemia and death can result in complicated and untreated cases. An ulcerative stomatitis-obstructive rhinitis-pneumonia disease complex has been reported in sea turtles and tortoises. Differential diagnoses include exposure gingivitis due to nutritional secondary hyperparathyroidism and neoplasia.

Post mortem, gross, or histologic findings: Gross findings: Yellow plaques with a diphtheritic membrane and caseous exudate covering eroded oral mucosa and surrounded by inflamed tissue that bleeds easily.

Histologic findings: Plaques consist of serofibrinous material, pyknotic nuclei, and cellular debris above an ulcerated, degenerated epithelium layer with lymphocytic infiltration and hyperplastic epithelium along the periphery of the ulcer.

ULCERATIVE STOMATITIS

<p>Diagnosis: Aerobic and anaerobic bacterial culture and sensitivity; fungal culture and sensitivity; cytology showing increased heterophils and large numbers of Gram-negative bacteria; acid-fast stain for <i>Mycobacterium</i>; radiographs to determine bone involvement; chemistry profile to detect underlying renal disease.</p>
<p>Material required for laboratory analysis: Culture swab or tissue sample of the affected area. A stab incision culture protocol may be necessary. Histopathology and molecular methods useful for viral identification.</p>
<p>Relevant diagnostic laboratories: Laboratories should be experienced with reptilian tissue and culturing from ectotherms. Samples should be incubated at the standard 37° C and also at 25° C.</p>
<p>Treatment: Periodic debridement possibly under anesthesia, irrigation with dilute antimicrobial solution (e.g. povidone-iodine, chlorhexidine, etc.), topical ointment (e.g. silver sulfadiazine, triple antibiotic, gentamicin/betamethasone, etc.), analgesia, and long term antibiotics (at least 4 weeks) or antifungal therapy (at minimum 4-6 weeks) based on culture and sensitivity testing. Antimicrobials should be given for both aerobic and anaerobic bacteria using doses established by species-specific pharmacokinetic testing, when available. Ensure proper husbandry and a low stress environment. Maintain animals at the high end of their optimal temperature range and also provide heat at night. Address any systemic or metabolic illness. Euthanasia should be considered for animals with non-healing lesions due to <i>Mycobacterium</i> spp. Commonly used drugs include tetracyclines, cephalosporins, trimethoprim-sulfa, aminoglycosides, fluoroquinolones, clindamycin, metronidazole, and chloramphenicol. Oral acyclovir for viral stomatitis at 40-80 mg/kg every 8-24 hours has been used. Laser therapy reduces inflammation and provides pain relief. Non-steroidal anti-inflammatories can be used if animal is well hydrated and does not have underlying renal disease. Supportive therapy with Vitamins A, B-complex, and C can be given to boost the immune system. For anorexic animals, avoid forced feeding of whole prey and instead, administer a puree or slurry via a gastric tube.</p>
<p>Prevention and control: Proper nutrition including adequate vitamin (especially Vitamin A) and mineral supplementation, appropriate temperatures, good hygiene, preventing oral trauma from food or habitat, minimizing stress, clearing mite infestations, and avoiding hibernating recently fed animals.</p>
<p>Suggested disinfectant for housing facilities: 1% sodium hypochlorite for most microbes; vinegar or 2% glutaraldehyde for mycobacteria</p>
<p>Notification: None</p>
<p>Measures required under the Animal Disease Surveillance Plan: None</p>
<p>Measures required for introducing animals to infected animal: Isolate infected animal until lesions are healed. Ensure good hygiene and appropriate husbandry practices. Do not introduce infected animal to immunocompromised animals. Avoid all stress or continued suppression of immune system.</p>
<p>Conditions for restoring disease-free status after an outbreak: Properly disinfect habitat.</p>
<p>Experts who may be consulted: Rob Coke, DVM, DACZM, DABVP (Reptile and Amphibian Practice), Senior Staff Veterinarian San Antonio Zoo (210) 734-7184 x1320 zoosrvet@sazoo-aq.org</p> <p>Dr. Jörg Mayer Associate Professor of Zoological Medicine, College of Veterinary Medicine, University of Georgia mayerj@uga.edu</p>
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American Association of Zoo Veterinarians Infectious Disease Manual
VESICULAR EXANTHEMA OF SWINE/SAN MIGUEL SEA LION VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Swine, various marine mammals.	Direct – contact with infected animal. Indirect - feeding uncooked infected products, fomites.	Pyrexia, anorexia, lameness, vesicles progressing to erosions (coronary bands, snout, lips, oral cavity, teats).	Moderately contagious. Moderate to high morbidity. Very low mortality.	None	Do not feed uncooked fish to swine.	No

Fact Sheet compiled by: Cora Singleton

Sheet completed on: 1 January 2011; updated 31 October 2012; updated 8 August 2018

Fact Sheet Reviewed by: Ryan Colburn

Susceptible animal groups: Swine, cattle, horses, skunk, primates, reptiles, fish, and various marine mammals.

Causative organism: Vesicular exanthema of swine (VES) and San Miguel sea lion virus (SMSV) are caliciviruses in the *Caliciviridae* family.

Zoonotic potential: VES has occasionally been isolated from humans with blisters, however, the virus is not considered to be a serious public health threat.

Distribution: VES has been eradicated worldwide. SMSV is found on Pacific coast of North America. The Opaleye fish is considered the primary host of SMSV.

Incubation period: 1-5 days

Clinical signs: Swine – Pyrexia, anorexia, lameness, vesicles progressing to erosions (coronary bands, snout, lips, oral cavity, teats). Clinically indistinguishable from foot and mouth disease, vesicular stomatitis, Seneca virus A, and swine vesicular disease.

Pinnipeds – Abortion; vesicles progressing to erosions on flippers.

Post mortem, gross, or histologic findings: Vesicles on coronary bands, snout, lips, oral cavity, teats. Hydropic degeneration and edema of stratum spinosum of the affected epidermis, followed by ballooning degeneration of keratinocytes that then float into the vesicular fluid. Stratum basale may be disrupted.

Diagnosis: Virus culture, antigen detection, or serology.

Material required for laboratory analysis: Vesicular fluid, epithelium covering a vesicle, heparinized whole blood, serum, tissues in formalin.

Relevant diagnostic laboratories:

Foreign Animal Disease Diagnostic Laboratory, Plum Island

40550 Route 25 (for packages)

Orient Point, NY 11957

P.O. Box 848 (for letters)

Greenport, NY 11944-0848

(631) 323-3256 Fax: (631) 323-3366

Since vesicular diseases cannot be distinguished clinically, contact the proper authorities prior to sample collection and shipment.

Treatment: No effective treatment. Supportive care and treatment of secondary problems.

Prevention and control: VES is thought to have emerged from feeding uncooked fish and marine mammal tissues containing SMSV to pigs. Strict enforcement of cooking of feed in conjunction with a slaughter program lead to eradication of the disease in swine in 1959. Early diagnosis and eradication by test and slaughter are important if VES were to recur. SMSV is endemic in pinnipeds along the western coast of the United States.

Suggested disinfectant for housing facilities: Phenols, sodium hydroxide, formalin, sodium carbonate, ionic and non-ionic detergents, strong iodophors in phosphoric acid, chloroform. Sodium hypochlorite (0.1% solution, or a 1:32 dilution) are effective in the absence of organic material.

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Notification: VES is not reportable to USDA/APHIS or OIE. However, this disease is considered eradicated and is clinically indistinguishable from other vesicular diseases that are reportable.

Measures required under the Animal Disease Surveillance Plan: None specifically but due to similar appearance to other reportable vesicular diseases.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Consult USDA/APHIS.

Experts who may be consulted: USDA State Veterinarians or federal Area Veterinarians in Charge.

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VESICULAR STOMATITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Swine, equids, bovids, South American camelids.	Biting insects (transovarial transmission in sandflies and blackflies), direct contact, fomites.	Pyrexia, anorexia, lameness, vesicles progression to erosions (coronary bands, oral cavity, teats).	Low to moderately contagious. Low to moderate morbidity.	None	No carrier state. Test and quarantine animals, disinfect environment, control insect vectors, do not feed uncooked pork products.	Yes

Fact Sheet compiled by: Cora Singleton

Sheet completed on: updated 8 August 2018

Fact Sheet Reviewed by: Kristi Delaski

Susceptible animal groups: Swine, horses, cattle; Tapirs serologically positive, but no clinical disease reported.

Causative organism: A vesiculovirus in the *Rhabdoviridae* family.

Zoonotic potential: Yes. Vesicular stomatitis virus causes pyrexia, headache, myalgia, and occasional blisters in the oral cavity of humans.

Distribution: enzootic in the US and present in North, Central, and South America.

Incubation period: 1-5 days

Clinical signs: Pyrexia, anorexia, lameness, vesicles progressing to erosions (coronary bands, oral cavity, teats). Clinically indistinguishable from foot and mouth disease, vesicular exanthema of swine, Seneca virus A, and swine vesicular disease. Epizootics in the United States occur about every 10-13 years, starting in early summer and ending with the onset of freezing weather.

Post mortem, gross, or histologic findings: Vesicles on coronary bands, snout, lips, oral cavity. Hydropic degeneration and edema of stratum spinosum of the affected epidermis, followed by ballooning degeneration of keratinocytes that then float into the vesicular fluid. Stratum basale remains intact.

Diagnosis: Agent identification – virus culture with electron microscopy, ELISA, complement fixation, PCR. Serology – ELISA, virus neutralization (often preferred – may need to test for Indiana and New Jersey strains), complement fixation.

Material required for laboratory analysis: Vesicular fluid, epithelium covering a vesicle, serum, tissues in formalin.

Relevant diagnostic laboratories:

Foreign Animal Disease Diagnostic Laboratory, Plum Island
40550 Route 25 (for packages)
Orient Point, NY 11957
P.O. Box 848 (for letters)
Greenport, NY 11944-0848
(631) 323-3256 Fax: (631) 323-3366

Since vesicular diseases cannot be distinguished clinically, contact the proper authorities prior to sample collection and shipment.

Treatment: No effective treatment. Supportive care and treatment of secondary problems.

Prevention and control: Prevention should include no feeding of uncooked pork products, regulation of movement of animals and animal products, and control of insect vectors. Vaccination has not been used routinely in the United States but might be useful during an epizootic. Control measures include notification of authorities, quarantine or depopulation of infected animals, and disinfection of the environment.

Suggested disinfectant for housing facilities: Phenols, sodium hydroxide, formalin, sodium carbonate, ionic and non-ionic detergents, strong iodophors in phosphoric acid, chloroform, ethanol, glutaraldehyde.

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Notification: Reportable to the USDA/APHIS through the State Veterinarian or the federal Area Veterinarian in Charge. The disease is also reportable to the World Organization for Animal Health (OIE).

Measures required under the Animal Disease Surveillance Plan: Reportable

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Infections must be reported to USDA/APHIS for management.

Experts who may be consulted: USDA State Veterinarians or federal Area Veterinarians in Charge.

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VIBRIOSIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Marine and brackish fish. Occasionally reported in freshwater fish. Commonly found in mollusks and crustaceans.	Unknown in many cases. Fish to fish contact and oral transmission is suspected. Some species may use invertebrate vector.	Acute or chronic forms. Nonspecific, e.g., lethargy, darkening, ulcers, petechial hemorrhages, erythema, coelomic distension, ocular, neurologic, or respiratory signs.	Significant mortalities possible in outbreaks (>50%).	Systemic antibiotics based on culture and sensitivity and regulations.	Appropriate water quality and reduction of other stressors (e.g., over-crowding, elevated temperature). Effective vaccines available for <i>V. anguillarum</i> .	Many strains are zoonotic.

Fact Sheet compiled by: Catherine A. Hadfield

Sheet completed on: 28 November 2010; updated 5 July 2013

Fact Sheet Reviewed by: Brent R. Whitaker, E. Scott Weber III

Susceptible animal groups: Over 50 species of marine and brackish fish (including elasmobranchs) are susceptible and disease is occasionally reported in freshwater fish.

Causative organism: *Vibrio* spp. are pleomorphic Gram negative rods. Some can be primary pathogens, but most are ubiquitous in the environment and cause secondary disease. More than 20 serovars may cause disease: *Vibrio anguillarum* (salt water furunculosis), *V. salmonicida* (hitra or cold water vibriosis), *V. alginolyticus*, *V. cholerae*, *V. fischeri*, *V. harveyi* (*carchariae*), *V. ichthyenteri*, *V. logei*, *V. ordalli*, *V. parahaemolyticus*, *V. pelagius*, *V. splendidus*, *V. tapetis*, *V. vulnificus*; *Moritella viscosa*, *M. marina*; *Photobacterium damsela*, *P. damsela piscicida*.

Zoonotic potential: Many species have zoonotic potential through skin wounds or ingestion of infected shellfish.

Distribution: Worldwide; first reported in North America in 1953.

Incubation period: Variable.

Clinical signs: Acute or chronic presentation occurs with non-specific clinical signs, e.g., lethargy, inappetance, skin darkening, scale loss, ulcers, hyperemia, petechiation, erythema, coelomic distension from ascites or organomegaly, corneal edema or ulceration, and exophthalmia. Neurologic or respiratory signs may be observed. Many fish die acutely without external signs and mortalities may be >50%. High index of suspicion in a zoo/aquarium setting after shipping or other stressors.

Post mortem, gross, or histologic findings: Visceral petechiation, congestion and/or necrosis of organs (especially kidney), organomegaly (especially spleen), and fibrinous adhesions can be observed. Weakly motile, pleomorphic, Gram negative rods may be present. Inflammation, which may be granulomatous, can be observed histologically.

Diagnosis: Pure bacterial culture from lesions, blood, or organs (especially kidney and spleen) with consistent

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clinical signs is supportive for diagnosis, although the organism may be commensal in elasmobranch tissues. Selective media available (e.g., TCBS) but these organisms can grow well on blood agar and other nutrient-rich media. Incubation temperature needs to be lower for *Vibrio salmonicida*. Serology not available.

Material required for laboratory analysis: Aerobic culturette and/or blood culture vials. Tissue swabs or preferably tissue samples for culture. Transport at 4°C.

Relevant diagnostic laboratories: Laboratories specializing in fish pathogens, although regular laboratories may be able to culture and identify *Vibrio* spp.

Treatment: Systemic antibiotics (e.g., trimethoprim sulfa, tetracyclines, florenfenicol, aminoglycosides) are needed but treatment should be adjusted as indicated by culture and sensitivity results and should follow all relevant legislation. For foodfish, follow guidelines for FDA-approved antibiotics (e.g., oxtetracycline). Nutritional support and supportive care can assist treatment. Immunostimulants, e.g., glucans, alginate or ascorbic acid.

Prevention and control: For outbreaks in aquaculture stocks, regulations may require movement restrictions, depopulation, and disinfection of premises. Most serovars, however, are ubiquitous, secondary pathogens. Control of stressors (e.g., temperature, water quality, stocking density, organic load, nutrition) is sometimes enough to control infection. Selective breeding has been used in salmonids to develop resistance to *V. anguillarum*. Immersion vaccine for *V. anguillarum* in salmonids (Novartis) is available and autogenous vaccines may be considered.

Suggested disinfectant for housing facilities: Susceptible to most common disinfectants (e.g., sodium hypochlorite and other chlorine-based disinfectants, ethanol, iodophors, quaternary ammonium compounds, and peroxygen compounds).

Notification: None required.

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: To be avoided with *V. anguillarum*. Other *Vibrio* spp. are ubiquitous, but avoid introducing animals if clinical signs are present.

Conditions for restoring disease-free status after an outbreak: Not applicable in most settings.

Experts who may be consulted: Most fish clinicians will be familiar with vibriosis and can be consulted if an outbreak is encountered.

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VIRAL HEMORRHAGIC SEPTICEMIA

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Fish: more than 80 susceptible marine and freshwater species	Horizontal transmission via fish waste, water, fomites, etc.	Clinical presentation varies by host species and strain, ranging from severe dermal, muscle, and organ hemorrhage to lethargy, to no significant lesions.	Dependent on species; from mild to lethal.	No effective treatment is available for free-ranging or captive fish.	Limit the spread through the movement of infected fish, water, and fomites.	No

Fact Sheet compiled by: Nicholas Phelps

Sheet updated on: 17 January 2018

Fact Sheet Reviewed by: James G. Johnson III, Elsburgh “Tres” Clarke

Susceptible animal groups: Fish: 80 susceptible marine and freshwater species. The virus has also been detected in amphipods, leeches, and turtles. It is not known what role these non-fish species play in the ecology of the virus.

Causative organism: Viral hemorrhagic septicemia virus (previously known as Egtved virus), in the Family Rhabdoviridae

Zoonotic potential: No

Distribution: Viral hemorrhagic septicemia virus has a broad distribution in the northern hemisphere. Four primary strains of VHSV are known to exist, distributed in Europe (VHSV-I, II, III), East Asia (VHSV-I, III, IV), and North America (VHSV-IV). VHSV-IV is further divided into marine (VHSV-IVa) and freshwater (VHSV-IVb). In North America, VHSV-IV has been detected off the Northern Pacific and Atlantic coasts as well as in the Great Lakes region.

Incubation period: An inverse correlation has been recorded between virus stability and water temperatures ranging from 1°C to 20°C. Transmission occurs at cooler temperatures (1-12°C) with an incubation time of 1-2 weeks at high temperature and 3-4 weeks at low temperatures.

Clinical signs:

Acute: Results in rapid destruction of endothelial cells and extravasation of the blood supply, which may ultimately result in diffuse or petechial hemorrhage, ascites, exophthalmia, organ failure, anemia (pale gills), and high mortality.

Chronic: Results in prolonged disease with neurologic-type behavior characterized by anorexia, erratic swimming, or lethargy.

Clinical presentation is dependent on a variety of factors including host, pathogen, or environmental variables. Some species exhibit no clinical lesions while infected with high levels of VHSV, while others develop severe lesions with low levels of VHSV. Presumptive diagnosis can be difficult and secondary testing is recommended.

Post mortem, gross, or histologic findings: VHSV has a predilection for endothelial cells and will often induce hemorrhagic lesions throughout the body visible by gross and histologic examination. The virus will also cause necrosis and degeneration of hematopoietic tissues, macrophage proliferation within renal tissue, and degeneration and vacuolization of hepatic tissue.

Diagnosis: The gold standard for VHSV detection is virus isolation by cell culture. Suitable cell lines include Epithelioma Papulosum Cyprini (EPC), Rainbow Trout Gonad (RTG-2), Bluegill fry (BF-2), Chinook salmon embryo (CHSE-214), and the Fathead minnow (FHM) cell lines incubated at 15°C. Cytopathic effects are typically observed within 4-6 days, but may take up to four weeks and two passages to appear. Secondary testing by RT-PCR or IFA are recommended. Real-time RT-PCR is becoming widely used for preliminary diagnosis and surveillance testing and can be performed on non-lethal samples (i.e., fin or gill biopsy).

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Material required for laboratory analysis:

Virology: Fresh tissue homogenate of the kidney and spleen should be placed in plastic tubes or whirlpack bags with 1g tissue to 10mL dilution with virus transport media (i.e. Hank's Balanced Salt Solution). Ship samples overnight on frozen gel packs.

RT-PCR: Tissue should be placed in tubes with RNAlater or immediately frozen. Tissue storage in 70% ethanol is an option, but freezing is necessary for long term storage. Contact the diagnostic lab where tissues will be sent to determine the types of tissues they will accept for non-regulatory testing and their recommended method of preservation.

Relevant diagnostic laboratories:

Please see the list of experts below; all of whom will accept diagnostic samples for preliminary testing. Additionally, USDA-APHIS approved labs for export certification of aquacultured species can be found at: http://www.aphis.usda.gov/animal_health/animal_dis_spec/aquaculture/

Virus Reference Laboratory
Diagnostic Virology Laboratory
USDA-APHIS National Veterinary Service Laboratory
1920 Dayton Avenue, Ames, Iowa 50010
janet.v.warg@aphis.usda.gov

Treatment: Therapeutics are not widely used to control VHS infection. General supportive care and stress reduction are recommended.

Prevention and control: Given the lack of available therapeutics, preventing the introduction of VHSV is the primary method of control. In addition, early detection of the virus by proactive surveillance programs provides value in determining areas or activities of risk. Strict biosecurity protocols should be implemented in areas of risk.

Prevention can be achieved by eliminating the transfer of the virus via contaminated fomites, eggs, fish, and water. Typical anti-viral disinfectants, such as chlorine, sodium hypochlorite, and UV irradiation are effective. Iodophor treatment of eggs is not always effective at removing the virus from the eggs' surface. However, at this time, no evidence for true vertical transmission of VHSV has been recorded; viral adherence to the egg surface and presence in ovarian fluid has been documented.

Suggested disinfectant for housing facilities: If captive fish test positive, the population should be isolated or euthanized. Housing facilities should be cleaned and disinfected with standard products such as chlorine and sodium hypochlorite. Facility effluent should also be disinfected with similar chemicals. For recirculating facilities, in-line UV sterilization should be incorporated to prevent the transmission of the virus via contaminated water.

Notification: VHSV is a reportable pathogen to the OIE and USDA. Upon suspicion or preliminary diagnosis, the area veterinarian in charge should be notified.

Measures required under the Animal Disease Surveillance Plan: The National Aquatic Animal Health Plan provides some guidelines for surveillance of aquatic animals. There is no coordinated surveillance plan for this disease in wild populations; however, regulatory and research surveys do occur. Any suspect case in a new species or geographic region needs to be reported to AVIC USDA APHIS.

Measures required for introducing animals to an infected animal(s): Susceptible species of naïve fish should not be introduced to a previously infected population. It may be possible to co-habitat non-susceptible species with a previously infected population; however, this approach is risky because the host range is broad and rapidly expanding.

Conditions for restoring disease-free status after an outbreak: No specific standards exist at this time; however, non-lethal antibody and quantitative RT-PCR methods are available to monitor a population over time.

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VIRULENT NEWCASTLE DISEASE
(Formerly Exotic Newcastle Disease)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Birds	Highly contagious Avian Paramyxovirus – 1 (APMV-1). Aerosol and ingestion are the primary routes. Inadvertent comingling asymptomatic with non-exposed birds.	Death; gastro-intestinal and respiratory signs	Severe; typically fatal	Not usually performed. In zoo specimens treatment is supportive care with consideration of vaccination.	Strict biohazard containment. Vaccination program may be considered. Depopulation and disinfection of premises then no new birds for 30 days	Rarely humans exposed may develop self-limiting infections. Cooked poultry products are safe to eat.

Fact Sheet compiled by: Nancy Carpenter

Sheet completed on: 1 January 2011; updated 18 March 2013, updated 2018

Fact Sheet Reviewed by: Erika Travis-Crook, Donald L. Janssen

Susceptible animal groups: Numerous species of birds (250 species to date) have been affected. Primary concerns are domestic poultry operations (chickens > turkey) and free-ranging double-crested cormorants are particularly susceptible. Penguins are highly susceptible and often die acutely; psittacines show varying susceptibility and prolonged shedding of virus. It has not been reported in mammals, except humans when they are exposed to a highly infected environment or during the vaccination process.

Causative organism: RNA virus within avian paramyxovirus-1 group (APMV-1) Genus *Avulavirus*, Family Paramyxoviridae. It should be noted that the mild strains are endemic to the U.S. with the most virulent strains being in other countries.

Zoonotic potential: Yes. Humans who have exposure to infected birds may get conjunctivitis or mild flu-like symptoms. No human cases of Newcastle Disease have ever occurred from eating poultry products.

Distribution: Worldwide but endemic in the Middle East, Asia, Africa, Central and South America.

Incubation period: 2-15 days and depends upon the virulence of the strain, the susceptibility of the population and the species affected.

Clinical signs: In rare human infections, clinical signs include self-limiting conjunctivitis and flu-like symptoms. In animals, clinical signs vary by pathotype:

Asymptomatic enteric – generally subclinical

Lentogenic or respiratory – mild or subclinical respiratory signs

Mesogenic – respiratory and occasional neurologic signs with low mortality

Velogenic – most virulent with high mortality rates.

a. neurotropic – respiratory (coughing, gasping) and neurologic signs (muscle tremors, circling, paralysis; green watery diarrhea; decreased egg production (NVND))

b. viscerotropic – hemorrhagic gastrointestinal disease and lesions (VVND)

In domestic laying hen operations, initially a drop in egg production occurs and then numerous deaths within 24-48 hours which will continue for 7-10 days. Birds that survive for 12-14 days may live but may have permanent neurologic damage.

Post mortem, gross, or histologic findings: No specific post mortem lesions are present. However, relevant gross lesions are usually found only in birds infected with velogenic strains include:

hemorrhage, ulcers, edema and/or necrosis often occur in the cecal tonsils and lymphoid tissues of the intestinal wall (including Peyer’s patches); this lesion is particularly suggestive of Newcastle disease. In chickens infected with less virulent strains, the lesions may be limited to congestion and mucoid exudates in the respiratory tract, and opacity and thickening of the air sacs.

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Diagnosis: Virus isolation and microscopic lesions identified in tissues. Differential diagnosis list for infectious diseases includes avian cholera (*Pasteurella multocida*), highly pathogenic avian influenza (HPAI), infectious aryngotracheitis (herpesvirus), infectious coryza (*Hemophilus paragallinarium*), diphtheritic avianpoxvirus, psittacosis (chlamydophylosis (*Chlamydophila psittaci*), mycoplasmosis, infectious bronchitis (coronavirus), and, in psittacines only, Pacheco's disease.

Material required for laboratory analysis: Contact laboratory in advance of collections to ensure proper collection storage and shipping methods. Brain and Heart infusion broth (BHI) with high concentrations of antibiotics should be used for transport. Freeze if samples will not be received by the laboratory within 24 hours.

Swabs of trachea, oropharynx, and cloaca can be collected from live birds. Tissue samples from dead birds include trachea, lung, spleen, cloaca, intestines, cecal tonsils, brain. Feces for culture can be collected from either live or dead birds. Serum for ELISA can be used but previous exposure and vaccination may affect results. Reverse Transcriptase PCR is also available. However, results returning before that particular animal might die may be problematic.

Relevant diagnostic laboratories: Testing is performed at numerous state labs.

Treatment: It is not recommended to pursue treatment and typically flock depopulation is performed in domestic poultry operations. However, in a zoo situation the benefit of treatment should outweigh the risk of transmission to other birds.

Prevention and control: **There is no effective cure for virulent Newcastle Disease.** Once identified, strict biohazard control methods should be immediately implemented. Slaughter and disposal of all infected and exposed birds is recommended. No new birds in for 30 days. Pests must be controlled to minimize mechanical transfer of the virus.

Suggested disinfectant for housing facilities: cresylics and phenolics

Notification: State and Federal veterinarians should be notified.

Federal: http://www.aphis.usda.gov/animal_health/area_offices/ - use map for regional instructions.

State: <http://www.usaha.org/Portals/6/StateAnimalHealthOfficials.pdf>

Measures required under the Animal Disease Surveillance Plan: This is a reportable disease and control must be managed with regional veterinary authorities. Once the disease is confirmed, strict biosecurity measures should be taken. Depopulation to prevent spread must be considered.

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: Do not repopulate infected areas for at least 30 days from final disinfection.

Experts who may be consulted:

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VISCERAL LEISHMANIASIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Canids Humans	Phlebotomine sand flies (<i>Lutzomyia</i> spp.); transplacental, sexual, and via blood transfusion also reported in dogs	Lymphadenopathy, onychogryphosis, weight loss, alopecia, conjunctivitis (Dogs); Fever, weakness, lethargy, weight loss, hepatosplenomegaly, lymphadenopathy (Humans)	Fatal if not treated	Allopurinol, meglumine antimoniate, liposomal amphotericin B	Insecticides for sandfly control	Yes, but requires vector

Fact Sheet compiled by: Christine Fiorello

Sheet completed on: 20 January 2011; updated 1 November 2012

Fact Sheet Reviewed by: Sara Childs Sanford, Walter Boyce

Susceptible animal groups Domestic and wild canids are the main host species. Humans are commonly infected, and the infection is becoming more commonly recognized in domestic cats. Opossums and some rodents are also commonly infected, although usually asymptomatic. However, the domestic dog is the only epidemiologically important reservoir.

Causative organism Protozoal organisms *Leishmania donovani* (Asia, Middle East, Africa) and *L. infantum* (Asia, Middle East, Europe, South America)

Zoonotic potential Transmission occurs via sandfly bites; dogs are the reservoir host. Humans are accidental hosts and not considered important in the epidemiology of the disease. Dog to human and human to human transmission does not seem to occur

Distribution Europe, South America, Africa, Middle East, Asia. Dogs in North America are occasionally infected.

Incubation period Weeks to months

Clinical symptoms Humans: Fever, weakness, lethargy, weight loss, muscle wasting, hepatosplenomegaly, lymphadenopathy, pallor; anemia & thrombocytopenia are common. **Dogs:** lymphadenopathy, onychogryphosis, weight loss, conjunctivitis, alopecia.

Post mortem, gross, or histologic findings: Inflammation and parasites found in macrophages of infected organs; specific findings vary with parasite and host species, chronicity of disease, and immune status of host.

Diagnosis Gold standard: demonstration of parasites (amastigote form) in splenic or bone marrow aspirates. Serologic tests include an IFAT, ELISA (rK39 antigen most promising), DAT, and immunochromagrapic test strip. Numerous blood and bone marrow PCR protocols are also often used.

Material required for laboratory analysis Depends on diagnostic method; could include bone marrow, lymph node, or splenic aspirates or blood.

Relevant diagnostic laboratories In the US, Cornell University Animal Health Diagnostic Lab, Michigan State Diagnostic Center for Population and Animal Health, National Bio Vet Lab are some of the many labs that have commercial tests available.

Treatment Humans: Liposomal amphotericin B is first choice. Meglumine antimoniate is less expensive but has more adverse effects. Miltefosine is a newer oral drug that has shown good efficacy in India. **Dogs:** Allopurinol, meglumine antimoniate, and liposomal amphotericin B have all been used; a complete cure is

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usually impossible and euthanasia is often recommended.

Prevention and control: Culling of dogs does not seem to be effective. Insecticide spraying around human settlements to control sandflies has been effective in some areas but not in others. Insecticide-impregnated nets can provide protection for individuals. Deltamethrin-impregnated collars and various insecticide pour-ons for dogs provide limited efficacy in decreasing transmission.

Suggested disinfectant for housing facilities Control of the disease is based on control of the insect vector.

Notification Not a nationally notifiable disease in the US; it is notifiable in a few states such as Texas.

Measures required under the Animal Disease Surveillance Plan:

Measures required for introducing animals to infected animal: Not relevant (vector-borne disease)

Conditions for restoring disease-free status after an outbreak: Not relevant (vector-borne disease)

Experts who may be consulted:

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WEST NILE VIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Avian Equids Squirrels Other mammals, Alligators	Primarily via mosquito bite, possibly ticks, less efficient means include ingestion of virus via infected carcasses or contaminated tissues or fluids (feces, urine, oral or cloacal secretions); occupational exposure, blood transfusion, organ transplant, and maternal transmission	Range: asymptomatic to non-specific (e.g., anorexia, weight loss, dehydration) to neurologic (e.g., ataxia, lethargy, paresis, paralysis, convulsions, seizures, death)	High fatality in some avian species (especially corvids and some raptors); 10-30% of equine clinical cases are fatal; <1% of human cases are severe (i.e., West Nile neuroinvasive disease)	Supportive care, immuno-globulin therapy	Mosquito control; avoiding mosquito bites (repellant, screens, clothing, staying indoors at dawn and dusk); vaccination licensed for horses also used extra-label in some birds (primarily captive)	Yes

Fact Sheet compiled by: Genevieve Vega Weaver

Sheet completed on: 15 January 2018

Fact Sheet Reviewed by: Heather Robertson

Susceptible animal groups: Changes in global climate, land-use, and biodiversity as well as potential virus evolution will continue to increase exposure as well as increasing the potential for disease in vulnerable, naïve species. Many avian species serve as amplifying hosts for WNV. American robins, house finches, house sparrows, and other species are considered high amplification hosts due to high proportions of WNV-positive mosquito blood meals. Several species of mammals (squirrels, chipmunks, and rabbits), a reptile (alligators), and an amphibian (lake frogs) are unlikely to serve as amplifying hosts for West Nile virus (WNV), as viremia titers are relatively low in these species as compared to birds, and the duration of infectious viremia is short (e.g., approximately 1 day). Most mammals are incidental (i.e., dead-end) hosts. Very young and old animals are likely most susceptible to adverse effects of infection.

Birds: WNV has been reported in at least 326 species of birds in North America and over 1,300 avian species worldwide. All bird species are likely susceptible to WNV infection, although most infections in most species are likely subclinical. North American birds that are of particularly high susceptibility to WNV-associated morbidity and mortality are the American crow as well as other corvids (e.g., blue jays, black- and yellow-billed magpies, fish crows, and others), and to a lesser extent, other passerine species (e.g., common grackles, house sparrows, house finches). Some competent mosquito vector species are ornithophilic and at least one study has shown that *Culex pipiens* preferentially feeds on raptor species in some situations (owls, eagles, falcons, hawks). Species of special concern due to apparently high rates of susceptibility and conservation status include California condors, Florida scrub jays, greater sage grouse, ruffed grouse, loggerhead shrike, and native Hawaiian birds. High rates of death were observed in free-ranging, juvenile American white pelicans in nesting colonies, captive lesser scaup ducklings, experimentally-infected and free-ranging greater sage grouse and experimentally-infected ruffed grouse. There is also concern over numerous raptor species, such as great horned owls and northern owl species; most observations come from rehabilitation facilities. Other birds have been documented with WNV infection, including flamingos, penguins, emus, wild turkeys, cormorants, bronze-winged ducks, sandhill cranes, common coots, red-legged partridges, and others. A variety of psittacine species housed in outdoor aviaries, many of which were of Australian origin, had clinical WNV disease. Reports of WNV disease and death in New World psittacines are relatively rare. Antibodies to WNV have been detected in a vast array of avian species, sometimes at high prevalences.

Mammal: WNV-associated disease in mammals is most severe in equids and can also be significant in squirrels. It has rarely been reported in alpacas, sheep, reindeer, harbor seals, Indian rhinoceroses, a polar bear,

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a wolf and several domestic canids, a Barbary macaque, white-tailed deer and a killer whale. Antibodies to WNV have been detected in a variety of mammals including cattle, dogs, cats, goats, rabbits, raccoons, Virginia opossums, coyotes, striped skunks, bats, black bears, wild boar, red foxes and killer whales, indicating prior infection.

Reptile and amphibian: Some reptiles and amphibians are susceptible to experimental infection, including the American alligator, Nile crocodile, green iguana, crocodile monitor, garter snake, various chelonians, lake frog and North American bullfrog. Among these, alligators had clinical (neurologic) signs.

Causative organism: West Nile virus is a single-stranded, enveloped, RNA virus of the Japanese encephalitis antigenic group, genus *Flavivirus* and Family *Flaviviridae*. It is an arthropod-borne virus (“arbovirus”) transmitted by mosquitoes. *Culex* spp. and *Aedes* spp. are primary vector species. *Culex pipiens*, in particular, is an important bridge vector from avian hosts to humans. There is one published study that suggests that WNV may be waterborne with at least one major outbreak (in bald eagles and eared grebes in Utah) caused by contaminated water and invertebrate prey.

Zoonotic potential: Yes. Transmission to humans is predominantly via mosquito bite but risk is also present during handling tissues and fluids, as transmission can occur via inhalation, mucous membrane contact, open cuts and puncture wounds from a needle stick or contaminated equipment. Mask with face shield and gloves should be worn when handling suspect animals and bedding.

Distribution: Worldwide

Incubation period: Approximately 3-15 days in horses, 2-14 days in humans, and 4-14 days in birds. Some birds become detectably viremic by 1 day post-inoculation.

Clinical signs:

People: The fatality rate is approximately 4%. The majority of infected people are asymptomatic, although some have mild, non-specific symptoms (“West Nile fever,” involving fever, headache, fatigue and/or myalgia/arthralgia; skin rash is also possible), and rarely (<1% of those diagnosed with WNV) experience severe, neurological symptoms (“West Nile neuroinvasive disease,” involving encephalitis, meningitis, and/or paralysis), leading to death in geriatric patients. There is evidence that the human fatality rate may actually be higher than what is currently reported especially in people less than 60 years old. Complications, such as cognitive dysfunction, can develop many years later related to the initial WNV infection.

Birds: Clinical signs vary and can include depression, ruffled feathers, anorexia, dehydration, rapid weight loss, decreased activity to lethargy, torticollis, opisthotonos, nystagmus, ataxia, diarrhea, nasal discharge, drooping wings, labored breathing, and sudden death. Most affected birds deteriorate rapidly following the onset of clinical signs; however, there are several reports of captive birds exhibiting clinical signs for weeks or months. In some cases, WNV-infected raptors have had feather abnormalities, including stunted growth and pinched-off feathers (at the quill).

Equids: About 10% of infected horses develop clinical disease that may include anorexia, depression, ataxia, paresis, paralysis, teeth grinding, aimless wandering, convulsions, circling, tremors of facial and neck muscles, cranial nerve deficits, difficulty swallowing, hyperesthesia, apprehension, hyperexcitability, facial edema, coma, impaired vision, conjunctivitis, abdominal pain, colic, urinary dysfunction, fever, and head pressing. Injuries and secondary pulmonary infections due to prolonged recumbency can also occur. Horses that recover usually show improvement within 7 days of onset of signs. About 10-20 % of recovered horses have residual effects, such as neurologic deficits.

Squirrels: Head tilt, tremors, paralysis, and ataxia.

Reptiles (alligators): Anorexia, weakness, tremors, slow reflexes, heat tilt, anisocoria, opisthotonos, circling, and lymphohistiocytic proliferative cutaneous lesions.

Postmortem, gross, or histologic findings: Lesions are variable among species and there or no pathognomonic findings.

Birds: Gross lesions are often absent, but can be non-specific, including white-tan mottling or streaking of the myocardium, splenomegaly, congested cerebral vessels, and poor nutritional condition. Histologic lesions can

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be minimal to severe, and can include lymphoplasmacytic myocarditis, encephalitis, ganglionitis, hepatitis, and nephritis and occasional adrenal gland and pancreatic necrosis. Vasculitis can also occur.

Equids: Gross lesions are usually absent. When gross lesions are present, they are most often in the CNS: submeningeal edema and hemorrhage of spinal cord, brain stem, and midbrain. Histologic findings: nonsuppurative encephalitis or encephalomyelitis.

Squirrels: No gross lesions are evident. Histologic findings may include lymphoplasmacytic encephalitis or meningoencephalitis with multifocal microglial nodules, perivascular and meningeal infiltrates of neutrophils, neuronal necrosis, and neuronophagia.

Other mammals: Few reports of gross lesions. Histologic lesions are similar to equids.

Reptiles: Fluid in coelomic cavity; mottled enlarged liver, spleen, and myocardium. Intracellular heterophilic infiltrates in epithelial cells and cellular necrosis.

Diagnosis

Serology: Increase in WNV-specific antibodies in acute and convalescent sera, IgM in CSF, or IgM in serum (suggestive). ELISA, with confirmation of results by plaque reduction neutralization test.

Virus isolation, or antigen or RNA detection: Infectious virus (virus isolation) or viral components (RT-PCR) can be detected in serum, CSF, homogenized tissues (brain, heart, kidneys and spleen), oral/cloacal swabs, and/or urine of some animals. The period in which virus can be detected in live animals is limited, and can be especially difficult in animals with low viremia titers (e.g., horses). RT-PCR can be more sensitive than virus isolation. Immunohistochemistry is most useful during active infection and in birds, viral antigen may be evident in kidney, heart, spleen, and to a lesser extent in other tissues such as brain, pancreas, liver, and intestine, and others.

Material required for laboratory analysis: Bodily fluids such as blood (centrifuged for separation of serum or plasma), CSF, urine, saliva, or swabs of body cavities (oropharyngeal or cloacal cavities, rectum), or tissues (heart, kidney, and spleen have been consistently useful for virus isolation and PCR testing in birds and can also be useful for immunohistochemistry [IHC] in birds); feather pulp, nonvascular feathers, brain, eye, spinal cord, liver, and others; tissues can be pooled to possibly increase sensitivity. Testing maggots from carcasses for RNA may be useful in decomposed birds.

Relevant diagnostic laboratories: Most state public health laboratories conduct WNV testing; however, virus isolation and plaque reduction neutralization tests are time and labor intensive and require BSL-3 laboratory conditions.

Arbovirus Diagnostic Laboratory, DRA

CDC/DVBID/ADB

3150 Rampart Road

Fort Collins, CO 80521

Phone: (970) 221-6445 http://www.cdc.gov/ncidod/dvbid/misc/arboviral_shipping.htm

Formalin-fixed specimens for immunohistochemistry:

Infectious Disease Pathology Activity

CDC (MS-G32)

1600 Clifton Rd, NE

Atlanta, GA 30333

Phone: 1-800-232-4636

National Wildlife Health Center, USGS

6006 Schroeder Road

Madison, Wisconsin 53711

Phone: (608) 270-2400 Fax: (608) 270-2415

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Treatment: Supportive care (i.e., fluids, nutrition, heat or cold) can be provided with the goal of reducing CNS inflammation, preventing injuries, minimizing the effects of prolonged recumbency, and nursing the animal beyond the severe morbidity that can occur. Morbid birds and horses can have neurologic deficits that range from ataxia to paralysis and seizures, and therefore, padded caging may be necessary to prevent further injury. Long-term (i.e., up to several years) neurologic defects have been observed in some raptors following WNV infection. Immunoglobulin therapy has been used in horses (serum based, Novartis Animal Health and a plasma based product, Lake Immunogenics, Inc.). L-lysine supplement and homeopathic treatments have been used with some success in raptors. Mild cases may resolve without treatment.

Prevention and control: Mosquito control measures should be implemented: screened housing, fans, repellants (10% DEET), avoiding stagnant water, larvicides, and stocking mosquito fish in ponds. Insect repellants listed by the CDC as being EPA-registered and providing long-lasting protection include: DEET, picaridin, natural or synthetic oil of lemon eucalyptus and IR3535 (3-[N-Butyl-N-acetyl]-aminopropionic acid, ethyl ester). Isolation of infected individuals and quarantine of new animals is recommended. Avoid feeding potentially contaminated meat/carcasses.

Four vaccines were developed for use in horses: a killed vaccine (West Nile-Innovator® DNA vaccine, Fort Dodge Animal Health), a recombinant vaccine in a canarypox vector (Recombitek®, Merial Animal Health), a flavivirus chimera vaccine (Equi-Nile™, Intervet), and a recombinant DNA plasmid-pCBWN (CDC/Fort Dodge Animal Health-not yet licensed). Many zoological facilities vaccinate equids and sensitive avian species with available vaccines. A hydrogen peroxide-inactivated whole virion WNV vaccine, HydroVax-001, for use in humans is currently in development.

Extra-label use of vaccines or use of vaccines that have not been adequately assessed in the target animal (i.e., controlled challenge studies) should be used with caution and not assumed to be protective. Numerous vaccines have been tested to various degrees in birds (some without challenge) with varied responses. Flamingoes failed to seroconvert after a single vaccination with the killed product. This vaccine provided some level of protection at a small dose in ruffed grouse; vaccinated grouse had no clinical disease, lower viremia titers and milder microscopic lesions than non-vaccinated grouse. A modified live vaccine was tested in domestic geese in Israel with 75-94% protection. The killed equine vaccine, DNA plasmid vaccine, and recombinant equine vaccine provided partial protection in island scrub jays. Some red-tailed hawks vaccinated with a DNA-plasmid vaccine had partial protection while American robins and California condors vaccinated with the same vaccine seroconverted. Results were variable among adult and juvenile thick-billed parrots vaccinated with the killed equine vaccine. Seroconversion occurred in some penguins following administration of DNA plasmid and killed vaccines. A DNA plasmid vaccine failed to protect greater sage grouse from mortality. Oral vaccines in fish crows were ineffective. The Recombitek vaccine was immunogenic in rhinos.

Suggested disinfectant for housing facilities: As an enveloped virus, WNV does not persist for long periods in the environment. 70% ethanol and bleach are sufficient for general cleaning. Viricides such as Virkon® are highly effective when concern is high but can be damaging to skin and mucus membranes.

Notification: Certain states require veterinary cases to be reported to the state animal health authority.

Measures required under the Animal Disease Surveillance Plan: Laboratory-confirmed positive cases in humans, horses, other mammals, birds and mosquitoes from across the United States are collected by ArboNET (Centers for Disease Control and Prevention; http://www.cdc.gov/ncidod/dvbid/westnile/usgs_frame.html). Equine cases are usually determined from passive reporting from private practitioners and diagnostic submissions.

Measures required for introducing virus to infected animals: WNV has been spread horizontally shortly after experimental inoculation in some birds that were housed in close captive quarters, as well as in the American alligator. Infected individuals should be isolated. Viremia usually wanes 5-10 days in birds and up to 14 days in alligators. However, experimental infection in hamsters resulted in urine viral shedding for over 300 days and infectious virus persisted in tissues of house sparrows for up to 43 days. Antibodies persist in some previously infected birds for years to life-long.

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Conditions for restoring disease-free status after an outbreak: WNV is firmly established in avian and mosquito populations worldwide. The virus is endemic and transmission is reinitiated annually in the summer within temperate areas of North America and Europe. Therefore, animals housed outdoors in endemic or at-risk areas will be at a continual risk. Seasonal and climatic factors may precipitate outbreaks of disease in wildlife (i.e., wild birds) that may spillover into captive populations and humans. Proper disinfection of housing facilities and equipment after an outbreak is necessary.

Experts who may be consulted:

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Southeastern Cooperative Wildlife Disease Study
Departments of Population Health and Pathology
University of Georgia
Athens, GA 30602

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WESTERN EQUINE ENCEPHALITIS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p>Mammals - Equids may show mild to severe clinical illness. Other mammals may be inapparently infected or show clinical illness. Humans may experience mild to severe disease.</p> <p>Birds- Emus, chukar partridges, pheasants, turkeys, and some other avian species may show clinical illness. Native birds generally serve as viral reservoirs without clinical illness</p> <p>Reptiles- Inapparent infection noted for snakes, tortoises, as well as amphibians (frogs)</p>	<p>Bite of WEE-infected mosquito, may also be transmitted by ticks.</p> <p>Possibly from direct contact with infected tissues at necropsy (i.e. through broken skin or mucous membranes)</p>	<p>Equids: Fever, anorexia, lethargy, impaired vision, dysphagia, circling, head pressing, paresis, paralysis, seizures</p> <p>Emus: asymptomatic infections are common; anorexia, watery diarrhea, weight loss, abnormal neck movements, neurologic signs</p>	Mild to severe, may be fatal	<p>No specific treatment but supportive care, hydration, and nutritional support are important. Anticonvulsant and anti-inflammatory treatment may be used.</p>	<p>Vaccination; mosquito control is important for routine exposure.</p> <p>Personal protective equipment when handling tissues and performing necropsies</p>	Yes, primarily by mosquito, less frequently via tick

Fact Sheet compiled by: Rose Borkowski

Sheet completed on: updated 2 August 2018

Fact Sheet Reviewed by: Danelle Okeson, Sarah Cannizzo

Susceptible animal groups: Mammals (Equids), Birds (emus, other exotics, turkeys, pheasants)

Causative organism: Western Equine Encephalitis Virus, an Alphavirus, Family Togaviridae

Zoonotic potential: Yes, primarily via mosquito bite. Primary mosquito vector is *Culex tarsalis*, although *Aedes* sp. may also transmit; ticks (*Dermacentor andersoni*) can serve as vectors as well.

Distribution: Argentina to Canada. In the US, it generally occurs west of Mississippi River. Currently a rare disease of humans and horses in the US.

Incubation period: 5-14 days

Clinical signs:

Animals: Equids - Fever, anorexia, lethargy, impaired vision, difficulty swallowing, circling, head pressing, paresis, paralysis, and seizures may be seen and disease is potentially fatal. Clinical signs may be similar to other neurologic disease including rabies, necessitating cautious examination and appropriate protective equipment.

Birds: Emus have demonstrated watery diarrhea, weight loss, neurologic signs, and fatalities. A drop in egg production may occur in poultry. The potential for WEE to cause disease in other avian species, particularly nonnative birds, exists. WEE generally causes inapparent infection in native birds as virus naturally cycles between mosquitoes and several passerine species.

Reptiles: Positive serologic tests indicating exposure have been demonstrated in reptiles. The ability of the virus to cause clinical disease in captive or wild reptiles is incompletely understood.

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<p>Humans: Fever, headache, stiff neck, disorientation, altered consciousness, coma, convulsions, and paralysis can be present, and death may occur. Infection is of particular concern for infants, elderly. Can have asymptomatic infections.</p> <p>Primates: Theoretic concern for nonhuman primates as fatal human illness has occurred.</p>
<p>Post mortem, gross, or histologic findings: Gross lesions are usually nonspecific. Congestion of brain and meninges may be seen as well as echymotic hemorrhages due to antemortem trauma. Severe inflammation of gray matter, neuronal degeneration, gliosis, perivascular cuffing and hemorrhage.</p>
<p>Diagnosis: Serology is from paired samples for virus neutralization (Plaque Reduction Neutralization Test), and IgM determination. Complement fixation (CF) and hemagglutination inhibition (HI) can be used for identification as well. Vaccination history is essential for accurate interpretation of serologic tests. Molecular Diagnostics (PCR) and virus isolation on brain and other tissues are available - brain preferred in equids, although many tissues may demonstrate virus in emus. Diagnostic testing to exclude rabies virus infection is required for submitted brain tissue.</p>
<p>Material required for laboratory analysis: Serum, Tissues (particularly brain. Note: rabies testing must be performed on brain tissue prior to submission of any additional brain samples from the same animal for WEE testing at National Veterinary Services Laboratory)</p>
<p>Relevant diagnostic laboratories: National Veterinary Services Laboratory 1920 Dayton Ave. Ames, IA 50010 Phone: (515) 337-7266 Fax: (515) 337-7397</p> <p>Will test serum for Eastern Equine Encephalitis as well as WEE. If submitting brain, cerebrospinal fluid, or whole blood for virus isolation, the brain must be tested for rabies prior to submission. https://www.aphis.usda.gov/animal_health/lab_info_services/downloads/AmesDiagnosticTestingCatalog.pdf</p>
<p>Treatment: No specific treatment is available for this disease. Identification of neutralizing antibodies that may have therapeutic value has been recently investigated. Patient management includes hydration, nutritional support, anticonvulsant, and anti-inflammatory treatment.</p>
<p>Prevention and control: Vaccination of equids is an important means of prevention. Extra-label use of vaccination for emus and potentially other ratites has been implemented. Prevention of mosquito and tick bites via use of repellants, protective clothing, screens, and fans. Enclosure modification to reduce areas for mosquito access and breeding. Avoidance of outdoor exposure during times of day when mosquitoes are most active. As viral neurologic diseases such as arboviral encephalitides and rabies cannot be distinguished from one another clinically, and may cause death, it is imperative that proper sharps handling and use of personal protective equipment occur when working with infected animals or their tissues. Although WEE is not believed to be directly transmissible from horses to humans under usual circumstances, performance of necropsies on infected animals of any species, and handling of their tissues, blood, and cerebrospinal fluid may pose risk. Prevent aerosolization of virus and contact of infected tissues and fluids with skin and mucous membranes. Do not use mechanical saws to obtain spinal cord samples due to risk of aerosolization. Additional recommendations for handling of potentially infected tissues include use of 3 pairs of gloves (inner layer disposable, middle layer waterproof, and outer layer of metal or Kevlar gloves), face shield or goggles plus a disposable “half mask” high efficiency particle arresting (HEPA) respirator.</p>
<p>Suggested disinfectant for housing facilities: The virus cannot survive outside of the host. It is susceptible to bleach, most disinfectants, aldehydes, ethanol, moist and dry heat, as well as drying.</p>
<p>Notification: A reportable animal disease in some states, refer to individual state veterinary regulations. It also is a notifiable disease in humans – www.cdc.org.</p>
<p>Measures required under the Animal Disease Surveillance Plan: Currently none</p>

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Measures required for introducing animals to infected animal: WEE is not known to be transmissible between mammals, birds, reptiles, or people. Vaccination against WEE is prudent for equids, rartites and potentially other mammalian and avian species in endemic areas.

Conditions for restoring disease-free status after an outbreak: Susceptible animals should be vaccinated. Ensure that veterinary equipment used with infected animals is discarded or disinfected prior to use with disease-free animals. Continue preventive measures against mosquito breeding and biting. If mosquito numbers are excessive, reduction in mosquito population via aerial spraying of pesticides can be discussed with public health officials, and state or county mosquito control agents.

Experts who may be consulted:

CDC/Division of Vector Borne Diseases
Arboviral Diseases Branch
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Fort Collins, CO 80521
(970) 221-6400

References:

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WHITE NOSE SYNDROME: Cutaneous Invasive Ascomycosis in Hibernating Bats

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
<p>Any hibernating bat in a WNS-affected area is considered at risk for the disease</p> <p>Microscopic lesions visible in hibernating bats in Europe and China without mass mortality</p>	Aerosol, direct contact, environmental exposure	<p>Abnormal hibernation activity (more frequent arousal, daytime flights during winter, congregating at or near cave openings)</p> <p>White mold (fungal hyphae) on muzzle, wings, or both may be present but <u>is neither necessary nor specific for WNS</u></p>	<p>North America: 90-100% mortality in some hibernacula. Population-wide losses of some species in the northeastern US are >80% since emergence of WNS. Recovery has been documented experimentally, and multi-year survival has been documented in wild, banded bats.</p> <p>Europe, Asia: Disease present with little noted morbidity or mortality</p>	<p>Supportive care (warmth, fluid & food supplementation)</p> <p>Natural recovery has been documented experimentally and in wild, banded bats upon emergence from hibernation</p>	<p>Biosecurity: limit human access to affected areas, decontaminate clothing and equipment after entering hibernacula or trapping bats in affected areas, biosecurity practices for handling <i>Pseudogymnascus destructans</i> (<i>Pd</i>) in laboratory (e.g. BSL-2)</p> <p>Reduce disturbance of hibernating bats: restrict human access to hibernacula</p>	<p>Not likely. <i>Pd</i> is a psychrophilic fungus; body temperature of humans is above that conducive to growth of <i>Pd</i>.</p>

Fact Sheet compiled by: Michelle L. Verant and Carol U. Meteyer

Sheet completed on: 3 August 2011; updated 10 July 2013; updated 15 February 2018

Fact Sheet Reviewed by: David Blehert, Anne Ballmann

Susceptible animal groups: Microchiropteran bats and primarily hibernating species. In North America, species confirmed with WNS include: little brown bat (*Myotis lucifugus*), tri-colored bat (*Perimyotis subflavus*), northern long-eared bat (*Myotis septentrionalis*), big brown bat (*Eptesicus fuscus*), eastern small-footed bat (*Myotis leibii*), Indiana bat (*Myotis sodalis*), gray bat (*Myotis grisescens*), southeastern bat (*Myotis austroriparius*), yuma bat (*Myotis yumanensis*), western long-legged bat (*Myotis volans*) and cave bat (*Myotis velifer*). Species or subspecies that have been detected with *Pd* but no diagnostic signs of WNS include: eastern red bat (*Lasiurus borealis*), silver-haired bat (*Lasionycteris noctivagans*), cave bat (*Myotis velifer*), Rafinesque's big-eared bat (*Corynorhinus rafinesquii*), Virginia big-eared bat (*Corynorhinus townsendii virginianus*), Townsend's big-eared bat (*Corynorhinus townsendii*), western small-footed bat (*Myotis ciliolabrum*) and Mexican free-tailed bat (*Tadarida brasiliensis*). In Europe and Asia (Russia, Mongolia, and China), 14 species of bats have been confirmed with WNS and an additional seven species have been detected with *Pd* but no diagnostic signs of WNS. For updates on affected species, see www.whitenosesyndrome.org

Causative organism: *Pseudogymnascus* (formerly *Geomyces*) *destructans*

Zoonotic potential: Not likely; psychrophilic character of fungus makes warm hosts unsuitable, although related *Geomyces* species have been known to rarely induce superficial infection of the skin and nails in humans.

Distribution: Since its first diagnosis in a New York cave in early 2007, WNS has continued its spread across eastern North America with newly affected sites identified annually. At this sheet completion, WNS has been

WHITE NOSE SYNDROME: Cutaneous Invasive Ascomycosis in Hibernating Bats

confirmed in hibernating bats in 32 states and 7 Canadian provinces. Additionally, *Pseudogymnoascus destructans* has been found in Mississippi, Texas and Wyoming without confirmation of disease to-date. The fungus remains viable in suitable underground environments year-round even in the absence of bats. Up-to-date distribution maps for North America can be found at: <http://whitenosesyndrome.org/resources/map>. The *Pd* fungus has also been found on bats or in caves across Europe and in Russia, Mongolia, and China, but without mass morbidity or mortality.

Incubation period: In the wild, WNS occurs seasonally with the earliest confirmed case in late September and peak infections and mortality occurring about 120 days after bats enter hibernation. Experimentally-induced infections result in epidermal pathology and mortality as early as 88 days post-infection.

Clinical signs: White-nose syndrome was named for the characteristic white fungal growth on the muzzles, pinnae, and wings of hibernating bats. However, this sign is not always apparent in bats with WNS, nor is it specific for the disease as other non-pathogenic dermatophytes may have a similar appearance. Epidermal erosions and destruction of wing tissue by *Pd* cause disruptions in homeostasis resulting in dehydration, electrolyte imbalances and acid-base disturbances. Abnormal behaviors associated with WNS include increased frequency of arousal from torpor, movement to roosting areas near cave entrances or other exposed sites, and increased day flights from hibernacula during mid-winter. This increased activity likely contributes to premature depletion of fat reserves seen in infected individuals. Bats with WNS may present with obvious damage to wing membranes (increased fragility, decreased elasticity, irregular pigmentation, and tears or holes in the patagium) as they emerge from hibernation and become euthermic. Wing damage may increase over the first few weeks post-emergence due to an excessive inflammatory response, but these lesions can heal completely by mid-summer.

Post mortem, gross, or histologic findings: Visible white fungal material on the muzzle and wings often disappears when a bat is removed from the hibernaculum. Infected wings typically look normal during hibernation, but areas of ‘contraction’ or tears can be present and wing membrane may stick together when the wing is extended. Bats that die from WNS during hibernation often have reduced subcutaneous fat and when touched with a gloved finger during necropsy, exposed pectoral muscle may be tacky suggesting ante-mortem dehydration. Microscopic findings are characterized by dense aggregations of Periodic acid-Schiff (PAS)-positive hyphae eroding through epidermis forming distinctive ‘cups’ filled with fungus. Invasion may extend into the deeper connective tissue. Hyphae are often seen replacing adnexal structures, filling skin glands and follicles. Curved conidia may be present on the surface of infected skin. Cellular inflammation is usually not present during hibernation but can become intense following emergence from hibernation as the bat becomes active and euthermic.

Diagnosis: Although gross lesions can be suggestive of WNS, confirmation of WNS requires histopathologic visualization of lesions (cupping erosion of dermis) with PAS stain and confirmation of presence of *Pd* by real-time polymerase chain reaction (PCR) analysis of wing tissue or a swab sample from the wing. Molecular detection of *Pd* has also been demonstrated in guano collected from bats. Alternatively, *Pd* may be cultured from samples using fungal media (Sabouraud dextrose agar or dextrose-peptone-yeast extract agar) and incubated at cold temperatures (approximately 5 - 10 °C) for six weeks until calling the culture negative. Curved conidia produced by *Pd* are morphologically distinct from other fungi generally found on bats, but molecular identification of the isolate by PCR is necessary for definitive confirmation. Biopsies of wing tissue can be taken as non-lethal samples (*vide infra*) for histological examination, but should be guided by visible signs to increase sensitivity. Ultraviolet light can be used as a screening tool to assist with targeted specimen selection in the field; the cupping dermal erosions have been associated with fluorescence under long-wave (368-385 nm) UVA light. The unknown specificity of UV fluorescence precludes this technique from being diagnostic.

Material required for laboratory analysis:

Non-lethal swab samples of the wing skin surface can be collected to test for the presence of *Pd*. However, confirmation of WNS requires histopathological examination of skin. Skin tissue from the wing and/or muzzle

WHITE NOSE SYNDROME: Cutaneous Invasive Ascomycosis in Hibernating Bats

can be submitted for PCR analysis, fungal culture, and histopathology (PAS stain). Areas submitted should preferably demonstrate white fungal growth or abnormal appearance. The use of long-wave UVA light can aid in identifying areas likely to be affected, particularly when non-lethal sampling is desired. A 3-5 mm biopsy of wing tissue may be submitted for histopathology if analysis of a whole carcass is not available or otherwise not an option. Although tape impressions of fungal growth on bats can be mounted on glass slides to search for conidia characteristic of *Pd*, suggestive samples should be confirmed by PCR and histopathology.

Relevant diagnostic laboratories:

Samples known or suspected to harbor viable *Pd* should, at minimum, be handled in a biosafety cabinet in a Biosafety Level-2 laboratory. Guidelines for decontamination of personal and equipment should be followed. <http://whitenosesyndrome.org/topics/decontamination>

Treatment and Management: At this time, the only effective treatment for WNS is supportive care of homeothermic bats. Natural recovery of free-ranging bats that survive infection during hibernation and subsequently clear all signs of disease has been documented. However, in wild free-flying bats, wing damage may prevent successful foraging, causing additional mortality. Disease management options are still in the discovery phase, including use of vaccination, antifungal compounds, ultraviolet light and biologic control. Although there has been some demonstration of effectiveness against *Pd* in the laboratory, safety, efficacy and transferability of this research to wild bats, as well as potential ecological impacts of these management actions, have not been determined. At this time, improving survival of bats outside of hibernation is a management action directed at population recovery in the face of WNS.

Prevention and control: Current prevention and control strategies focus on biosecurity and restricting access to hibernacula (primarily caves and mines) to limit movement of people and contaminated equipment between hibernacula and other sites used by bats. To support this effort, a national cave access advisory and standardized decontamination protocols have been developed (see www.whitenosesyndrome.org). Other studies assessing the utility of artificial hibernacula, chemical and biocontrol agents, and vaccination are currently in progress.

Suggested disinfectant for housing facilities: To minimize the spread of *Pd*, decontamination protocols should be followed whenever moving bats or equipment that may have been exposed to *Pd* or contaminated environments (see <http://whitenosesyndrome.org/topics/decontamination>). Biosecurity and decontamination procedures should also be implemented for rehabilitation facilities to limit spread of *Pd* between individuals and geographic areas following release of bats back into the wild. To date, there are no disinfection methods that are considered safe and effective for natural hibernacula.

Notification: At this time, notification of WNS or detection of the *Pd* fungus is voluntary. Reports of WNS observations can be sent to the state wildlife resources agency, the U.S. Fish and Wildlife Service, or the USGS National Wildlife Health Center. Instructions for reporting mortality events to the USGS can be found here: http://www.nwhc.usgs.gov/mortality_events/reporting.jsp. For inclusion of information on the WNS Occurrence Map (<https://www.whitenosesyndrome.org/resources/map>), report updates to: GS_wnsmap@usgs.gov

Measures required under the Animal Disease Surveillance Plan: There are no national requirements. See section above (Notification) for recommendations for reporting pathogen detections and disease observations. (see Bat Submission Guidelines: http://www.nwhc.usgs.gov/disease_information/white-nose_syndrome/)

Measures required for introducing animals to infected animal: Not recommended.

Conditions for restoring disease-free status after an outbreak: None. Underground sites where *Pd* has been detected are considered permanently contaminated until an effective environmental treatment method is identified.

WHITE NOSE SYNDROME: Cutaneous Invasive Ascomycosis in Hibernating Bats

Experts who may be consulted:

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References:

An updated full WNS bibliography can be found at: <https://www.whitenosesyndrome.org/wns-bibliography>

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YABAPOXVIRUS

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Primates, including human	Accidental inoculation, insect vectors, body fluids, wounds. Humans infected via skin wounds.	Benign growths on the face and limbs (histiocytoma) which may reach several cm in diameter	Mild to severe	Supportive. Usually spontaneous regression in 3-6 weeks	Careful handling of nonhuman primates. Disinfection of fomites and vector control.	Yes
Fact Sheet compiled by: E. Marie Rush						
Sheet completed on: 3 December 2010; 25 March 2013; May 1 2018						
Fact Sheet Reviewed by: Marc Valitutto						
Susceptible animal groups: Primates, human and non-human						
Causative organism: Yabapoxvirus (genus <i>Yatapoxviridae</i>)						
Zoonotic potential: Yes						
Distribution: Western Africa (originated in Yaba, Nigeria)						
Incubation period: Unknown, but clinical signs can appear within days of inoculation						
Clinical signs: In non-human primates, subcutaneous tumors begin as small erythematous areas, but can quickly proliferate once the histiocytes become infected. The infected animal develops a high titer during tumor growth, and regression of the tumor is likely caused by <i>in vivo</i> cytopathic effects of virus. Signs in humans are similar to nonhuman primates. Lesions typically regress spontaneously within 3-6 weeks. Pruritus may accompany lesions. This disease is different from Yaba-like disease virus, which is in the same genus <i>Yatapoxviridae</i> .						
Post mortem, gross, or histologic findings: Grossly apparently subcutaneous tumors that when biopsied, show large pleomorphic histiocytic cells loosely arranged in a vascular network.						
Diagnosis: History of direct or indirect contact with non-human primates or transport from and travel to west Africa, ELISA, PCR, histopathology of tumors, EM						
Material required for laboratory analysis: Serum, tissue for histopathology or EM.						
Relevant diagnostic laboratories: This is an uncommon disease, but has been noted in North American collections. Most laboratories that process non-human primate samples can either run the PCR for this virus or can direct personnel accordingly to an appropriate laboratory facility for testing of samples. Histopathology and EM can be done at most laboratories that normally process tissues and have the capabilities for these procedures.						
Treatment: Supportive – spontaneous resolution usually in ~3-6 weeks						
Prevention and control: Avoid contact with primates that have had potential exposure. Proper quarantine and testing of animals with history of exposure or recent shipment from west Africa. Humans should keep all skin wounds cleaned, bandaged and covered when working with non-human primates. Thorough disinfection of all potential fomites in housing areas for primates in collections and protection of animal care staff through education and proper clothing and protective wear (gloves, long sleeves). Vector control.						
Suggested disinfectant for housing facilities: Detergents, hypochlorite, alkalis, Virkon® and glutaraldehyde.						
Notification: Public health officials may need to be notified if zoonotic transmission occurs, depending on the state.						
Measures required under the Animal Disease Surveillance Plan: Currently none						

YABAPOXVIRUS

Measures required for introducing animals to infected animal: Do not introduce animals with clinical disease (active or resolving pustules/lesions) to non-infected or new animals. Allow resolution of all lesions completely prior to introduction and follow proper quarantine measures for individual facility.

Conditions for restoring disease-free status after an outbreak: Condition typically spontaneously resolves within weeks with supportive care. Treatment of any secondary infections should assist in wound healing. Immunosuppressed animals may be more susceptible to infection and secondary disease and complications. Proper disinfection of animal area and fomites should be done following an outbreak or care of an infected animal prior to housing new animals in the area.

Experts who may be consulted:

Centers for Disease Control and Prevention

Poxvirus and Rabies Branch, Division of High-Consequence Pathogens and Pathology 1600

Clifton Rd

Atlanta, GA 30333

800-CDC-INFO

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YELLOW FEVER

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Non-human primates; humans	Mosquito bites	Bleeding diathesis, fever, hepatopathy, death	Mild to severe to fatal.	Supportive	Mosquito control, vaccination	Yes

Fact Sheet compiled by: Ellen Wiedner

Sheet completed on: 11 November 2010; updated 1 March 2013

Fact Sheet Reviewed by: Jim Wellehan; Alan Barrett; Ramiro Isaza

Susceptible animal groups:

Africa: *Colobus*, *Cercopithecus*, *Cercocebus*, *Papio*, *Galago*, *Pan*

South America: *Alouatta*, *Aotus*, *Saguinus*, *Ateles*, *Callicebus*, *Cebus*, *Saimiri*

Causative organism: Family Flaviviridae, Genus *Flavivirus* at least 7 genotypes. Mosquito genera vectors include *Aedes*, *Haemagogus*, and *Sabethes*.

Zoonotic potential: Yes. Sylvatic cycle has monkey reservoir; transmission to humans occurs when virus-infected mosquito bites a person. Urban cycle involves man and mosquitoes only.

Distribution: Disease has been eliminated in North America and Europe but it still occurs in tropical South America, Caribbean, and Sub-Saharan Africa.

Incubation period: In humans, 3-6 days; in monkeys, 2-3 days.

Clinical signs:

New World monkeys: fever, leukopenia, death

Old World monkeys: none, except in *Galago* which has high mortality rate and may show signs as in New World monkeys. In *Galago*, serum may turn green for 2 to 5 days during period of viremia.

Humans: variable ranging from mild and self-limiting febrile disease to severe hepatitis to fulminant hemorrhagic fever. In humans, mortality rate from up to 50%.

Post mortem, gross, or histologic findings:

New world monkeys: bleeding diathesis, shock, severe hepatocellular necrosis

Diagnosis: Serology: paired serum titers showing four-fold increase in IgG or presence of yellow fever specific IgM. Isolation of virus in tissues, particularly liver, can be performed or PCR identification of viral genome in blood or tissues. Immunohistochemical detection of viral antigen in tissues is possible.

Material required for laboratory analysis: Liver, other organ tissues, whole blood, serum

Relevant diagnostic laboratories:

CDC Arbovirus Diagnostic Laboratory. For details and contact information, refer to:

http://www.cdc.gov/ncidod/dvbid/misc/arboviral_shipping.htm

Treatment: Symptomatic, including fluids, anti-inflammatories, and blood transfusions. Ribavirin has been used in some cases.

Prevention and control: Vaccination is recommended for travelers and for personnel in face of outbreak. (Specific documentation required for movement into and between yellow fever endemic countries per International Health Regulations guidelines). Yellow fever 17D vaccine is a live attenuated vaccine. Mosquito control necessary in primate facilities.

Suggested disinfectant for housing facilities Mosquito control required.

Notification: As eliminated, it is a reportable disease and state health department should be contacted. All yellow fever cases must be reported to WHO within 24 hours of confirmation.

YELLOW FEVER

Measures required under the Animal Disease Surveillance Plan: None.

Measures required for introducing animals to infected animal: The disease is arthropod borne. However, infected animals can infect mosquitoes and contribute to the transmission cycle. Thus, insect control is essential. Experimentally, contact with contaminated blood can infect some primate species, so do not introduce animals to each other when they are clinically sick.

Conditions for restoring disease-free status after an outbreak:: Outbreak control requires elimination of infected mosquitoes and their larvae.

Experts who may be consulted

Centers for Disease Control & Prevention
Division of Vector-Borne Diseases
Arboviral Diseases Branch
3156 Rampart Road
Ft. Collins, CO 80521
(970) 221-6400

World Health Organization
Department of Pandemic and Epidemic Diseases
Avenue Appia 20
1211 Geneva 27
Switzerland
<http://www.who.int/csr/disease/en/>

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PLAGUE (*Yersinia pestis*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Rodents, human and non-human primates, felids, mustelids, lagomorphs	1) Vector-borne >30 species of fleas, possibly lice and ticks 2) Aerosol 3) Direct contact with infected tissues, bite from infected animal 4) Oral - ingestion of infected tissue or feces 5) Fomites	Peracute mortality without signs; swelling, abscess and hemorrhage at inoculation site; lymphadenopathy; lethargy; fever Three classic forms in humans 1) Bubonic 2) Septicemic 3) Pneumonic	Subclinical in resistant species, 80-100% mortality in others	Streptomycin, gentamicin, tetracyclines, ciprofloxacin, sulfonamides	Flea and rodent control program; quarantine prairie dogs and other rodents; personal protective equipment	Yes

Fact Sheet compiled by: Rae Gandolf

Sheet completed on: 1 January 2011; updated 1 November 2012

Fact Sheet Reviewed by: Tonie E. Rocke, Mark Drew, Genevieve Vega Weaver

Susceptible animal groups: > 200 different species of mammals including humans, rodents, felids, and black-footed ferrets. Between 30 and 40 rodent species are considered important as reservoir hosts. In the literature, susceptible species have commonly been grouped into four categories: (1) enzootic hosts (California voles, deer mice, grasshopper mice) (2) epizootic hosts (prairie dogs, ground squirrels) (3) resistant non-rodent hosts (coyotes, badgers, domestic dogs, ungulates) and (4) susceptible non-rodent hosts (bobcats, mountain lions, Canada lynx, black-footed ferrets, lagomorphs, primates including humans, domestic cats). More recently, however, the distinction between enzootic and epizootic host species has become less clear; it appears that both cycles can occur in the same species.

Causative organism: *Yersinia pestis* is a small, non-spore forming Gram-negative facultative anaerobic coccobacilli in the Enterobacteriaceae family consisting of one serotype that is divided into four biovars: Antiqua, Medievalis, Orientalis, and Microtus.

Zoonotic potential: Yes

Primary disease concerns: Urban human plague pandemics may occur; sylvatic plague is a major threat to black-footed ferret and prairie dog populations; felids (domestic cats, Canada lynx) are susceptible; they can develop a highly contagious form of the disease (pneumonic plague) and can further represent a health threat to people who come in contact with them.

Distribution: *Y. pestis* has a patchy global distribution in semi-arid regions of Africa, Middle East, Asia, and South America. In North America, it occurs in the western one third of the continent from Canada to Mexico. Plague is also divided into two epidemiologic forms: sylvatic and urban.

Incubation period: 1-6 days in humans; 1-4 days in felids; 3-7 days in black-footed ferrets

Clinical signs: Rodent species, such as prairie dogs, frequently present with peracute mortality and without

PLAGUE (*Yersinia pestis*)

demonstrating signs of disease. In all animal species affected, swelling and hemorrhage can develop at the inoculation site and progress to abscessation. Other signs may include fever, depression and lymphadenopathy. In resistant species, such as canids and some rodents, infection may be subclinical or mild. Wild ungulates (mule deer and the black-tailed deer) have been reported to acquire ocular plague characterized by keratoconjunctivitis, endophthalmitis and panophthalmitis.

In humans, there are three classic forms of plague: bubonic, septicemic, and pneumonic. Similar signs may be seen in animals, although this terminology is generally restricted to human and felid cases:

1) **Bubonic**: fever, anorexia, lethargy, lymphadenopathy, draining lymph nodes, abscesses, cellulitis, oral ulceration, vomiting, diarrhea, ocular discharge, dehydration, and weight loss. If acquired via ingestion, severe pharyngitis and tonsillitis can occur. If not treated, this form can progress to the septicemic or pneumonic form.

2) **Septicemic**: shock, DIC, respiratory distress due to secondary pneumonia. No obvious involvement of the lymph nodes in primary septicemic plague may be seen but the other signs of bubonic plague may be present.

3) **Pneumonic**: dyspnea, hemoptysis, cough, neurologic signs. This form can occur via primary inhalation of the organism or following blood-borne dissemination to the lungs from bubonic or septicemic plague.

Post mortem, gross, or histologic findings: Lesions are variable depending on host susceptibility and route of infection, and may include: large numbers of the organism in lesions; necrotic foci in liver, spleen, lungs, and other internal organs; hepatomegaly and splenomegaly; enlarged, hemorrhagic and necrotic lymph nodes; soft tissue abscesses with cellulitis; hemorrhagic gastritis and colitis; interstitial pneumonia, pulmonary edema, and pulmonary hemorrhage; keratoconjunctivitis, panophthalmitis, and endophthalmitis; subcutaneous vascular hemorrhage.

Diagnosis: Presumptive diagnosis can be made by identifying the characteristic organism in stained samples of lymph node aspirates or draining lesions. *Yersinia pestis* has a bipolar or safety pin-like staining pattern with Wright-Giemsa or Wayson stain and will be positive with an immune-fluorescence stain for the presence of *Y. pestis* F1 antigen. Definitive diagnosis is made by *Y. pestis* isolation, rapid immunoassays, PCR, and paired sera demonstrating a four- fold titer increase to *Y. pestis* F1 antigen using agglutination testing. Differentials include bacterial infections such as *Pasteurella*, *Franciella tularensis*, *Y. pseudotuberculosis*, and *Y. enterocolitica*.

Material required for laboratory analysis: Blood, nasal/oral swabs, lymph node aspirates, swabs of draining lesions, transtracheal aspirates, tissue samples from liver, spleen, lungs, and lymph nodes

Relevant diagnostic laboratories: Plague diagnosis should be conducted by state public health laboratories or the CDC under Biosafety level-2 practices. Contact the laboratory before collecting samples.

Treatment: Prompt treatment within 24 hours is necessary for survival from pneumonic plague. *Yersinia pestis* is susceptible to streptomycin, fluoroquinolones, trimethoprim-sulfamethoxazole, and tetracyclines. Personal protective gear consisting of gown, gloves, surgical mask/respirator, and eye protection is important to prevent transmission when treating affected animals. Clinical cases should also be given a flea treatment.

Prevention and control:

- Close parks and campgrounds during plague outbreaks to prevent transmission to humans from rodents.
- Quarantine any wild caught rodents, including prairie dogs, for at least two weeks and treat all animals with an insecticide. Flea and rodent control programs are critical in facilities that are located in plague endemic regions.
- Insecticides like deltamethrin and flea growth regulators like pyriproxyfen can be sprayed into prairie dog burrows to control flea populations to slow or stop outbreaks.
- Private ownership of prairie dogs is restricted or prohibited in some states in the U.S. Interstate shipment in

PLAGUE (*Yersinia pestis*)

the U.S. is regulated by the Center for Disease Control.

- Personal protective gear should be used when handling any potential cases including during post-mortem examinations.
- An F1-V fusion protein vaccine for subcutaneous injection is used in black-footed ferrets. An oral vaccine has been recently developed for use in prairie dogs and appears to confer better immunity than the subcutaneous vaccine.

Suggested disinfectant for housing facilities: 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, iodines, phenolics, formaldehyde, moist heat (121° C for at least 15 min), dry heat (160-170° C for at least 1 hour).

Notification: Nationally notifiable infectious disease. Report cases to the CDC.

Measures required under the Animal Disease Surveillance Plan: Reportable disease

Measures required for introducing animals to infected animal: Not recommended. Potential carrier animals should be screened for disease before introduction, and diseased animals must be quarantined during curative course of treatment.

Conditions for restoring disease-free status after an outbreak: *Yersinia pestis* is endemic to certain regions of the world. Sporadic and seasonal outbreaks occur in endemic regions. Within a limited environment such as a zoological facility, elimination of the rodent and flea population, along with proper disposal of infected tissues is critical to eliminating disease.

Experts who may be consulted:

Tonie E. Rocke, PhD, Epizootiologist
National Wildlife Health Center, 6006 Schroeder Rd., Madison, WI 53711
608-270-2451

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Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Mammals (including humans), Birds Reptiles Fish	Ingestion of fecal contaminated food and water; ingestion of raw meat and milk; blood transfusions (humans)	Diarrhea, abdominal pain, fever, weakness, septicemia, weight loss, enlarged lymph nodes, sudden death	Ranges from subclinical to acutely fatal or a chronic wasting form, depending on individual and species	Third generation cephalosporins, fluoroquinolones; supportive therapy	Good hygiene protocols; pest control program; vaccination; minimize stress	Yes

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Sheet completed on: 15 January 2018

Fact Sheet Reviewed by: Lynnette Waugh

Disease Significance: Yersiniosis is considered among the most important diseases of farmed deer in the U.S. as well as New Zealand and Australia. Among zoological facilities in the U.S., major outbreaks with mortalities have occurred among captive antelope, birds and non-human primates, with sporadic events in other species such as a cougar, lion and Siberian tiger. Enzootics have affected free-ranging musk ox in Canada, and brown hares and hedgehogs in Europe. Yersiniosis is also zoonotic; human exposure is typically foodborne.

Susceptible animal groups: *Yersinia pseudotuberculosis* has been detected in >110 species including humans, other mammals (squirrels and other rodents, non-human primates, hedgehogs, hares, meerkats, domestic dogs, ruminants, bats, suids and felids), birds (guinea fowl, turkey, collared doves, parrots), reptiles, and fish. Rodents, wild boar, deer, insects, and wild birds are believed to be reservoirs; however, there has been some debate about their exact role in transmission. In *Amazona* spp. parrots, hemosiderosis may predispose to systemic infection with *Y. pseudotuberculosis* after enteric disease. Outbreaks occur in farmed 4-8 mo old deer in fall/winter in the U.S. *Yersinia pseudotuberculosis* can also survive for months to years in the soil, water and vegetation. Sensitive species and groups include: callitrichids, capybaras, agouti, mara, turacos, toucans, lemurs, guenons, fruit bats, squirrels, and deer.

Yersinia enterocolitica, in contrast, is a less common cause of yersiniosis and has only been reported to cause disease in a few species of non-human primates such as the African Green monkey, chinchillas, guinea pigs, domestic pigs, wild boars, deer, dogs, cats, and humans. Young, old, immunosuppressed, and animals with chronic liver illness appear to be most susceptible to severe disease associated with both *Yersinia* species.

Alpine ibex have been identified as a potential carrier of pathogenic *Y. enterocolitica*. Pathogenic *Y. enterocolitica* has also been detected in asymptomatic dogs, cats, Djungarian hamsters, pigs, cattle, goats, rats, mice, voles, shrews, mongooses and beavers, and a bird species, the dunnoek. Carriers have the potential to cause water and soil contamination as well as direct zoonotic transmission.

Outbreaks of *Y. pseudotuberculosis* most commonly occur during winter months, due to stress and overcrowding as well as the enhanced virulence factors of the organism at lower temperatures. In contrast, *Y. enterocolitica* occurs more commonly in the summer and autumn.

Causative organisms: *Yersinia pseudotuberculosis* and *Y. enterocolitica* are non-spore forming Gram-negative aerobic coccobacilli belonging to the Enterobacteriaceae family. They are facultative intracellular bacteria. *Yersinia pseudotuberculosis* consists of 15 serotypes plus additional subtypes, while *Y. enterocolitica* has over 60 serotypes of which four (O3, O5/27, O8, and O9) are believed to be pathogenic.

Zoonotic potential: Yes

Distribution: Worldwide except Antarctica, especially in temperate climates. Highly prevalent in Europe.

Incubation period: < 10 days

Clinical signs: Disease is predominantly gastrointestinal, although extraintestinal yersiniosis also occurs.

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Disease may be acute, subacute or chronic. Signs vary by species and individual, but most typically consist of gastroenteritis. Severe cases progress to septicemia. Signs may include lethargy, anorexia, green to bloody diarrhea, respiratory distress, incoordination, emaciation, and sudden death. Other forms of the disease include: abortion and mastitis in ungulates; chronic disease resulting in wasting syndrome and enlarged, palpable mesenteric lymph nodes in various species; skin rash, desquamation, erythema nodosum and arthritis caused by certain serotypes; granulomatous conjunctivitis in dairy goats; and appendicitis, gastroenteritis, abdominal pain and reactive arthritis in humans. A carrier state can also develop. Clinical symptoms are similar to salmonellosis.

Post mortem, gross, or histologic findings: *Yersina pseudotuberculosis* can cause ulcerative enterocolitis, hepatomegaly and splenomegaly, multifocal necrosis seen as white-gray nodules on the liver and spleen and possibly the lungs and kidneys (organisms are seen in the lesions), interstitial pneumonia, enlargement and abscessation of abdominal lymph nodes and adhesive peritonitis. *Yersinia enterocolitica* often results in lesions in the lymphoid tissue of the head and neck, particularly the tonsils and submandibular lymph nodes. Subclinical cases may demonstrate minimal gross and histologic changes.

Diagnosis: Diagnosis is based on characteristic gross and histopathologic lesions with the presence of gram-negative coccobacilli, identification from bacterial culture (cold enrichment), and identification using conventional or real-time PCR. A commercially available IgM ELISA for domestic pigs has been used with muscle and tonsillar tissue. Serotyping can be done by slide agglutination or PCR. Isolates can be further characterized using pulse field gel electrophoresis (PFGE). Rule out similar diseases including salmonellosis by culture. *Yersinia enterocolitica* O:9 shares common antigenic epitopes with *B. abortus* and is known to cross-react in diagnostic testing of African buffalo.

Material required for laboratory analysis: The organism is most reliably cultured from organs demonstrating lesions, particularly liver and spleen, but also lungs, mesenteric lymph nodes, and intestines. Blood culture is used in humans and can be used in non-human primates in cases of suspected septicemia. Culture may also be performed on feces and postmortem tissues showing lesions. However, shedding of the organism can be intermittent; therefore, fecal culture is not always reliable. For PCR: Rectal or cloacal swab, 0.5 g feces, 0.5 g fresh, frozen or fixed tissue, or (*Y. pseudotuberculosis*) 0.5 ml whole blood in EDTA (purple top) or ACD (yellow top) tube.

Relevant diagnostic laboratories: Any diagnostic laboratory with Biosafety Level 2 practices that can perform bacterial culture and sensitivity. Care should be taken because of the zoonotic potential. *Yersinia pseudotuberculosis* and *Y. enterocolitica* do not grow well on routine culture media, therefore submitted samples should indicate that these organisms are suspected. For more rapid detection, PCR is available: Zoologix (B0062) for qualitative ultra-sensitive detection of *Yersinia pseudotuberculosis*, and (B0073) for detection of *Yersinia enterocolitica*.

Treatment: There has been little success with treatment of clinical cases. Prophylactic treatment of animals in contact with an individual demonstrating clinical signs is recommended. Antibiotic treatment should be based on sensitivity. Although different strains have demonstrated variable sensitivities, most strains are susceptible to third generation cephalosporins, fluoroquinolones and chloramphenicol. There have been reports of some resistance by certain strains, particularly those of *Y. enterocolitica*, to amoxicillin-clavulanic acid, ampicillin, tetracyclines, sulfonamides, macrolides, florfenicol, and fluorquinolones. Multi-drug resistant strains of *Y. enterocolitica* have been found in humans and pigs. In patients with chronic liver lesions, long term antibiotic treatment might be needed. Fluid therapy should be administered as dehydration is a common development.

Prevention and control: The bacteria can survive in animal and environmental reservoirs. Outbreaks of are associated with stressors such as cold and wet weather, decreases or changes in food availability, overcrowding, intestinal parasitism, or animal capture. Measures should therefore be taken to minimize these stressors. Affected animals should be isolated and enclosures should be disinfected. In some cases, euthanasia of groups of animals may be necessary. Preventive measures include: implementing a rodent and bird control program; practicing good hygiene including disinfection, changing substrate, removing contaminated or old food and water from enclosures; minimizing stress, competition, and overcrowding in enclosures; and avoiding raw meat in non-human primates.

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A killed whole cell vaccine for *Y. pseudotuberculosis* (Pseudovac®, Department of Veterinary Pathology, Utrecht University, The Netherlands) is available and used mainly in European zoos before the winter, and a killed vaccine (Yersiniavax®, Intervet) used for cervid farms in New Zealand. A new vaccine of a live, attenuated strain (IP32680) of *Y. pseudotuberculosis* administered orally has shown to provide adequate protection against severe infection in experimentally infected guinea pigs and mice and has demonstrated superior efficacy over Pseudovac®. The development of a recombinant vaccine for *Y. pseudotuberculosis* is in the research phase but has also had positive results.

Suggested disinfectant for housing facilities: 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, iodines, phenolics, formaldehyde, moist heat (121° C for at least 15 min), dry heat (160-170° C for at least 1 hour). Be aware that organic material, such as soils, plant debris, blood, manure, can inactivate some disinfectants (e. g. chlorine-based products). Removal of organic material should be conducted prior to disinfection.

Notification: Notification of public health officials is required in human cases

Measures required under the Animal Disease Surveillance Plan: None

Measures required for introducing animals to infected animal: Not recommended. In some cases, euthanasia of symptomatic individuals may be warranted to avoid a carrier state. Following exposure, only animals free of clinical signs and with multiple negative cultures should be allowed to comingle with new animals.

Conditions for restoring disease-free status after an outbreak: Since the organism is ubiquitous in the environment and appears sporadically in some zoos, it is problematic to designate an institution disease-free. Yersiniosis appears to be endemic in some European zoos.

Experts who may be consulted:

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