## PRESENTATION SCHEDULE

**MONDAY *morning*, August 10**

**8:00 - 8:20 WELCOME AND OPENING COMMENTS**
**WDA President: Bob McLean**
**Conference Chairperson: Tonie Rocke**

**8:20 - 9:50 FORUM: "IS WILDLIFE DISEASE RESEARCH MEETING THE NEEDS OF RESOURCE AGENCIES?"**
**Moderator: Tom Yuill**
**Participants: Michael Anderson, Chief Scientist, Institute for Wetland and Waterfowl Research, Ducks Unlimited, Canada**
**Steve Miller, Administrator, Land Division, Wisconsin DNR**
**William Molini, Administrator, Nevada Division of Wildlife**
**John Rogers, Deputy Director-Line, USFWS**
**Steve Wilds, Chief, Migratory Birds, USFWS-Region 3**

**9:50 - 10:10 BREAK**

**10:10 - 10:40 FORUM (Continued)**

**10:40 - 11:25** ***CARLTON M. HERMAN LECTURE***
**(1) RABBIT HAEMORRHAGIC DISEASE IN AUSTRALIA.**
**BRIAN D. COOKE**

**11:25 - 11:45** ***WDA STUDENT RESEARCH RECOGNITION AWARD***
**(2) BRUCELLOSIS IN ELK: STUDIES OF EPIZOOTIOLOGY AND CONTROL.**
**WALTER E. COOK, ELIZABETH S. WILLIAMS**

**11:45 - 12:45 LUNCH**

**MONDAY *afternoon*, August 10**

### TERRY AMUNDSON STUDENT PRESENTATION COMPETITION
**Moderator: Todd O'Hara**

**12:45 - 1:00 (3) EXERTIONAL MYOPATHY IN RECENTLY CAPTURED NORTH AMERICAN RIVER OTTERS (*LONTRA CANADENSIS*) FROM NEW YORK.**
**BARRY K. HARTUP, GEORGE V. KOLLIAS, MATTHEW C. JACOBSEN, BETH A. VALENTINE, KEVIN R. KIMBER**

**MONDAY *afternoon*, August 10 (Continued)**

**1:00 - 1:15** **(4) PREVALENCE OF HEMATOZOA IN SORAS (*PORZANA CAROLINA*) AND VIRGINIA RAILS *(RALLUS LIMICOLA*) IN SOUTHEASTERN WISCONSIN.**
**KATHERINE LEWANDOWSKI, CHRISTINE RIBIC, JENNIFER SKOLODA, LINDA SULLIVAN**

**1:15 - 1:30** **(5) THE EFFECT OF BOVINE BRUCELLOSIS AND TUBERCULOSIS ON PREGNANCY RATES IN WOOD BISON: PRELIMINARY RESULTS FROM WOOD BUFFALO NATIONAL PARK, CANADA.**
**DAMIEN O. JOLY, FREDERICK A. LEIGHTON, FRANÇOIS MESSIER**

**1:30 - 1:45** **(6) CAN THE ASIAN TIGER MOSQUITO, *AEDES ALBOPICTUS*, FACILITATE THE EVOLUTION OF CALIFORNIA SEROGROUP BUNYAVIRUSES IN NORTH AMERICA?**
**LI-LEN CHENG, JUAN D. RODAS, THOMAS M. YUILL, BARBARA A. ISRAEL**

**1:45 - 2:00** **(7) BIOSAFETY STUDY OF *BRUCELLA ABORTUS* STRAIN ARS/1 VACCINATION IN BISON BULLS: EVALUATION OF IMMUNOLOGIC RESPONSES.**
**STEVEN C. OLSEN, JACK RHYAN, THOMAS GIDLEWSKI, MITCHELL V. PALMER, AMY M. HAYEK JONES**

**2:00 - 2:15** **(8) A VACCINE EFFICACY TRIAL OF *BRUCELLA ABORTUS* STRAIN RB51 IN BISON.**
**MATTHEW D. EDMONDS, DAVID HUNTER, MICHAEL GILSDORF, JOE TEMPLETON, DONALD DAVIS, PHILIP ELZER**

**2:15 - 2:30** **(9) RECENT ESTABLISHMENT OF AN ISOLATED ENDEMIC POPULATION OF *IXODES SCAPULARIS*, THE VECTOR OF LYME BORRELIOSIS, AT POINT PELEE, ONTARIO, CANADA.**
**L.R. LINDSAY, T.H. AKWAR, I.K. BARKER, M.D. RAVYN**

**2:30 - 3:00** **BREAK**

## STUDENT PRESENTATION COMPETITION (Continued)
## Moderator: Ellis Greiner

**3:00 - 3:15** **(10) MULTIPLE ENDOCRINE NEOPLASIA TYPE IIA IN A RED WOLF (*CANIS RUFUS*).**
**FELICIA B. NUTTER, FRANK J. GEOLY**

**MONDAY *afternoon*, August 10 (Continued)**

**3:15 - 3:30** **(11) COMPARING SEROLOGICAL RESPONSES OF CAPTIVE BIGHORN SHEEP (*OVIS CANADENSIS*) TO A MULTIVALENT *PASTEURELLA HAEMOLYTICA* SUPERNATANT VACCINE DELIVERED VIA HAND INJECTION, REMOTE IMPLANTATION, OR ORAL SUSPENSION.**
**HEATHER J. MCNEIL, MICHAEL W. MILLER, HARM HOGENESCH, JENNIFER A. CONLON, IAN K. BARKER, PATRICIA E. SHEWEN**

**3:30 - 3:45** **(12) DEVELOPMENT OF A MUSKOX LUNGWORM, *UMINGMAKSTRONGYLUS PALLIKUUKENSIS*, IN THE SLUG, *DEROCERAS LAEVE*, UNDER ARCTIC FIELD CONDITIONS WITH COMMENTS ON THE IMPACT OF CLIMATE CHANGE.**
**SUSAN J. KUTZ, ERIC P. HOBERG, JOHN S. NISHI, LYDDEN POLLEY**

**3:45 - 4:00** **(13) ASSESSING HEALTH STATUS OF FREE-RANGING PSITTACINES IN THE BOLIVIAN AMAZON.**
**CATHERINE SOOS, BRUCE HUNTER, DAVID N. PHALEN, LAUREL NEUFELD, CHARLES A. MUNN**

**4:00 - 4:15** **(14) HEARTWORMS AND LUNGWORMS IN ILLINOIS' CANIDS AND THEIR POSSIBLE EFFECT ON COYOTE CONDITION AND REPRODUCTION.**
**DAVID G. GREGORY, TOM NELSON, JEFF LAURSEN**

**4:15 - 5:00** **(15) EXPERIMENTAL INFECTIONS OF *PARELAPHOSTRONGYLUS TENUIS* IN WHITE-TAILED AND RED DEER.**
**MICHAEL S. DUFFY, MICHAEL D. B. BURT**

**TUESDAY *morning*, August 11**

## *SYMPOSIUM* - ALGAL BIOTOXINS
### Moderator: Lynn Creekmore

**8:00 - 8:30** **(16) HARMFUL ALGAL BLOOMS IN COASTAL AND ESTUARINE WATERS: CURRENT STATUS OF AN EXPANDING PROBLEM.**
**DONALD M. ANDERSON**

**8:30 - 9:00** **(17) CYANOBACTERIAL TOXINS: HEALTH EFFECTS AND RISK ASSESSMENT.**
**WAYNE W. CARMICHAEL**

**9:00 - 9:30** **(18) THE TOXIC *PFIESTERIA* COMPLEX.**
**JOANN M. BURKHOLDER, HOWARD B. GLASGOW**

**9:30 - 10:00** **(19) EXPOSURE OF MARINE ANIMALS TO BIOTOXINS: IS THERE A CHRONIC DISEASE PROBLEM?**
**JAN H. LANDSBERG**

**TUESDAY *morning*, August 11 (Continued)**

**10:00 - 10:30 BREAK**

**10:30 - 11:00** **(20) HISTOPATHOLOGIC INVESTIGATIONS OF LESIONS IN FISHES FROM CHESAPEAKE BAY TRIBUTARIES.**
**VICKI S. BLAZER, CHRISTINE DENSMORE**

**11:00 - 11:15** **(21) BREVETOXIN AS CAUSE OF SUMMER MORTALITY IN COMMON MURRES (*URIA AALGE)* IN CALIFORNIA.**
**DAVID A. JESSUP, JACK AMES, GREG BOSSART, JAMES HILL, MELISSA CHECHOWITZ, ANDREW DEVOGELAERE**

## INFORMATICS & TECHNIQUES
**Moderator: JoAnn Paul-Murphy**

**11:15 - 11:30** **(22) ACAD (1.0): THE ANIMAL CAPTURE AND ANESTHESIA DATABASE.**
**KEITH BEHELER-AMASS, DAVID BRUNSON**

**11:30 - 11:45** **(23) LONDON WATERFOWL PROJECT.**
**DEBRA BOURNE, SUZANNE BOARDMAN, JOSHUA DEIN**

**11:45 - 12:00** **(24) DISEASE MONITORING IN CYBERSPACE: AN INTERNET-BASED SURVEY FOR CONJUNCTIVITIS IN HOUSE FINCHES.**
**BARRY K. HARTUP, GEORGE V. KOLLIAS, ANDRE A. DHONDT**

**12:00 - 12:15** **(25) EVALUATION OF A PORTABLE AUTOMATED SERUM CHEMISTRY ANALYZER FOR ASSESSMENT OF HEALTH STATUS OF HARLEQUIN DUCKS, *HISTRIONICUS HISTRIONICUS*, IN THE FIELD.**
**MICHAEL K. STOSKOPF, DANIEL M. MULCAHY, DANIEL ESLER**

**12:15 - 1:15 LUNCH**

**TUESDAY *afternoon*, August 11**

**1:15 - 1:35** ***AMERICAN ASSOCIATION OF WILDLIFE VETERINARIANS: CUTTING EDGE SPEAKER***
**(26) CONTROL OF PRION DISEASES THROUGH GENETICS AND LIVE ANIMAL TESTING: VARIATION BETWEEN SHEEP AND CERVIDS.**
**KATHERINE O'ROURKE**

**TUESDAY *afternoon,* August 11 (Continued)**

## CHRONIC WASTING DISEASE
**Moderator: Sarah Shapiro Hurley**

**1:35 - 1:50** **(27) EPIDEMIOLOGY OF CHRONIC WASTING DISEASE IN NORTHEASTERN COLORADO DEER AND ELK POPULATIONS.**
**MICHAEL W. MILLER, TERRY R. SPRAKER, ELIZABETH S. WILLIAMS**

**1:50 - 2:05** **(28) SURVEY FOR CHRONIC WASTING DISEASE IN CAPTIVE AND FREE-RANGING ELK AND DEER IN ALBERTA.**
**M.J. PYBUS , D.K. ONDERKA, S. HONOUR**

**2:05 - 2:20** **(29) THE HOST RANGE OF CHRONIC WASTING DISEASE IS ALTERED UPON PASSAGE IN FERRETS.**
**JASON C. BARTZ, RICHARD F. MARSH, DEBBIE I. MCKENZIE, JUDD M. AIKEN**

## BRUCELLOSIS
**Moderator: Sarah Shapiro Hurley**

**2:20 - 2:35** **(30) BRUCELLOSIS IN YELLOWSTONE NATIONAL PARK BISON (*BISON BISON*): QUANTITATIVE SEROLOGY AND TISSUE LOCALIZATION OF INFECTION.**
**THOMAS J. ROFFE, JACK C. RHYAN, KEITH AUNE, L. MICHAEL PHILO, DARLA R. EWALT, T. GIDLEWSKI**

**2:35 - 2:50** **(31) SAFETY AND EFFICACY OF *BRUCELLA ABORTUS* VACCINE STRAIN RB51 IN CAPTIVE ADULT COW ELK.**
**TERRY J. KREEGER, MICHAEL MILLER, MARGARET WILD, PHILIP ELZER**

**2:50 - 3:05** **(32) BRUCELLOSIS IN EUROPEAN BROWN HARES (*LEPUS EUROPAEUS*) IN DENMARK. A RESERVOIR FOR PORCINE BRUCELLOSIS?**
**HANS H. DIETZ, ERIK RATTENBORG, THOMAS H. ANDERSEN, STEEN B. GIESE**

**3:05 - 3:30** **BREAK**

## CERVIDS
**Moderator: Beth Williams**

**3:30 - 3:45** **(33) DISSEMINATED *ACTINOMYCES PYOGENES* INFECTION IN A WILD FREE-RANGING WHITE-TAILED DEER, AND POSSIBLE EMERGENCE OF A NEW DISEASE SYNDROME IN DEER; FOOTWARTS (PAPILLOMATOUS DIGITAL DERMATITIS/INTERDIGITAL DERMATITIS).**

KERRY BEHELER-AMASS, KATHY STRELOW, KAREN WOODS, NEIL WISELEY

**TUESDAY *afternoon,* August 11 (Continued)**

**3:45 - 4:00** **(34) PREVALENCE AND DISTRIBUTION OF TOXOPLASMOSIS IN URBAN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*).**
**KARMEN M. HOLLIS, LAURA L. HUNGERFORD, J.P. DUBEY, CHRIS ANCHOR, JAMES CHELSVIG**

**4:00 - 4:15** **(35) CYCLIC PATTERNS OF HEMORRHAGIC DISEASE IN GEORGIA WHITE-TAILED DEER.**
**D.E. STALLKNECHT, V.F. NETTLES**

**4:15 - 4:30** **(36) SELECTIN REGULATION IN WHITE-TAILED DEER INFECTED WITH EPIZOOTIC HEMORRHAGIC DISEASE VIRUS.**
**ELIZABETH W. HOWERTH, MOLLY MURPHY, DAVID E. STALLKNECHT**

**4:30 - 4:45** **(37) MODEL HEALTH PROTOCOL FOR IMPORTATION OF WILD ELK FOR RESTORATION.**
**JOSEPH L. CORN, VICTOR F. NETTLES**

**4:45 - 5:00** **(38) HEALTH MONITORING OF FLORIDA'S ENDANGERED KEY DEER.**
**CHARLOTTE F. QUIST, VICTOR F. NETTLES, THOMAS J. WILMERS**

**WEDNESDAY, August 12**

**POSTER SESSION - Authors Available During the Morning and Afternoon Breaks**
**Posters on Display Wednesday 8:00 am - 5:00 pm, Thursday 8:00 am - Noon**

**(39) CASE REPORT: FIRST REPORT OF *LEYOGONIMUS POLYOON* (TREMATODA: STOMYLOTREMATIDAE) IN AMERICAN COOT (*FULICA AMERICANA*) IN NORTH AMERICA.**
**C. L. RODERICK, R. A. COLE, KAY BROCKMAN-MEDERAS**

**(40) ECTOPIC PREGNANCY IN THE BOBCAT.**
**MILLER, D.L., B.J. WOODY, B.D. LEOPOLD, E.D. STYS**

**(41) VACCINE-INDUCED CANINE DISTEMPER IN A GRAY FOX.**
**MILLER, D.L., S. MCKINNEY, B.D. LEOPOLD, R. WILBUR**

**(42) OCULAR ABNORMALITIES IN DUCKS EXPOSED TO HYPERSALINE WATER FROM THE PLAYA LAKES REGION OF SOUTHEASTERN NEW MEXICO.**
P.E. MILLER, L.A. BAETEN, S. SISSLER, F.J. DEIN, R.R. DUBIELZIG, J. PAUL-MURPHY, C.J. MURPHY

**(43) MITIGATING FOR BREEDING BIRDS THAT NEST AT SELENIUM CONTAMINATED EVAPORATION BASINS.**
**ANDREW G. GORDUS, JEFF L. SEAY, SCOTT TERRILL**

WEDNESDAY *Poster Session*, August 12 (Continued)

**(44) GRANULOCYTIC EHRLICHIOSIS IN A CAPTIVE REINDEER AND STRAY CAT FROM WISCONSIN.**
**KURT D. REED, PAUL D. MITCHELL, SANJAY K. SHUKLA, EDWARD A. BELONGIA**

**(45) GENDER VARIATION IN ELEPHANT SEAL (*MIROUNGA ANGUSTIROSTRIS)* BLOOD PARAMETERS DURING THE BREEDING SEASON.**
**PAMELA K. YOCHEM, BRENT S. STEWART, DAVID A. JESSUP**

**(46) THE RACCOON DOG IS A POSSIBLE RESERVOIR FOR CANINE HEARTWORM?**
**MOTONOBU YOSHIDA, KAZUHIDE NAKAGAKI, SADAO NOGAMI, RYUICHIRO MAEDA, HIROMI KATAE, SHIN-ICHI HAYAMA,**

**(47) MONITORING HEALTH OF TROPICAL PELAGIC SEABIRDS IN HAWAII.**
**THIERRY M. WORK, ROBERT A. RAMEYER**

**(48) NECROPSY FINDINGS IN GREAT HORNED OWLS (*BUBO VIRGINIANUS*) FROM ONTARIO, 1990-98.**
**G. DOUGLAS CAMPBELL, CHRISTINE BISHOP**

**(49) EXPOSURE OF EMPEROR GEESE TO SELENIUM IN WESTERN ALASKA.**
**J. CHRISTIAN FRANSON, LYNN H. CREEKMORE, JOEL A. SCHMUTZ, ADA C. FOWLER**

**(50) LEAD POISONING AND CONCENTRATIONS OF SELECTED TRACE ELEMENTS IN COMMON EIDERS FROM FINLAND.**
**TUULA HOLLMÉN, J. CHRISTIAN FRANSON, ROBERT H. POPPENGA, MARTTI HARIO, MIKAEL KILPI**

**(51) NORTHERN FUR SEAL (*CALLORHINUS URSINUS*) STRANDINGS ALONG THE CENTRAL CALIFORNIA COAST OVER TWENTY-THREE YEARS, 1975-1997.**
**DEBORAH FAUQUIER, FRANCES GULLAND, MARTIN HAULENA, LINDA LOWENSTINE**

**(52) HUMAN INTERACTION RELATED INJURIES OBSERVED IN PINNIPEDS AT A REHABILITATION CENTER IN CENTRAL CALIFORNIA 1986-1996.**
**TRACEY GOLDSTEIN, SHAWN JOHNSON, KRISTA HANNI, DEBORAH FAUQUIER, FRANCES GULLAND**

**(53) ISOLATION OF MHC CLASS II BETA GENES AND ITS APPLICATION TO WILDLIFE DISEASE RESEARCH.**
**ANDREW J. PACEJKA, WAYNE K. POTTS**

**(54) THE HAWAIIAN MONK SEAL (*MONACHUS SCHAUINSLANDI*) EPIDEMIOLOGY PLAN: HEALTH AND DISEASE CONSIDERATIONS IN THE MANAGEMENT OF AN ENDANGERED SPECIES.**
**A. ALONSO AGUIRRE**

WEDNESDAY *Poster Session*, August 12 (Continued)

**(55) EXPOSURE TO FISHING TACKLE IN SELECTED AVIAN SPECIES.**
**SCOTT P. HANSEN, J. CHRISTIAN FRANSON, TERRY E. CREEKMORE**

**(56) SELECTED BLOOD PARAMETERS COLLECTED FROM WILD MANATEES IN FLORIDA.**
**SCOTT D. WRIGHT, J. MARK SWEAT, CATHY PERRY**

## WEDNESDAY *morning*, August 12

### *SYMPOSIUM*: AMPHIBIAN DECLINE
### Moderator: Kathy Converse

**8:00 - 8:30** **(57) OVERVIEW OF ISSUES SURROUNDING AMPHIBIAN DECLINE AND THE ROLE OF THE DECLINING AMPHIBIAN POPULATIONS TASK FORCE.**
**GARY S. CASPER**

**8:30 - 9:00** **(58) GLOBAL AMPHIBIAN POPULATION DECLINES & THE ROLE OF INFECTIOUS DISEASES.**
**D. EARL GREEN**

**9:00 - 9:30** **(59) ARE CONTAMINANTS IMPACTING AMPHIBIAN POPULATIONS?**
**ROBIN E. JUNG**

**9:30 - 10:00** **(60) FROG MALFORMATIONS: MORE QUESTIONS THAN ANSWERS.**
**CAROL U. METEYER**

**10:00 - 10:45 BREAK - POSTER SESSION in Inn Wisconsin**

**10:45 - 11:00** **(61) EPIDEMIC EPIDERMAL CHYTRIDIOMYCOSIS IS A CAUSE OF AMPHIBIAN POPULATION DECLINES IN CENTRAL AMERICA.**
**D. EARL GREEN, KAREN R. LIPS, PETER DASZAK**

### AMPHIBIANS
### Moderator: Kathy Converse

**11:00 - 11:15** **(62) A DIE-OFF OF TIGER SALAMANDERS, *AMBYSTOMA TIGRINUM*, CAUSED BY A NEW IRIDOVIRUS.**
**TRENT BOLLINGER, DANNA SCHOCK, JINGHE MAO, V. GREGORY CHINCHAR**

**11:15 - 11:30** **(63) HELMINTH COMMUNITIES IN SIX SPECIES OF AMPHIBIA FROM SOUTHEASTERN WISCONSIN.**
**H. RANDALL YODER**

**WEDNESDAY *morning*, August 12 (Continued)**

**11:30 - 11:45** **(64) A SEASONAL AND COMPARATIVE STUDY OF HELMINTH PARASITES IN WISCONSIN AMPHIBIANS.**
**MATTHEW G. BOLEK**

**11:45 - 12:00** **(65) DEVELOPMENT AND DISTRIBUTION OF IMMUNOGLOBULIN-CONTAINING CELLS IN THE AMERICAN LEOPARD FROG, *RANA PIPIENS*: EMBRYOGENESIS THROUGH ADULTHOOD.**
**LESLIE D. ZETTERGREN, BRIAN J. HALSTEAD**

**12:00 - 1:00 LUNCH**

**WEDNESDAY *afternoon*, August 12**

## TURTLES
## Moderator: Doug Docherty

**1:00 - 1:15** **(66) FIBROPAPILLOMATOSIS IN OLIVE RIDLEY TURTLES (*LEPIDOCHELYS OLIVACEA*).**

**A. ALONSO AGUIRRE, TERRY R. SPRAKER, ANNY CHAVES , LESLIE DU TOIT, WHITNEY EURE, GEORGE H. BALAZS**

**1:15 - 1:30** **(67) FIBROPAPILLOMATOSIS OF GREEN TURTLES IN HAWAII...WHAT'S NEW?**
**THIERRY M. WORK, GEORGE H. BALAZS, JIM CASEY, SANDRA QUACKENBUSH, JOEL ROVNAK, RUFINA CASEY, PAUL BOWSER, DOUG DOCHERTY, MELODY MOORE, ROSE RASKIN , SCOTT WHITTAKER**

**1:30 - 1:45** **(68) IMPACTS OF PCB EXPOSURE ON SNAPPING TURTLE REPRODUCTION, HATCHLING DEVELOPMENT, AND BEHAVIOR.**
**KATHLEEN A. PATNODE, BARB L. BODENSTEIN, RANDALL R. HETZEL, MIEL A. BARMAN**

## BIRDS I
## Moderator: Kerry Beheler-Amass

**1:45 - 2:00** **(69) THE EFFECT OF NEWCASTLE DISEASE AND OTHER CAUSES OF MORTALITY ON THE REPRODUCTIVE SUCCESS OF DOUBLE-CRESTED CORMORANTS.**
**THIJS KUIKEN, FREDERICK A. LEIGHTON, GARY WOBESER**

**2:00 - 2:15** **(70) NEWCASTLE DISEASE VIRUS IN DOUBLE-CRESTED CORMORANTS; CULTURE CHARACTERISTICS AND SEROLOGY.**
**DOUGLAS E. DOCHERTY, RENEE R. LONG, KRISTINA F. JAQUISH, DIANA R. GOLDBERG, LINDA C. GLASER**

**WEDNESDAY *afternoon*, August 12 (Continued)**

**2:15 - 2:30** **(71) *MYCOPLASMA GALLISEPTICUM* IN HOUSE FINCHES AND OTHER PASSERINE SPECIES IN GEORGIA 1997-1998.**
**M.P.LUTTRELL, D.E. STALLKNECHT, D.M. KAVANAUGH, J.L CORN**

**2:30 - 2:45** **(72) MOLECULAR EPIDEMIOLOGY OF SONGBIRD CONJUNCTIVITIS ASSOCIATED WITH *MYCOPLASMA GALLISEPTICUM* AND *M. STURNI*.**
**DAVID H. LEY, STEVEN J. GEARY**

**2:45 - 3:30** **BREAK - POSTER SESSION in Inn Wisconsin**

**3:30 - 3:45** **(73) RESERVOIRS FOR AVIAN CHOLERA: WETLANDS OR WATERFOWL?**
**MICHAEL D. SAMUEL, DIANA R. GOLDBERG, DANIEL J. SHADDUCK, LYNN H. CREEKMORE**

**3:45 - 4:00** **(74) HABITAT MANAGEMENT AND VECTOR CONTROL: PROSPECTS FOR MANAGING AVIAN DISEASE IN HAWAIIAN FOREST BIRDS.**
**CARTER T. ATKINSON, JULIE K. LEASE, NICHOLAS P. SHEMA, ROBERT J. DUSEK, BETH M. DRAKE**

**4:00 - 4:15** **(75) IMMUNIZATION OF DUCKS FOR TYPE C BOTULISM.**
**ROBERTO MARTINEZ, GARY WOBESER**

**4:15 - 4:30** **(76) MARINE BIRDS AS POTENTIAL MONITORS OF MARINE ECOSYSTEM HEALTH.**
**DAVID A. JESSUP, MELISSA CHECHOWITZ, MIKE ZICCARDI, SCOTT NEWMAN, JONNA MAZET, FLO TSENG**

**THURSDAY *morning*, August 13**

## BIRDS II
## Moderator: Gary Wobeser

**8:00 - 8:15** **(77) CAUSES OF MORBIDITY AND MORTALITY IN BALD EAGLES FROM FLORIDA.**
**DONALD J. FORRESTER, NANCY J. THOMAS**

**8:15 - 8:30** **(78) SCHISTOSOMIASIS IN A COLLECTION OF CAPTIVE CHILEAN FLAMINGOS.**
**JEAN A. PARE, SANDRA R. BLACK**

**8:30 - 8:45** **(79) PARASITIC MITES OF NORTH AMERICAN OWLS.**
**JAMES R. PHILIPS**

**THURSDAY *morning*, August 13 (Continued)**

**8:45 - 9:00** **(80) SALMONELLOSIS IN WILD AND CAPTIVE BIRDS: PATHOLOGY AND CHARACTERIZATION OF ISOLATES.**
**CHARLOTTE F. QUIST, SARAH V. MEADS, CESAR A. MORALES, MARGIE D. LEE, JOHN J. MAURER**

**9:00 - 9:15** **(81) IDENTIFICATION OF DUCK PLAGUE VIRUS BY POLYMERASE CHAIN REACTION.**
**WALLACE R. HANSEN, SUSAN E. BROWN, SEAN W. NASHOLD, DENNIS L. KNUDSON**

**9:15 - 9:30** **(82) CHARACTERIZATION OF DUCK PLAGUE ISOLATES BY AMPLIFIED RESTRICTION FRAGMENT POLYMORPHISM (AFLP) ANALYSIS.**
**ROSER VELARDE, WALLACE R. HANSEN, SEAN NASHOLD**

**9:30 - 9:45** **(83) MOLECULAR AND IMMUNOLOGICAL EVIDENCE FOR DIVERSITY OF *PLASMODIUM RELICTUM* IN HAWAII.**
**SUSAN I. JARVI, JEFFREY J. SCHULTZ, CARTER T. ATKINSON**

**9:45 - 10:15** **BREAK**

## ENVIRONMENTAL CONTAMINANTS
### Moderator: Lou Sileo

**10:15 - 10:30** **(84) PASSERINE AND WATERFOWL REPRODUCTIVE AND IMMUNE STATUS ON RECLAIMED WETLANDS ON OIL SANDS MINING SITES.**
**JUDIT E.G. SMITS, MARK WAYLAND**

**10:30 - 10:45** **(85) ECOLOGICAL RISK ASSESSMENT AND CLEAN-UP GOALS FOR THE MOTHER LODE MERCURY MINE, PRINEVILLE, OR.**
**ANNE FAIRBROTHER, RICHARD S. BENNETT**

**10:45 - 11:00** **(86) HEAVY METAL, RADIONUCLIDE AND ORGANOCHLORINE CONTAMINANT LEVELS IN ESKIMO HARVESTED BOWHEAD WHALES OF ARCTIC ALASKA.**
**TODD O'HARA, GERALD BRATTON, PEGGY KRAHN, VICTORIA WOSHNER, LEE COOPER**

**11:00 - 11:15** **(87) EXPOSURE OF GREAT EGRET NESTLINGS TO MERCURY THROUGH DIET IN THE EVERGLADES ECOSYSTEM.**
**PETER C. FREDERICK, MARILYN G. SPALDING, MARIA S. SEPULVEDA, GARY E.WILLIAMS, LEO NICO, ROBERT ROBINS**

**THURSDAY *morning*, August 13 (Continued)**

**11:15 - 11:30** **(88) LEAD POISONING IN BIRDS IN SWEDEN.**
**TORSTEN MÖRNER, THOMAS JÅGAS, CARL HÅRD AF SEGERSTAD, DESIRÉE S. JANSSON, LARS PETERSSON**

**11:30 - 11:45** **(89) EFFECTS OF DIET AND SOIL INGESTION ON THE TOXICITY OF ZINC TO GAME-FARM MALLARDS, *ANAS PLATYRHYNCHOS*: HEMATOLOGY AND SERUM CHEMISTRY.**
**JEFFREY M. LEVENGOOD, GLEN C. SANDERSON, WILLIAM L. ANDERSON, GEORGE L. FOLEY, PATRICK W. BROWN, JAMES W. SEETS**

**11:45 - 12:00** **(90) WILDLIFE POISONING IN THE UNITED STATES: A LAW ENFORCEMENT PERSPECTIVE.**
**RICHARD K. STROUD, RHODA M. RALSTON, MARK KIRMS,**

**12:00 - 1:00** **LUNCH**

## MAMMALS I
**Moderator: Bill Samuels**

**1:00 - 1:15** **(91) PATHOGENESIS OF TUBERCULOSIS IN FERRETS (*MUSTELA FURO*) EXPOSED TO LOW AND HIGH DOSES OF *MYCOBACTERIUM BOVIS* INFECTION.**
**TARIQ QURESHI, ROB LABES, COLIN MACKINTOSH, FRANK GRIFFIN**

**1:15 - 1:30** **(92) VACCINATION OF BLACK-FOOTED FERRET X SIBERIAN POLECAT HYBRIDS AGAINST CANINE DISTEMPER WITH RECOMBINANT AND MODIFIED-LIVE VIRUS VACCINES.**
**ELIZABETH S. WILLIAMS, RICHARD J. MONTALI**

**1:30 - 1:45** **(93) SUSCEPTIBILITY OF RED FOXES (*VULPES VULPES*) AND GRAY FOXES (*UROCYON CINEREOARGENTEUS*) TO INFECTION BY *EHRLICHIA CHAFFEENSIS*.**
**WILLIAM R. DAVIDSON, J. MITCHELL LOCKHART, DAVID E. STALLKNECHT, ELIZABETH W. HOWERTH**

**1:45 - 2:00** **(94) HEMATOLOGIC VALUES IN *CYTAUXZOON FELIS* INFECTED FLORIDA PANTHERS (*FELIS CONCOLOR CORYI*) AND TEXAS COUGARS (*FELIS CONCOLOR STANLEYANA*).**
**DAVID S. ROTSTEIN, SHARON K. TAYLOR, JOHN W. HARVEY, JUDY BEAN**

**THURSDAY *afternoon*, August 13**

**2:00 - 2:15** **(95) SEROLOGICAL SURVEY OF INFECTIOUS DISEASE AGENTS OF BLACK BEARS, *URSUS AMERICANUS*, IN NORTHERN CALIFORNIA, OREGON AND WASHINGTON.**
**JACK A.MORTENSON, BRUNO B. CHOMEL, DAVE A. IMMELL**

**THURSDAY *afternoon*, August 13**

**2:15 - 2:30** **(96) THE ROLE OF AN ENDEMIC VIRAL INFECTION ON INDIVIDUALS AND ON POPULATION DYNAMICS IN EUROPEAN WOOD MICE, *APODEMUS SYLVATICUS* AND BANK VOLES, *CLETHRIONYMUS GLAREOLUS*.**
**JULIAN CHANTREY, SARAH FEORE, MALCOLM BENNETT, MIKE BEGON**

**2:30 - 3:00** **BREAK**

## MAMMALS II
## Moderator: Torsten Möerner

**3:00 - 3:15** **(97) PATHOLOGY OF HEPATITIS B IN ARCTIC GROUND SQUIRRELS.**
**JOHN BLAKE, CHRISTINE TERZI, KIMBERLEE BECKMEN.**

**3:15 - 3:30** **(98) PROBABLE MALIGNANT CERVICAL NONCHROMAFFIN PARAGANGLIOMA WITH PULMONARY AND CARDIAC METASTASES IN A SOUTHERN SEA OTTER (*ENHYDRA LUTRIS*).**
**HOWARD STEINBERG, CAROL U. METEYER, ELIZABETH J. GALBREATH, DOUG M. ENGLAND**

**3:30 - 3:45** **(99) FORENSIC EVALUATIONS OF WOLF CARCASSES: IS MS RED RIDING HOOD GUILTY?**
**RICHARD K. STROUD**

**3:45 - 4:00** **(100) IMPLANTATION OF INTRAPERITONEAL RADIOTRANSMITTERS IN BROWN BEARS (*URSUS ARCTOS*), WOLVERINES (*GULO GULO*) AND LYNX (*LYNX LYNX*): ANESTHETIC AND SURGICAL PROCEDURES FOR FIELD USE.**
**JON M. ARNEMO, PER DYPSUND, FINN BERNTSEN, JOHAN SCHULZE, SARI J. WEDUL, BIRGIT RANHEIM, LINE G. LUNDSTEIN**

**4:00 - 4:15** **(101) PORCINE ZONA PELLUCIDA IMMUNOCONTRACEPTION IN COYOTES, *CANIS LATRANS*.**
**THOMAS J. DE LIBERTO, FREDERICK F. KNOWLTON, J. RUSSELL MASON, LOWELL MILLER, MICHAEL K. HOLLAND**

**4:15 - 4:30** **(102) SHOULD TRADITIONAL RABIES DIAGNOSIS BE ATTEMPTED ON FIXED TISSUE SAMPLES?**
**CATHLEEN A. HANLON, JOHN SHADDOCK, CHARLES E. RUPPRECHT**

**4:30 - 4:45** **(103) WHEN SIZE MAY MATTER: RABIES IN THE BEAVER (*CASTOR CANADENSIS*).**
**C.E. RUPPRECHT, J. SHADDOCK, M. NIEZGODA, C.A. HANLON, L.A. ORCIARI, J.E. CHILDS, J.T. McPHERSON, L. HUNTER**

## (1) RABBIT HAEMORRHAGIC DISEASE IN AUSTRALIA.

**BRIAN D. COOKE, CSIRO, Wildlife and Ecology, PO Box 84, Lyneham, ACT, 2602, Australia.**

**Wild European rabbits, introduced in 1859, became a major pest of agriculture in Australia and caused the loss of many native plants and wildlife species. Despite the introduction of myxomatosis in 1950, the rabbits' continuing devastation of arid zone ecosystems has required serious action, mechanical and chemical solutions to the problem being impractical, particularly in vast uninhabited areas.**

**First described in domestic rabbits in China in 1984, RHD soon spread world wide. Its appearance in wild rabbits in Spain alerted Australian scientists to its potential for biological control. The virus was imported into Australia's Animal Health Laboratory for testing of host specificity, pathogenicity and likely efficacy in controlling rabbits. Transferred later to quarantine facilities on Wardang Island, for testing in rabbits living in natural warrens, the virus escaped and spread across southern Australia.**

*Initial Spread*

**The initial spread of RHD through the naive rabbit population yielded important epidemiological data. Most outbreaks were seen in spring and autumn when RHD spread at up to 100 km/week. Detection in summer was difficult. Retrospective analysis of climatic data showed most activity occurred with daily maxima of 15°- 32°C (optimum 23°C). Within eighteen months it had colonised the rabbit's natural range although deliberate releases of virus ensured establishment in some local areas.**

*Initial impact on rabbits*

**In arid areas rabbit populations were initially reduced by over 90%. However, results were less dramatic and patchier in temperate regions. Nevertheless, a substantial reduction in rabbits was achieved. Importantly, in arid areas it brought the density of rabbits below the level critical for regeneration of arid-zone plants (1 rabbit/ha).**

*Epidemiology*

**RHD is difficult to follow in the field. Rabbits show few signs of disease and many die underground. We have used sera from a population of rabbits, live-trapped every 4-8 weeks, to obtain most data. Antibodies to RHD and myxomatosis are detected using ELISAs; virus in cadavers is detected using virus capture ELISA and PCR.**

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**Antibody isotypes (IgG, IgA and IgM) are used to distinguish young rabbits with maternal antibodies from rabbits recovered from RHD. Individual rabbits followed over time confirm the reliability of methodologies. The role of maternal antibodies in the timing of RHD outbreaks is now understood. Combining antibody data with data on rabbit survival enables unravelling of the mortalities caused by RHD and myxomatosis. RHD kills about 86% of infected rabbits; myxomatosis still kills 50%.**

**In arid areas, RHD persists locally. As most young rabbits carry maternal antibodies, they are seldom infected with RHD until their maternal antibodies are lost at about 8 weeks. The disease "trickles" through the population in spring, and few young survive. As summer approaches, the rate of spread of virus slows, and many late-born young lose their maternal antibodies without becoming infected. However, the return of RHD in autumn accounts for most of these rabbits. In arid areas rabbit numbers fluctuate seasonally but in general are kept at about 10 - 20% of former levels.**

***Transmission studies***

**RHD spreads by several routes, including social contact between rabbits, fomites and insects. Using PCR, eight species of flies, one species of rabbit flea and two species of mosquito have been found contaminated with RHDV. PCR also shows that virus can be detected in the gut of blowflies (Calliphoridae) for up to 9 days after feeding on infected rabbit liver. Flyspots (crop contents and faeces) produced by these flies contain viable virus; a single flyspot given orally is enough to infect experimental rabbits. As these flies do not visit live rabbits, transmission probably occurs through rabbits eating contaminated pastures. Laboratory experiments show that flies, fleas and mosquitoes can transmit RHD. RHDV detected in rabbit urine on the surface of rabbit warrens may be another mechanism for spread between rabbits.**

***Conclusions and Future Work***

**RHD has significantly reduced rabbits in inland Australia and continues to keep rabbit numbers low. With myxomatosis, which still kills significant numbers of rabbits after almost 50 years, this new biological control is a very useful tool to help in the fight against the rabbit. Regeneration of native pastures and shrubs has begun, but long-term benefits to conservation and primary production are yet to be demonstrated. Other immediate economic and environmental benefits are apparent, including reduced costs of rabbit control and substantial reduction in the use of the poison '1080'.**

**While farmers are encouraged to get rid of remaining rabbits, future plans for managing RHD include the use of infective baits to release virus into susceptible rabbit populations when conditions are suitable. Monitoring long-term changes in rabbit and virus populations is also planned for determining how long RHD will remain useful as a biological control. This will provide comparative information on the co-evolution of viruses and their host.**

## (2) BRUCELLOSIS IN ELK: STUDIES OF EPIZOOTIOLOGY AND CONTROL.

**WALTER E. COOK and ELIZABETH S. WILLIAMS, Department of Veterinary Science, University of Wyoming, 1174 Snowy Range Road, Laramie, WY 82070.**

**Brucellosis is a bacterial disease of cattle that has become established in elk (*Cervus elaphus*) of the Greater Yellowstone Area. The fear that elk may spread the disease to livestock has prompted efforts to reduce or eliminate the disease in wildlife. We examined efficacy of *Brucella abortus* strain RB51 vaccine for preventing *Brucella* induced abortions in elk. We also tested the vaccine for safety in bull elk. The risk of transmission of *B. abortus* from elk to cattle depends in part on how long an aborted fetus remains in the environment and how long the bacteria are able to survive on the fetus. We used healthy bovine fetuses as surrogates to estimate how long an aborted elk fetus will remain prior to being scavenged. In addition, we examined duration of viability of *B. abortus* in the Wyoming environment. We found that one dose of $1X10^{9}$ or $1X10^{8}$ colony forming units of strain RB51 did not provide elk significant long term protection against challenge. The vaccine was safe in bull elk. Duration that fetuses remained in the environment varied from an average of 26.8 hours (sd=25.3 hr) to 57.5 hours (sd=48.0 hr) depending on location. *Brucella* survived on fetuses for over 60 days in the winter, but died off much quicker in hotter months.**

**(3) EXERTIONAL MYOPATHY IN RECENTLY CAPTURED NORTH AMERICAN RIVER OTTERS (*LONTRA CANADENSIS*) FROM NEW YORK.**

**BARRY K. HARTUP, Wildlife Health Laboratory, Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853; GEORGE V. KOLLIAS, Wildlife Health Laboratory, Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853; MATTHEW C. JACOBSEN, Department of Pathology, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853; BETH A. VALENTINE, Department of Pathology, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853 and KEVIN R. KIMBER, Wildlife Health Laboratory, Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853.**

**Acute exertional myopathy (EM) was documented in four North American river otters (*Lontra canadensis*) captured by foothold traps for a population restoration project. Gross lesions in one otter were characterized by locally extensive linear, pale areas within the subscapularis, rectus abdominis and quadriceps muscles. Microscopic lesions were characterized by acute to subacute myofiber necrosis of varying severity. A retrospective review of records of surviving, released otters (n=69) from 1995 to 1997 showed significant elevations in serum aspartate aminotransferase (AST) and creatine kinase (CK) in 19 (28%) of the otters, suggesting active myonecrosis as a result of capture, despite a lack of overt physical evidence of EM in any otter. No significant relationships were found between elevated muscle enzymes and geographic origin, ambient temperature at capture, restraint method used for trap removal, injury severity, weight, sex or age of the otters. A significant association ($P < 0.001$) was found between elevated AST and CK levels and the fewer number of days between capture and sampling. The data suggest that AST and CK levels elevate within 48 hours in foothold trapped otters, and then decrease to normal levels in approximately 5-6 days. These findings indicate that river otters are susceptible to acute severe and subclinical EM when captured, and early therapeutic intervention and reduction of stress is warranted to decrease capture-related morbidity and mortality.**

**(4) PREVALENCE OF HEMATOZOA IN SORAS (*PORZANA CAROLINA*) AND VIRGINIA RAILS (*RALLUS LIMICOLA*) IN SOUTHEASTERN WISCONSIN.**

**KATHERINE LEWANDOWSKI, School of Veterinary Medicine, University of Wiconsin-Madison, 601 Wingra St., Madison, WI, 53715; CHRISTINE RIBIC & JENNIFER SKOLODA, USGS-BRD Wisconsin Cooperative Wildlife Research Unit, Department of Wildlife Ecology, University of Wisconsin-Madison, Madison, WI 53706; and LINDA SULLIVAN, School of Veterinary Medicine, University of Wiconsin-Madison, Madison, WI, 53706.**

**Birds from two species of Rallidae: Virginia rails (*Rallus limicola*)(n=31) and soras (*Porzana carolina*) (n=16), were trapped at Horicon National Wildlife Refuge, Wisconsin, during June, July, and August of 1996 and 1997. Peripheral blood smears from each bird were examined for hematozoa. The overall prevalence of blood parasites in the 47 Rallids was 48.9%. Parasites identified included *Plasmodium* spp. (40.4% prevalence), *Haemoproteus* spp. (14.9%), and nematode microfilariae (4.3%). The high prevalence of parasitism could reflect the vector potential and parasite prevalence of the habitat, the time of year sampled, or the host-parasite relationship which has developed among Rallidae in this region. No significant differences in prevalences of overall parasitism, *Plasmodium*, or *Haemoproteus* were noted between species of rail, sex, year, or month sampled. Intensities of infection were low.**

**(5) THE EFFECT OF BOVINE BRUCELLOSIS AND TUBERCULOSIS ON PREGNANCY RATES IN WOOD BISON: PRELIMINARY RESULTS FROM WOOD BUFFALO NATIONAL PARK, CANADA.**

**DAMIEN O. JOLY, Department of Biology, University of Saskatchewan, 112 Science Place, Saskatoon, SK, S7N 5E2, Canada; FREDERICK A. LEIGHTON, Department of Veterinary Pathology, Western College of Veterinary Medicine, 52 Campus Drive, Saskatoon, SK, S7N 5B4, Canada; and FRANÇOIS MESSIER, Department of Biology, University of Saskatchewan, 112 Science Place, Saskatoon, SK, S7N 5E2, Canada.**

**We present preliminary data on the effect of bovine tuberculosis (*Mycobacterium bovis*) and brucellosis (*Brucella abortus*) on pregnancy rates in wood bison. In the winter of 1997, bison were captured and tested as part of an ongoing investigation into the effects of these diseases on the population dynamics of bison. Tuberculosis infection was determined using the caudal fold and an enzyme-linked immunosorbent assay (ELISA). The complement fixation and buffered plate antigen tests were used to test for brucellosis. We determined pregnancy rates by testing for pregnancy-specific protein B and evaluating serum progesterone levels. The total pregnancy rate for adult female bison (> 2 years) was 78% (n = 73). We were unable to detect an effect of brucellosis infection on pregnancy rate, however a lack of statistical power prevents us from making conclusions in this regard. Further, pregnancy determination was done in late February and early March, before the period where most brucellosis-induced abortions would occur. We found that bison that tested positive for tuberculosis had a significantly lower pregnancy rate than those that tested negative (66% vs. 95%). This is the first report of an effect of tuberculosis on pregnancy rates in bison.**

## (6) CAN THE ASIAN TIGER MOSQUITO, *AEDES ALBOPICTUS*, FACILITATE THE EVOLUTION OF CALIFORNIA SEROGROUP BUNYAVIRUSES IN NORTH AMERICA?

**LI-LIN CHENG, JUAN D. RODAS, THOMAS M. YUILL, and BARBARA A. ISRAEL, Department of Pathobiological Sciences, School of Veterinary Medicine, University of Wisconsin-Madison, 2015 Linden Dr. West, Madison, WI 53706; KEVIN T. SCHULTZ, Merill Inc., P.O. Box 2000, RY33-216, Rahway, NJ 07065.**

***Aedes albopictus*, the Asian tiger mosquito, was introduced into the United States in used tires in 1985. Its successful colonization into the upper regions of the Midwest may alter the current patterns of arboviral diseases. *Ae. albopictus* is permissive for the replication of several arboviruses, including La Crosse (LACV) and Jamestown Canyon (JCV) viruses, members of the family Bunyaviridae. LACV and JCV are often present in the same geographic area of the Upper Midwest, but remain genetically distinct because they are maintained in transmission cycles involving different mosquito vectors and vertebrate hosts. In this study, we demonstrated the ability of LACV and JCV to co-infect *Ae. albopictus*, and to form six genotypes of reassortants. All reassortants can infect *Ae. albopictus* and can be transmitted to suckling mice. However, reassorted viruses carrying the LACV M segment in the foreign genetic background of JCV were more neuroinvasive than JCV, or any other reassorted genotype. In addition, these reassortants were able to amplify in gerbils and infect *Ae. triseriatus*; characteristics of LACV, but not JCV. Our study has at least 3 significant implications: first, LACV and JCV can reassort in *Ae. albopictus* despite being in different antigenic subgroups; second, reassortment of LACV and JCV can also alter the vertebrate amplifying host range, and expand the mosquito vector species for the viruses; third, *Ae. albopictus* could facilitate virus evolution and the emergence of bunyavirus diseases in North America via virus reassortment.**

**(7) BIOSAFETY STUDY OF *BRUCELLA ABORTUS* STRAIN ARS/1 VACCINATION IN BISON BULLS: EVALUATION OF IMMUNOLOGIC RESPONSES.**

**STEVEN C. OLSEN, Zoonotic Diseases Research Unit, National Animal Disease Center, Agricultural Research Service, U.S. Department of Agriculture, P.O. Box 70, Ames, Iowa, 50010; JACK RHYAN, Animal and Plant Health Inspection Service, U.S. Department of Agriculture, 1716 Heath Parkway, Fort Collins, Colorado 80524; THOMAS GIDLEWSKI, Animal and Plant Health Inspection Service, U.S. Department of Agriculture, Lincoln Way, Ames, Iowa 50010; MITCHELL V. PALMER, Zoonotic Diseases Research Unit, National Animal Disease Center, Agricultural Research Service, U.S. Department of Agriculture, P.O. Box 70, Ames, Iowa, 50010; AMY M. HAYEK JONES, Animal and Plant Health Inspection Service, U.S. Department of Agriculture, Center for Epidemiology and Animal Health PO Box 1601, Fort Collins, Colorado 80522.**

**The infection of free-ranging bison in the Greater Yellowstone Area with field strain *Brucella abortus* has been well documented. It has been suggested that bison may infect the cattle populations of Wyoming and Montana outside of the park, threatening the brucellosis free status of these two states. Although data suggests that bulls do not play a significant role in transmission of brucellosis in cattle, the role of bison bulls is not clear regarding the epidemiology of the disease in the Yellowstone National Park bison herds. In 1991-1992, when evaluated for brucellosis serologically, 49.7 percent of the bulls tested were positive or suspect as compared to 39.6 percent of the adult females. During the winter of 1996-1997, captured or killed bulls greater than 1 year of age were serologically tested for brucellosis with 69.3 percent positive or suspect on brucellosis surveillance tests.**

**The Brucellosis Eradication program for cattle does not include vaccination of adult males. While the National Park Service policy of natural regulation precludes any management procedures which might influence age or sex classes within the population, it is likely that any management plan to reduce or eliminate brucellosis in Yellowstone National Park bison wo uld need to address the prevalence of brucellosis in bison males. The purpose of the studies reported here were to evaluate the biosafety of the current cattle vaccine, *B. abortus* strain ARS/1 (RB51), in bison bulls and to characterize immune responses following vaccination. Bison were vaccinated by hand and with ballistic bullets. Bison vaccinated with RB51 showed vaccine strain *B. abortus* localized in lymphatic tissues at 10 weeks post vaccination, and clearance at 20 weeks post vaccination. At necropsy, 10 of a total 42 RB51 vaccinated bison were culture positive. While no inflammation or histopathological lesions were found, some of the bison showed transient shedding in semen. The significance of this transient shedding is unknown at this time.**

## (8) A VACCINE EFFICACY TRIAL OF *BRUCELLA ABORTUS* STRAIN RB51 IN BISON.

**MATTHEW D. EDMONDS, Department of Veterinary Microbiology and Parasitology, Louisiana State University School of Veterinary Medicine, Baton Rouge, LA 70803; DAVID HUNTER, Idaho Fish and Game, Caldwell, ID 83605; MICHAEL GILSDORF, USDA, Animal and Plant Health Inspection Service, Veterinary Services, Riverdale, MD 20737; JOE TEMPLETON, DONALD DAVIS, Department of Veterinary Pathobiology, Texas A&M University, College Station, TX 77843; and PHILIP ELZER, Department of Veterinary Science, Louisiana State University Agricultural Center, Baton Rouge, LA 70803.**

***Brucella abortus*** **is a gram-negative facultative intracellular pathogen known to cause abortions in both cattle and bison. This study is an ongoing evaluation of the vaccine efficacy of *B. abortus* strain RB51 in bison. We have previously reported on the safety and colonization of RB51 in a *Brucella*-exposed bison herd. The animals used in the current study included nine adult cows obtained from a commercial Kansas ranch and 20 adults cows from Yellowstone National Park. During the fall, spring and winter of 1997 all of these animals were subcutaneously vaccinated three times with 1 x $10^9$ colony forming units (c.f.u.) *B. abortus* strain RB51. For controls, 18 bison from a *Brucella*-free Colorado herd were injected with saline. Following vaccination and confirmation of pregnancy, both the test and control animals were inoculated in the conjunctival sac with 1 x $10^7$ c.f.u. of virulent *B. abortus* strain 2308. Following abortion or parturition, the delivery status of each calf was recorded and rectal swabs obtained for the culture of *B. abortus*. At calf necropsy culture samples were also obtained from the liver, spleen, lung, and abomasal fluid of each calf. Following completion of the calving season, the dams will be necropsied and the standard samples obtained for culture analysis.**

**(9) RECENT ESTABLISHMENT OF AN ISOLATED ENDEMIC POPULATION OF *IXODES SCAPULARIS*, THE VECTOR OF LYME BORRELIOSIS, AT POINT PELEE, ONTARIO, CANADA.**

**L.R. LINDSAY, Federal Laboratories of Health Canada, 1015 Arlington St., Winnipeg, MB, Canada R3E 3M4; T.H. AKWAR, Department of Environmental Biology, Ontario Agricultural College, University of Guelph, ON, Canada N1G 2W1; I.K. BARKER, Department of Pathobiology, Ontario Veterinary College, University of Guelph, ON, Canada, N1G 2W1; and M.D. RAVYN, Department of Microbiology, University of Minnesota, MN 55455, U.S.A.**

**Long Point, Ontario, on the north shore of Lake Erie, was until recently the only Canadian locality known to be endemic for the black-legged tick, *Ixodes scapularis*, the vector of Lyme borreliosis. We report here the establishment of a second population of this tick in Canada. Point Pelee National Park (PPNP), at the west end of Lake Erie (41º80' N, 82º51'W), concentrates birds migrating across the Great Lakes. From 1988 to 1990, 66 *Peromyscus leucopus*, 22 *Microtus pennsylvanicus* and one *Zapus hudsonius* were trapped at PPNP and examined for ticks, and limited drag sampling was carried out. No *I. scapularis* were detected. Between October 1994 and October 1995 a few adult *I. scapularis* were reported on dogs or people who had visited PPNP, and such reports recurred in 1996. In 1996 and 1997, surveys of small mammals and drag sampling were carried out to re-evaluate the status of *I. scapularis* at PPNP. In July 1996, a single larval *I. scapularis* was found on 1/54 *P. leucopus*. In June 1997, 1/55 *P. leucopus* was infested with a single larva, likely overwintered from 1996. However, on 7 August, 1997, after the expected hatch date of tick egg masses, 19/56 *P. leucopus* were infested with larval *I. scapularis* (range 1-12; mean 2.7). In April 1998, 9/33 *P. leucopus* were infested with larval (n=6) or nymphal (n=6) *I. scapularis* (range 1-2 ticks). From October 1997 - April 1998, 33 adult *I. scapularis* (16 ÿÿ, 17ÿÿ) were collected in PPNP by dragging. The presence of all stages of *I. scapularis* on small mammals or in the environment confirms that PPNP is endemic for this tick. The sharp increase in numbers of larvae on mice in summer 1997 suggests that the population is expanding from a narrow base, which likely established in the early-mid 1990's, possibly originating from ticks translocated by birds migrating from endemic areas to the south. However, there is no firm evidence that *Borrelia burgdorferi* is present in the host-vector system at PPNP. Antibody titres (IFA cutoff 1:20 or 1:32) against *B. burgdorferi* have been uncommon in small mammals between 1988 and 1997 (4/228 *P. leucopus*; 5/18 *M. pennsylvanicus*), and low (~1:20-1:32), possibly reflecting serologic cross-reactions, rather than genuine exposure to the agent of Lyme borreliosis. *Borrelia burgdorferi* has not been detected in the tissues of 165 *P. leucopus* examined since 1996. Nor has it been detected by PCR in 22 adult *I. scapularis* collected in 1997-98, though an adult recovered from a dog which had been to PPNP in 1994 was reported by others as positive for *B. burgdorferi* by PCR. The founders of this tick population, probably larvae or nymphs translocated by birds, likely were not infected with the Lyme borreliosis agent. Not until an infected host is fed upon by an immature tick at PPNP, or an infected tick is imported, molts and survives to feed, will this agent become established. As the indigenous tick population expands, the probability of an individual infected host being fed upon by a competent vector increases concomitantly, and with it the likelihood of establishment of *B. burgdorferi* in this isolated vector-host system.**

**(10) MULTIPLE ENDOCRINE NEOPLASIA TYPE IIA IN A RED WOLF (*CANIS RUFUS*).**

**FELICIA B. NUTTER, Environmental Medicine Consortium and Department of Companion Animal and Special Species Medicine, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough St., Raleigh, NC 27606; and FRANK J. GEOLY, Department of Microbiology, Pathology, and Parasitology, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough St., Raleigh, NC 27606.**

**An 11-year-old intact male red wolf necropsied at the North Carolina State University Veterinary Teaching Hospital was diagnosed with carcinoma of the left thyroid gland, C-cell hyperplasia and adenomas of the right thyroid gland, parathyroid adenoma of the right parathyroid gland, and adrenal pheochromocytoma. The triad of thyroid carcinoma, parathyroid adenoma, and pheochromocytoma comprises the syndrome of multiple endocrine neoplasia type 2a (MEN 2a; aka MEN 2, Sipple's syndrome). In humans this is a genetic disease inherited as an autosomal dominant gene with age-related penetrance. The RET proto-oncogene responsible for multiple endocrine neoplasia type IIa in humans has been localized on chromosome 10. Most human families with MEN 2a have been found to carry mutations in a cysteine-rich region of the RET gene, which convert the RET gene into a dominantly acting oncogene. If the genetic basis of this disease is similar in canids, there may be implications for the red wolf recovery program because of the small founder population. Genetic testing of surviving siblings and offspring and a review of post-mortem findings for all related individuals is in progress.**

**(11) COMPARING SEROLOGICAL RESPONSES OF CAPTIVE BIGHORN SHEEP (*OVIS CANADENSIS*) TO A MULTIVALENT *PASTEURELLA HAEMOLYTICA* SUPERNATANT VACCINE DELIVERED VIA HAND INJECTION, REMOTE IMPLANTATION, OR ORAL SUSPENSION.**

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**The efficacy and safety of a multivalent *Pasteurella haemolytica* supernatant vaccine (serotypes A2 and T10) using different delivery systems was examined in captive bighorn sheep (*Ovis canadensis*). Thirty bighorn sheep were grouped according to baseline leukotoxin neutralizing antibody titers (ÿ2 or >2) and vaccination history (previously vaccinated or unvaccinated). Within these groups, animals were randomly assigned to one of three delivery treatments: hand injection (control), biobullet implantation, or oral alginate microsphere suspension. All bighorns received a single dose from the same lot of vaccine (n=10/treatment). To monitor potential changes in baseline titers, an additional four animals were injected intramuscularly with 0.9% saline. Mild, transient lameness in most bighorns one day after receiving vaccine either by hand injection or biobullet implantation was the only adverse effect observed. Sera were collected immediately prior to vaccination (wk 0) and at 1, 2, 4, 8, and 12 weeks postvaccination. Sera were tested for both leukotoxin neutralizing and surface antigen (A2, T10) agglutinating antibody levels. Serum neutralizing antibody titers to *P. haemolytica* leukotoxin differed among delivery treatments (P=0.009) and among baseline titer/vaccination history groups (P=0.013). Neutralizing titers were highest among hand-injected bighorns; no serum antibody response was detected among bighorns vaccinated orally. Although neutralizing titers were lower among implanted bighorns than hand-injected controls for ÿ2 weeks after vaccination (P<0.021), seroconversion rates in response to implantation (6/10) and hand injection (9/10) did not differ (P=0.303). Agglutinating antibody titers to T10 were high and did not vary over time or between delivery treatments. Agglutinating antibody titers to A2 differed between the hand-injected controls and bighorns vaccinated with biobullet implantation (P=.0374) as well as between the hand-injected controls and those animals vaccinated orally (P=.0106). These data show that although hand injection elicits higher absolute titers than either biobullet implantation or oral vaccination, biobullet implantation may also stimulate effective antibody responses to *Pasteurella haemolytica* supernatant vaccine. Whether lack of serum antibody responses to oral vaccination truly reflects failure to stimulate immunity remains undetermined. Further evaluation of biobullet vaccination against pneumonic pasteurellosis in free-ranging populations of wild bighorn sheep appears warranted.**

**(12) DEVELOPMENT OF A MUSKOX LUNGWORM, *UMINGMAKSTRONGYLUS PALLIKUUKENSIS*, IN THE SLUG, *DEROCERAS LAEVE*, UNDER ARCTIC FIELD CONDITIONS WITH COMMENTS ON THE IMPACT OF CLIMATE CHANGE.**

**SUSAN J. KUTZ, Department of Veterinary Microbiology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK, S7N 2B4; ERIC P. HOBERG, ARS, USDA, Beltsville, Maryland, 20705, USA; JOHN S. NISHI, DRWED, Gov. NWT, Fort Smith, NT; LYDDEN POLLEY, Department of Veterinary Microbiology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK, S7N 2B4.**

***Umingmakstrongylus pallikuukensis* is a protostrongylid lungworm first recognized in 1988 in the muskox population west of Kugluktuk, NT (67°54'N, 116°38'W). The prevalence approaches 100% in adult muskoxen in this region yet the parasite appears to be absent from the arctic islands. First stage larvae (L1) shed in the feces can develop to infective third stage larvae (L3) in at least 4 terrestrial and 1 freshwater gastropod species found near Kugluktuk. Development rates of L1 to L3 are highly dependent on temperature. Field studies to determine whether larval development can occur within a single season in *D. laeve* were carried out during summer 1997. Slugs infected on June 19, July 3 and July 17 contained L3 by 4 to 6 weeks post infection while those infected July 31 or later did not produce L3 before winter. Recovery in September of live L3 from the vegetation in plots of June 19 and July 3 experiments confirmed laboratory findings of larval emergence from slugs although we could not determine if emergence was from live or dead slugs. Research has indicated that *U. pallikuukensis* in muskoxen is a temperature dependent system in which larval development and potential patterns of parasite transmission and distribution may be influenced by alteration in global temperatures. Predicted global warming is anticipated to have impacts on arctic ecosystems. Perhaps significantly, historical temperature records indicate a warming trend in the Mackenzie District of 1.29 °C over the last 50 years. *U. pallikuukensis* in muskoxen offers a possible theoretical and applied model system to elucidate the complex linkage of global climate change to wildlife health, emerging helminthic disease, and the potential to impact populations of arctic ruminants.**

**(13) ASSESSING HEALTH STATUS OF FREE-RANGING PSITTACINES IN THE BOLIVIAN AMAZON.**

**CATHERINE SOOS, Veterinary Teaching Hospital, University of Guelph, Guelph, Ontario, CanadaN1G 2W1; BRUCE HUNTER, Department of Pathobiology, University of Guelph, Guelph, Ontario, Canada N1G 2W1; DAVID N. PHALEN, Department of Large Animal Medicine and Surgery, Texas A&M University, College Station, Texas 77843-4475; LAUREL NEUFELD, University of Manitoba, Winnipeg, Manitoba, Canada R3T 2N2; and CHARLES A. MUNN, Wildlife Conservation Society, 185th and Southern Boulevard, Bronx, New York 10460.**

**A study to assess the health status of individual free-ranging psittacine birds was performed in the Bolivian Amazon within the Madidi National Park. Between 15 August and 1 September 1996, thirteen blue-headed pionus (*Pionus menstruus*), three mealy Amazon parrots (*Amazona farinosa*), and one juvenile yellow-crowned Amazon parrot (*Amazona ochracephala*) were captured and released from a series of clay cliffs along the Heath River. Data was obtained from physical examinations, blood smears, complete blood cell counts, fecal flotations, and serology. Plasma samples were analysed for antibody titers to Pacheco's disease herpesvirus, polyomavirus, paramyxovirus-1, avian influenza virus, and *Chlamydia psittaci*. Lice and hippoboscid flies were observed on many of the *P. menstruus* and *A. farinosa*. Three *P. menstruus* exhibited cloacal lesions, while five *P. menstruus* and one *A. farinosa* possessed periocular scars and corneal ulcerations. *Hemoproteus sp.* was detected in a blood smear of one *P. menstruus*. Strongyle-like eggs and coccidia oocysts were found in some individuals of all three species; ascarid eggs were detected in two *P. menstruus*. All plasma samples were negative for antibodies to avian influenza (agar gel immunodiffusion), paramyxovirus-1 (hemagglutination inhibition), and avian polyomavirus (virus neutralization). Three *P. menstruus* had positive antibody titers to *Chlamydia psittaci* using the complement fixation test, however all birds had negative titers using elementary body agglutination. Five *P. menstruus* had low antibody titers to Pacheco's disease herpesvirus (virus neutralization), and one *A. farinosa* had a markedly elevated titer, consistent with active infection with the virus.**

**(14) HEARTWORMS AND LUNGWORMS IN ILLINOIS' CANIDS AND THEIR POSSIBLE EFFECT ON COYOTE CONDITION AND REPRODUCTION.**

**DAVID G. GREGORY, Department of Biological Sciences, Eastern Illinois University, Charleston, IL 61920; TOM NELSON, Zoology Department, Eastern Illinois University, Charleston, IL 61920; JEFF LAURSEN, Zoology Department, Eastern Illinois University, Charleston, IL 61920.**

**This study focuses on the prevalence of heartworm and lungworms in Illinois' canid species, and the effect that they may have on condition (body weight, kidney fat, marrow fat) and reproduction (placental scars) on coyote populations. A total of 1,150 coyotes (*Canis latrans*), 2,269 domestic dogs, 47 red foxes (*Vulpes vulpes*), and 2 gray foxes (*Urocyon cinereoargenteus*) were examined. Prevalence of heartworms (*Dirofilaria immitis*) averaged 17.8% in coyotes, 3.0% in domestic dogs, 2.0% in red foxes, and 0% in gray foxes. Domestic dogs not receiving any type of prophylactic treatment had a higher prevalence (12.5%) of heartworms than dogs on a prophylactic program (0.3%). Heartworm prevalence varied regionally throughout the state in both coyotes and domestic dogs reflecting a lower prevalence in the northern regions of the states and a higher prevalence in the south. Of the 341 coyotes examined for lungworms, 52 (15.2%) were infected with *Capillaria aerophila*, 10 (2.9%) with *Fillaroides sp*., 8 (2.3%) with *Paragonimus kellicotti*, and 2 (0.6%) with *Crenosoma vulpis*. A stomach parasite, *Physaloptera rara*, was also recovered from 58 (17%) coyotes. Fifteen red foxes were examined for the presence of lung parasites, of which, 11 (73.3%) were infected with *Capillaria aerophila*, 1 (1.1%) was infected with *Crenosoma vulpis*, and 1 (1.1%) with *Physaloptera rara*. Heartworm and/or a lungworm infection did not appear to significantly impact the condition or reproduction of coyotes since no significant differences were observed in the body weight, fat reserves, or number of placental scars of uninfected and infected individuals.**

**(15) EXPERIMENTAL INFECTIONS OF *PARELAPHOSTRONGYLUS TENUIS* IN WHITE-TAILED AND RED DEER.**

**MICHAEL S. DUFFY & MICHAEL D. B. BURT, Department of Biology, University of New Brunswick, Fredericton, New Brunswick, Canada, E3B 6E1.**

**Experimental infections of *Parelaphostrongylus tenuis* were attempted in 12 white-tailed deer (natural host) and 12 red deer (atypical host). In each deer species, 3 groups of 4 animals were fed 10, 25 and 100 third-stage larvae (L3) of *P. tenuis*, respectively. Infections were monitored for up to 3 1/2 years in white-tailed deer and for up to 2 1/2 years in red deer. The responses of both species of deer in the groups fed 100 L3 of *P. tenuis* differed from that of groups fed 10 or 25 L3. The white-tailed deer fed 100 L3 were found to have the onset of patency delayed, whereas the same number of larvae fed to red deer resulted in debilitation, presumably neurologic, in 2 of the 4 animals. The pre-patent period in these white-tailed deer was extended, up to 5 (and possibly 9) times longer than that for animals in groups fed lower doses. Patent infections in red deer were found to persist, on average, for only 6 months, whereas most white-tailed deer still had patent infections at necropsy. We suggest that red deer represent an inferior host for *P. tenuis* with respect to biotic potential of the parasite. We also suggest that exposures of 100 L3, or more, of *P. tenuis* are well above that perceived as natural in the field and are therefore undesirable for future experimental studies which attempt to mimic natural infection levels or utilize atypical hosts.**

**(16) HARMFUL ALGAL BLOOMS IN COASTAL AND ESTUARINE WATERS: CURRENT STATUS OF AN EXPANDING PROBLEM.**

**DONALD M. ANDERSON, Biology Department, Woods Hole Oceanographic Institution, Woods Hole MA 02543 U.S.A.**

**Among the thousands of living marine phytoplankton species are a few dozen which cause harm, either because of the potent toxins they produce or the biomass of their "blooms". Impacts from these tiny organisms are many and diverse, ranging from the death or illness of humans, whales, or other marine animals to discoloration of the water and fouling of beaches with foam and dead fish. This talk will review the many types of harmful algal blooms (HABs) and their impacts throughout the U.S., focusing where relevant on effects on wildlife and ecosystems. Another emphasis will be on the view held by many scientists that the problem is growing worse - possibly as a result of human activities. Arguments will be presented that many new or expanded red tide problems can be linked to pollution, aquaculture development, or other human alterations to coastal waters, but that many HAB problems are natural phenomena. In either case, the critical importance of coastal waters for aquaculture, fisheries, and wildlife habitat dictates that urgent steps be taken to better understand and manage harmful algal species and the resources they threaten. A summary of ongoing national programs and initiatives will be provided to demonstrate the current status of research and monitoring efforts.**

**(17) CYANOBACTERIAL TOXINS: HEALTH EFFECTS AND RISK ASSESSMENT.**

**WAYNE W. CARMICHAEL, Department of Biological Sciences, Wright State University, Dayton, Ohio 45435.**

**Increasingly, harmful algal blooms (HAB's) are being reported worldwide due to several factors, primarily - eutrophication, climate change and more scientific investigation. HAB organisms include those causing: PSP (paralytic shellfish poisoning), DSP (diarrhetic shellfish poisoning), NSP (neurotoxic shellfish poisoning), ASP (amnesic shellfish poisoning) and CTP (cyanobacteria toxin poisoning). All but CTP organisms are mainly a marine occurrence. CTP's occur in freshwater lakes, ponds, rivers and reservoirs throughout the world. Organisms responsible include an estimated 40 genera but the main ones are *Anabaena, Aphanizomenon, Cylindrospermopsis, Microcystis, Nostoc* and *Oscillatoria (Planktothrix)*. Cyanobacteria toxins (cyanotoxins) include cytotoxins and biotoxins with biotoxins being responsible for acute lethal, acute, chronic and sub-chronic poisonings of wild/domestic animals and humans. The biotoxins include the neurotoxins; anatoxin-a, anatoxin-a(s) and saxitoxins plus the hepatotoxins; microcystins, nodularins and cylindrospermopsin. Risk assessment of microcystins indicate that a level of 1 ug/L should be considered a maximum allowable concentration (MAC) based upon an adult consumption of 2L/day. Other MAC's for the neurotoxins are being evaluated.**

**Implication of microcystins as the major contributing factor in liver failure and death of at least 52 humans in 1996, at a haemodialysis center in Caruaru, Brazil point to the importance of cyanotoxins as health hazards in drinking waters. Since most of the world's reservoir and lake based water supplies are subject to increasing nutrient levels, it is probable that episodes of cyanotoxin poisoning will continue unless measures are taken to improve our understanding of their role in water-based diseases.**

**Ref:**

**The Cyanotoxins. 1997. Carmichael, W.W. Advances in Botanical Research, Vol. 27. Ed. by C.A. Callow. Academic Press, London.**

**Liver Failure and Death after Exposure to Microcystins at a Hemodialysis Center in Brazil. 1998. Jochimsen, E.M., Carmichael, W.W., An, Jlsi, Cardo, D.M., Cookson, S.T., Holmes, C.E.M., Antunes, B de C., Filho, D.A. de Melo, Lyra, T.M., Spinelli, T.B., Azevedo, S.M.F.O. and Jarvis, W.R. New England J. Medicine, 338(13):873-878.**

**(18) THE TOXIC *PFIESTERIA* COMPLEX.**

**JOANN M. BURKHOLDER and HOWARD B. GLASGOW, College of Agriculture and Life Sciences, Department of Botany, Campus Box 7612, North Carolina State University, Raleigh, NC 27685-7612.**

**Outbreaks of the icthyotoxic, ambush-predator dinoflagellate, *Pfiesteria piscicida*, have provided a compelling illustration of strong linkages between fish kills/epizootics and subtle but serious impacts on human health. Through field studies supported by experimental verification of toxicity in bioassays with fish, *P. piscicida* has been implicated as a causative agent of major fish kills/epizootics ($10^3$ to $10^9$ fish) in nutrient-degraded areas of the two largest estuaries on the U.S. mainland, Chesapeake Bay and the Albermarle-Pamlico. A second, mildly toxic *Pfiesteria*-like species (designated species "B" prior to completion of formal naming procedures) sometimes co-occurs with *P. piscicida* in certain estuaries. In earlier research (1994), fish bioassays were used to detect at least one other toxic *Pfiesteria/Pfiesteria*-like species in estuaries along both coasts of Florida and other Gulf Coast states.**

**Many "morphospecies" that resemble *Pfiesteria* occur in the mid-Atlantic and Southeast, but they have not been examined for toxicity. Tests with fish (in required biohazard III facilities to prevent human exposure to *Pfiesteria's* airborne neurotoxins) are underway to determine which of the dinoflagellates present should be added to the toxic *Pfiesteria* complex. Logically, since the species of concern from fish and human health perspectives are the toxic species, the terms "*Pfiesteria*" or "*Pfiesteria*-like" should be reserved for species that not only look like *Pfiesteria*, but are like *Pfiesteria.* True "*Pfiesteria*-like" species should have (I) demonstrated chemosensory stimulation by live fish or their fresh tissues; (II) toxicity toward fish; (III) a complex life cycle with multiple flagellated and amoeboid stages; and (IV) chloroplasts absent or, if present, retained in an epithecal food vacuole as kleptochloroplasts. As other newly known species are verified to have these demonstrated traits, they should be added to the toxic *Pfiesteria* complex. To facilitate identifications, we are working with colleagues who have developed a molecular probe for *Pfiesteria piscicida,* and probe development for the second toxic *Pfiesteria*-like species is in progress.**

***P. piscicida* and the second known toxic *Pfiesteria*-like species are eurythermal and euryhaline, with optima for toxic activity by the most lethal stages at ~26°C and 15 psu, respectively (toxic zoospores - NC isolates). In warmer waters (≥15°C) flagellated stages of the best known species, *P. piscicida*, predominate while fish are dying, whereas toxic amoebae are more abundant in colder conditions. Both water-soluble and lipophilic toxins have been isolated from *P. piscicida* (with colleagues at the National Ocean Service - Charleston). The toxins are highly biologically**

**-continued-**

**active, thus far unique, and unstable in water and air. The lipid-soluble fraction destroys fish epidermis and osmoregulatory function. Upon exposure to a water-soluble neurotoxin, test fish become moribund in 2-3 seconds, with death in 3-5 minutes. A reporter gene assay is being developed that has been used successfully in the laboratory to detect extremely low levels of *P. piscicida'*s toxins in water samples and human blood serum. Field tests/calibrations of this assay are in progress.**

**Human exposures to *Pfiesteria'*s toxins have occurred primarily through inhalation of airborne toxins from fish-killing cultures or over toxic outbreaks in estuaries, as well as through water contact. Impacts sustained by laboratory workers have included central nervous sytem dysfunction (severe learning impairment, memory loss); autonomic and peripheral nervous system dysfunction; severe headaches and respiratory distress; open epidermal lesions; renal and hepatic dysfunction; nausea, vomiting and other symptoms. Under field conditions, similar profound cognitive impairment has been sustained by people exposed to *Pfiesteria*-related fish epizootics or kills. In repeated experiments, small mammals exposed to *Pfiesteria*'s toxins have sustained significant learning disabilities. We are working to strengthen general understanding of the nutritional ecology and chronic/sublethal impacts of the toxic *Pfiesteria* complex, and to characterize their interactions with other pathogens in impairing finfish and shellfish growth, reproduction, recruitment, and survival.**

## (19) EXPOSURE OF MARINE ANIMALS TO BIOTOXINS: IS THERE A CHRONIC DISEASE PROBLEM?

**JAN H. LANDSBERG, Florida Department of Environmental Protection, Florida Marine Research Institute, 100 Eighth Ave SE., St. Petersburg, Florida, 33701.**

**The effects of harmful algal blooms (HABs) on marine animals that are usually considered are direct toxicity during or immediately following the acute planktonic phase. Such direct impacts on specific groups of marine animals include water-borne exposure to the toxin through algal cell lysis (fish, shellfish); bioaccumulation by ingestion of toxic prey or substrate (fish, birds, turtles, mammals); inhalation of aerosolized toxin (mammals, turtles, birds); creation of toxic sediment sinks (benthic animals); and consumption of toxic benthic stages (shellfish). Acute exposure to lethal doses of toxins can result in massive animal mortalities over a relatively short time. There is little information concerning chronic, lethal or sublethal effects on marine organisms caused by exposure to low toxin concentrations. The potential for some biotoxins to act as immuno-modulators has not been well explored. Numerous unexplained marine animal mortalities and disease processes may be attributable to chronic exposure to biotoxins. Chronic exposure can lead to impaired feeding, avoidance behavior, physiological dysfunction, increased susceptibility to disease, reduced growth and reproduction, and, ultimately, pathological effects and death. The linkages between chemical contaminants and neoplasia or disease susceptibility in marine animals have been relatively well described. Examination of selected fish, shellfish, and turtle mortalities, disease, or incidences of neoplasia with unexplained or incomplete etiologies suggests strong circumstantial evidence for exposure to biotoxins. While many marine biotoxins are known to be tumor promoters in experimental studies, their effects on aquatic animals have only recently been considered. The association of known bacterial animal pathogens with HABs is also an unknown factor. It is critical to relate incidences of disease and neoplasia to the distribution of harmful algal blooms, associated environmental factors, and the potential for accumulation of biotoxins.**

**(20) HISTOPATHOLOGIC INVESTIGATIONS OF LESIONS IN FISHES FROM CHESAPEAKE BAY TRIBUTARIES.**

**VICKI S. BLAZER and CHRISTINE DENSMORE, National Fish Health Research Laboratory, BRD/USGS, 1700 Leetown Road, Kearneysville, WV 25430.**

**In Fall 1996 and again in Spring 1997 watermen reported unusually high incidence of fish with lesions in their catch. The reports came primarily from the Pocomoke River but included other tributaries as well. In August, 1997, two fish kills involving primarily juvenile Atlantic menhaden (*Brevoortia tyrannus*) occurred in the Pocomoke River. This resulted in the closure of portions of the river for commercial or recreational purposes. The appearance of menhaden with lesions (although no actual kills) also led to the closure of portions of King's Creek off the Manokin River and the Chicamicomico River. These closures occurred because the fish kills and the variety of fish lesions have been attributed to *Pfiesteria* or *Pfiesteria*-like toxic dinoflagellates. We have examined over 200 fish, most with lesions, from a number of tributaries. We believe we have three "categories" of fish lesions.**

**1) There are a variety of fish lesions in a variety of fish species. The lesions range from small pinpoint hemorrhages to very deep ulcerations of skin and muscle. Infectious agents in these lesions include *Aeromonas hydrophila, Streptococcus* sp., *Mycobacteria* sp., protozoa, and a fungal agent. These findings suggest some factor or factors which are stressing the fish, causing immunosuppression and/or damage to the epithelium and allowing a variety of opportunistic pathogens to cause infection.**

**2) Mycobacterial infections in striped bass (*Morone saxatilis*). The majority of skin lesions in striped bass are composed of numerous focal granulomas with acid-fast bacteria. Many of these infections are also systemic.**

**3) "Typical *Pfiesteria*" lesion, primarily in menhaden. These are circumscribed areas of dermal erosion, often perianal but also in the epaxial, abdominal or caudal areas. Lesions often penetrate deeply into muscle and sometimes into the abdominal cavity. These lesions are composed of chronic, granulomatous inflammation surrounding aseptate fungal hyphae. We believe this fungus to be *Aphanomyces*, a fungal pathogen causing major losses of wild and cultured, estuarine and freshwater fishes throughout the Asia-Pacific area. The chronic nature of the lesion suggests no direct relationship with an ongoing bloom of toxic dinoflagellates. However, a number of menhaden collected during the Pocomoke fish kills had these deep mycotic lesions overlaid with massive necrosis. This suggests the mycotic lesions were there prior to the fish kill and the massive necrosis could be a result of toxins released during the bloom.**

**These findings will be discussed in relationship to our knowledge of the above infectious agents, potential relationship with toxic algal blooms, current management practices in Maryland and water quality problems in the Chesapeake.**

**(21) BREVETOXIN AS CAUSE OF SUMMER MORTALITY IN COMMON MURRES (*URIA AALGE)* IN CALIFORNIA.**

**DAVID A. JESSUP, JACK AMES, California Department of Fish and Game, Marine Wildlife Veterinary Care and Research Center, 1451 Shaffer Road, Santa Cruz, CA 95060 USA; GREG BOSSART, University of Maimi Medical Center and Miami Sea Aquarium, 4400 Rickenbacker Causeway, Miami, FL 33149 USA; JAMES HILL, CVD-Idexx, 2075 KOVR Drive, West Sacramento, CA 95605 USA; MELISSA CHECHOWITZ, Wildlife Health Center, University of California, Davis, CA 95616 USA; ANDREW DEVOGELAERE, Monterey Bay National Marine Sanctuary, 299 Foam Street, Monterey, CA 93940 USA.**

**In August of 1997 approximately 400 common murres (*Uria aalge*) were found dead in a relatively confined area of the southern end of the Monterey Bay National Marine Sanctuary, California. This dieoff occurred at a time when there was a "red tide" event in the area and shellfish consumption warnings had been issued. The primary gross lesions were serosanguinous rhinitis, severe pulmonary congestion and edema. Immunohistochemical staining showed presence of brevetoxin within lung and lymphoid tissues in patterns similar to those seen in manatees dying of brevetoxicosis. This is the first report of inhaled brevetoxin killing birds on the Pacific coast. In retrospect, brevetoxicosis is suspected in several previous common murre dieoffs in Monterey Bay, California, each involving hundreds of murres and each occurring during months with warmer ocean temperatures. Changes in seawater temperature and quality, which may be related to human activity within the near shore environment, as well as global climate change, El Nino events and natural ocean cycles may influence algal species and abundance.**

## (22) ACAD (1.0): THE ANIMAL CAPTURE AND ANESTHESIA DATABASE.

KEITH BEHELER-AMASS Safe-Capture International, PO Box 206, Mount Horeb, Wisconsin, 53572; DAVID BRUNSON, Dept. of Surgical Sciences, School of Veterinary Medicine, University of Wisconsin-Madison, 2015 Linden Drive West, Madison, WI, 53706.

While expertise on restraint, immobilization, anesthetic, and analgesic techniques exists for many species, access is limited in our current system of information dissemination. Currently, research on these topics is published in a multitude of periodicals and references often with limited circulation. Other techniques are disseminated by personal communication only, without ever being published. Techniques which are associated with mortality and/or morbidity may never be reported or published. Many references provide only dosage information, while information concerning drug concentration, time parameters (induction time, working restraint time, recovery time), confinement, and temperament of the animal are not included. These parameters are often essential to determine applicability of the technique, and to assist in making rational anesthetic decisions. Additionally, there is often urgency for anesthetic information, while locating a source, acquiring original research material, and extracting the needed data can be difficult and time consuming. The Animal Capture and Anesthesia Database (ACAD 1.0) was developed to provide a comprehensive, peer reviewed, central computerized database for animal capture, immobilization, and anesthetic techniques. This database is accessible electronically, affording quick access to information necessary to make informed choices for anesthetic regimens. Database sources include peer reviewed references, textbooks, meeting proceedings, and personal communications of field techniques from primary researchers. Data Records contain: *Information Source, Species, Scientific Name, Drug Used, Dosage, Concentration, Induction Time, Antagonist, Total Restraint Time, Total Time to Recovery, Confinement, Degree of Restraint Prior to Anesthesia, Method of Drug Delivery, Number of Animals Studied, Mortality Rate, Comments, and Warnings.* Records can be sorted by selecting Keywords including: *Species, Family, Drug Used, Pre-Anesthetic Considerations, Drug Regimens Reported to be Safe in Sick or Debilitated Animals, Anesthetic Effects on Clinical Pathology Results, Anesthetic Effects Associated with Pregnancy, Reproduction or Obstetrical Procedures, Oral Medications, Author, and Title.* Users are easily alerted to techniques associated with mortality or morbidity by selecting species specific *Drug and Drug Interaction Warnings.* The base program used to organize ACAD easily produces bibliographies with an additional option of printing reference synopses as well. Access to ACAD will be by internet, hard copy, and distribution of periodically updated discs. The Animal Capture and Anesthesia Database (1) Centralizes rapid access to validated, peer reviewed, detailed species specific anesthetic, immobilization, and capture information. (2) Allows comprehensive species and topic bibliographies to be readily obtained. (3) Alerts and warns of anesthetic related mortality or morbidity, and the effects of anesthetic procedures on clinical pathology results or reproduction. (4) Encourages better data collection and sharing of information among professionals, and (5) Enhances the ability to make informed choices on anesthetic regimens in captive and free-ranging species. For updated information on ACAD visit the website: www.safecapture.com

**(23) LONDON WATERFOWL PROJECT.**

**DEBRA BOURNE, Wildlife Information Network, The Royal Veterinary College, Royal College Street, London NW1 0TU, England, SUZANNE BOARDMAN, Wildlife Information Network, The Royal Veterinary College, Royal College Street, London NW1 0TU, England and JOSHUA DEIN, National Wildlife Health Center, USGS-BRD, Madison, WI 53711.**

**Greater London contains a large number of waterfowl, including for some species populations of national or international importance. A large number of different organisations have responsibility for waterfowl and their habitats in Greater London, including local government bodies, central government bodies, NGO's and private companies. At present there is no organised system in place for the different organisations to communicate with each other with regards to disease contingency planning, waterfowl management strategies, and mechanisms to deal with public concerns.**

**The London Waterfowl Project was initiated by the Wildlife Information Network to develop a communication and information network to assist waterfowl managers in the Greater London area. This will include an Early Warning System to coordinate responses to large scale pollution events or disease outbreaks. Information will be made available primarily through the development of a WILD*Pro* database module on waterfowl, containing a range of population health management data, and rehabilitation strategies.**

**(24) DISEASE MONITORING IN CYBERSPACE: AN INTERNET-BASED SURVEY FOR CONJUNCTIVITIS IN HOUSE FINCHES.**

**BARRY K. HARTUP, GEORGE V. KOLLIAS, Wildlife Health Laboratory, College of Veterinary Medicine; ANDRE A. DHONDT, Cornell Laboratory of Ornithology, Cornell University, Ithaca, NY 14853.**

**The monitoring of disease in large, mobile wildlife populations presents numerous logistical and economic difficulties. Input from trained volunteers across large geographical areas can minimize these difficulties, while providing reliable, quality data. A mailed survey conducted by the Cornell Laboratory of Ornithology since November 1994 has been instrumental in documenting the spread of mycoplasmal conjunctivitis in the eastern house finch (*Carpodacus mexicanus*) population. Beginning in November 1997, a redesigned survey was available to all participants in paper and Internet-based formats, in order to acquire more quantitative data from sites monitored by project participants. In the first five months, 252 participants from 33 eastern states and 2 Canadian provinces provided 780 observations via the Internet. When the species was present, participants counted a mean maximum of 9.3 + 0.4 (+SE) house finches per observation period. 26% of these counts included house finches with conjunctivitis, with a mean maximum of 1.6 + 0.1 diseased finches. The estimated mean prevalence of conjunctivitis at sites with house finches was 5.9% + 0.6%. There were no significant differences in the mean prevalence of disease by month. The highest prevalences were noted to occur in the mid-Atlantic region similar to previously published reports. Significantly more females were observed with conjunctivitis than males in a given count period ($P < 0.05$). Also, significantly more house finches exhibited unilateral conjunctivitis than bilateral lesions ($P < 0.01$). These results will be discussed in light of forthcoming results from the mailed survey.**

**(25) EVALUATION OF A PORTABLE AUTOMATED SERUM CHEMISTRY ANALYZER FOR ASSESSMENT OF HEALTH STATUS OF HARLEQUIN DUCKS, *HISTRIONICUS HISTRIONICUS*, IN THE FIELD.**

**MICHAEL K. STOSKOPF, Environmental Medicine Consortium, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough Street, Raleigh, North Carolina 27606; DANIEL M. MULCAHY, U.S. Geological Survey, Biological Resources Division, Alaska Biological Science Center, 1011 E. Tudor Road, Anchorage, Alaska 99503; DANIEL ESLER, U.S. Geological Survey, Biological Resources Division, Alaska Biological Science Center, 1011 E. Tudor Road,Anchorage, Alaska 99503.**

**Clinical serum chemistry parameters are routinely employed in the evaluation of the health status of free ranging wildlife when physical manipulation of individual animals makes sample collection possible. Unfortunately these parameters have been of limited use as cohort inclusion criteria or for making field decisions primarily because of the logistics required to obtain test results within a useful time frame. A portable analytical chemistry analyzer (VetScan® ) capable of operating off of 12 volt batteries and quickly performing multiple blood tests from a single specimen was made available by Abaxis (1320 Chesapeake Terrace, Sunnyvale, Ca 94089) for the conduct of field assessments of harlequin duck (*Histrionicus histrionicus*) serum chemistry in association with telemetry studies of winter survival in Prince William Sound, Alaska utilizing implanted radio telemetry. The primary concern was to evaluate serum glucose and potassium concentrations as potential indicators of high risk surgical candidates. The prepackaged reagent rotors also included tests for alanine aminotransferase (ALT), albumin, alkaline phosphatase (ALP), amylase, calcium, cholesterol, creatinine, total bilirubin, total protein and urea nitrogen. Preliminary trials using mallard duck blood, suggested the system which was developed for mammalian blood, could be used with avian blood. The 6.9 kg analyzer (29.2 cm long, 15.3 cm wide, 24.2 cm high) was operated off 110 V service supplied by generator. Blood samples were drawn from the jugular vein immediately after morphometric data were collected from each newly-captured duck. Fresh blood was analyzed opportunistically. Samples from each subject were allowed to clot for 30 minutes prior to centrifugation and serum separation. Each serum sample was divided into two aliquots. Fresh serum was analyzed using the VetScan system within hours of sample collection. The other aliquot was frozen at -10 C and transported frozen to North Carolina State University. A matching panel of chemistries was performed for comparison on samples with no visible hemolysis using a Monarch 2000 Analyzer (Instrumentation Laboratories, 113 Hartwell Ave, Lexington, MA 02173). The median differential for glucose values between the two systems (N=83) was 1mg/dl (quartiles -4.1 and 8.5 mg/dl). Median glucose value returned for 105 samples run on VetScan equipment was 328 mg/dl (quartiles 301 and 363 mg/dl). Serum potassium values were too low for quantitation on the Monarch equipment. Analysis on the ABAXIS equipment returned a median of 2.8 mmol/L (N=105; quartiles 2.4 and 3.1 mmol/L).**

**(26) CONTROL OF PRION DISEASES THROUGH GENETICS AND LIVE ANIMAL TESTING: VARIATION BETWEEN SHEEP AND CERVIDS.**

**KATHERINE O'ROURKE, USDA, Agricultural Research Service, Animal Disease Research Unit, 337 Bustad Hall, Washington State University, Pullman, WA 99165.**

**The prion diseases of ruminant animals include sheep scrapie, bovine spongiform encephalopathy and chronic wasting disease of cervids. Sheep scrapie and chronic wasting disease each represent a family of disorders; clinical signs, histologic lesions, and prion distribution vary with strain differences, the host species, and individual host genetics. The prion genetic changes associated with relative susceptibility to sheep scrapie and the pre-clinical, live animal tests for sheep scrapie are being validated as control measures for the disease in U.S. sheep. Application of those findings to cervids has been challenging. The current status of genetic susceptibility of elk and mule deer to CWD and the prospects for a live animal test in captive elk will be presented.**

**(27) EPIDEMIOLOGY OF CHRONIC WASTING DISEASE IN NORTHEASTERN COLORADO DEER AND ELK POPULATIONS.**

**MICHAEL W. MILLER, Colorado Division of Wildlife, Wildlife Research Center, 317 West Prospect Road, Fort Collins, Colorado 80526-2097; TERRY R. SPRAKER, Colorado State University Diagnostic Laboratory, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, Colorado 80523; and ELIZABETH S. WILLIAMS, Department of Veterinary Sciences, University of Wyoming, 1174 Snowy Range Road, Laramie, Wyoming 82070.**

**Chronic wasting disease (CWD) is a naturally-occurring transmissible spongiform encephalopathy of native free-ranging deer (*Odocoileus* spp.) and elk (*Cervus elaphus nelsoni*) populations in northeastern Colorado and southeastern Wyoming, USA. We used harvest surveys to estimate CWD prevalence and study select aspects of its epidemiology in northeastern Colorado. Brain tissues from over 2,500 deer and elk harvested throughout northeastern Colorado during 1995-1997 were examined microscopically after staining with anti-prion protein immunostains, as well as hematoxylin and eosin; deer and elk showing positive immunostaining, with or without typical light microscopic lesions, were regarded as CWD-affected. Because prevalence estimates did not differ among the three years studied, we pooled data for other comparisons. Survey data revealed that CWD was at least five times more prevalent in deer than in elk in northeastern Colorado during 1995-1997. Similarly, geographic distribution of CWD was wider among deer than among elk; the primary focus of CWD in free-ranging deer appeared to be the eastern half of Larimer County, although a few positive deer were also detected along the South Platte River corridor. Contrary to previous observations derived from clinical case submissions, CWD prevalence among male and female deer did not differ. Age distributions of positive cases among male and female deer did not differ from age distributions of unaffected deer harvested. Our data support the hypothesized importance of lateral transmission in CWD epidemiology; moreover, they will serve as a pretreatment baseline for management experiments designed to reduce CWD prevalence and distribution in northeastern Colorado.**

**(28) SURVEY FOR CHRONIC WASTING DISEASE IN CAPTIVE AND FREE-RANGING ELK AND DEER IN ALBERTA. CANCELLED**

**M.J. PYBUS, Alberta Fish and Wildlife, 6909-116 Street, Edmonton T6H 4P2 and D.K. ONDERKA and S. HONOUR, Alberta Animal Health, 6909-116 Street, Edmonton T6H 4P2.**

## REPLACE WITH:

**(28) MENINGEAL WORM (*PARELAPHOSTRONGYLUS TENUIS*) IN TERRESTRIAL GASTROPODS OF MICHIGAN'S UPPER PENINSULA.**

**PAMELA J. BOPPEL and WILLIAM M. SAMUEL, Department of Biological Sciences, University of Alberta, Edmonton.**

**Moose were reintroduced to Michigan's upper peninsula in 1985 and 1987. According to reproductive rates of radio-collared females, results from computer modeling projected the population to reach 300 animals by 1995. However, recent aerial surveys revealed that population growth has not progressed as expected. Meningeal worm has caused over 30% of diagnosed deaths of moose in Michigan's upper peninsula and probably contributed to the slow population growth. A total of 9,552 terrestrial gastropods were collected from three broad habitat types and in areas where deer congregated, or areas inhabited by moose. Objectives of this study were to determine what species of intermediate hosts are important for the transmission of meningeal worm, and if particular habitat types pose greater risk for moose to acquire infection. Overall, less than 1% of the gastropods were infected and most had three larvae or fewer. Lowland areas where deer congregated tended to have higher numbers of gastropods in general and higher numbers of infected gastropods than other areas. Infected gastropods were also found in areas inhabited by moose therefore, moose are vulnerable to infection within their preferred range. Some potential intermediate host gastropods were associated with particular habitat types. For example, *Zonitoides nitidus* and *Cionella lubrica* were most often associated with wet, open areas and could be of importance for transmission of meningeal worm to moose.**

**(29) THE HOST RANGE OF CHRONIC WASTING DISEASE IS ALTERED UPON PASSAGE IN FERRETS.**

**JASON C. BARTZ, RICHARD F. MARSH, DEBBIE I. MCKENZIE and JUDD M. AIKEN**
**Department of Animal Health and Biomedical Sciences. University of Wisconsin, Madison 53706.**

**Chronic wasting disease (CWD), a member of the transmissible spongiform encephalopathies (TSEs), was first identified in captive mule and black-tail deer in 1967. Due to the failure to transmit CWD to rodents, we investigated the use of ferrets (*Mustela putorius furo*) as a small animal model of CWD. We transmitted CWD into ferrets with an incubation period of 17-21 months on primary passage. The incubation period shortens to 5 months by third ferret passage. The brain tissue of animals inoculated with ferret-passaged CWD exhibits spongiform degeneration and reactive astrocytosis. Western blot analysis of ferret-passaged CWD demonstrated the presence of PrP-res that is similar in mobility and abundance to other ferret-passaged TSEs. Unlike mule deer CWD, ferret-passaged CWD can be successfully transmitted to Syrian golden hamsters (*Mesocricetus auratus*). Increasing the passage number of CWD in ferrets was found to increase the pathogenicity of the agent for hamsters. Subsequent passages of hamster CWD shorten the incubation period and select for a strain that resembles the 263K/HY strains of hamster-adapted TSEs in incubation period, clinical signs, and PrP-res properties.**

**(30) BRUCELLOSIS IN YELLOWSTONE NATIONAL PARK BISON (*BISON BISON*): QUANTITATIVE SEROLOGY AND TISSUE LOCALIZATION OF INFECTION.**

**THOMAS J. ROFFE, Midcontinent Ecological Science Center, FWP Bldg, Montana State University, Bozeman, MT 59717; JACK C. RHYAN, National Veterinary Services Laboratory, PO Box 844, Ames, IO 50010; KEITH AUNE, Montana Fish Wildlife and Parks, Montana State University, Bozeman, MT 59717; L. MICHAEL PHILO, Animal and Plant Health Inspection Service, 9439 Owl Way, Bozeman, MT 59715; DARLA R. EWALT, National Veterinary Services Laboratory, PO Box 844, Ames, IO 50010; and T. GIDLEWSKI, National Veterinary Services Laboratory, PO Box 844, Ames, IO 50010.**

**We collected complete sets of tissues from 37 Yellowstone National Park (YNP) female bison killed as a result of management actions. Twenty-eight were seropositive adults (27) or calf (1). Samples from all 37 bison were collected to maximize the opportunity to culture *Brucella abortus.* Samples were processed following the research sampling protocol established by the Greater Yellowstone Interagency Brucellosis Committee for bison. Thirty different tissues, fluids and swabs from each animal were cultured for *Brucella* using macerated whole tissues plated onto 4 *Brucella* selective media and incubated with added $CO_2$ for two weeks. Twelve seropositive female bison (43%), including the 1 calf, cultured positive from one or more tissues for *Brucella abortus.* Culture positive adult females were characterized serologically by high quantitative titers. All 11 measured less than .115 on the Particle Concentration Fluorescent Immuno Assay (PCFIA), and 9 (82%) were less than .080. Complement fixation (CF) titers were above 3+ at 1:40 in 10 of 11 (91%). All 11 culture positive females had either a PCFIA $\leq$.080 or a CF reaction $\geq$4+ at 1:80. However 6 (38%) seropositive, but culture negative, adult females were also high titered. Eleven of the 12 *Brucella* cultures were biovar 1, and one was biovar 2. Tissues culture positive for *Brucella abortus* were restricted to lymph nodes with the exception of one animal that recently aborted. She had a retained placenta, was culture positive in many tissues, and was shedding *Brucella* in feces. Our findings on the relationship between serology and culture, and the tissue localization of infection is similar to that reported in chronically infected cattle herds.**

**(31) SAFETY AND EFFICACY OF *BRUCELLA ABORTUS* VACCINE STRAIN RB51 IN CAPTIVE ADULT COW ELK.**

**TERRY J. KREEGER, Wyoming Game and Fish Department, 2362 Highway 34, Wheatland, WY 82201, MICHAEL MILLER, MARGARET WILD, Colorado Division of Wildlife, Wildlife Research Center, 317 W. Prospect Rd., Fort Collins, CO 80526, PHILIP ELZER, Louisiana State University, Baton Rouge, LA.**

**Brucellosis is endemic in elk (*Cervus elaphus*) and bison (*Bison bison*) in the Greater Yellowstone Area. For economic and health purposes, a brucellosis eradication program began in 1934 with the goal of eliminating brucellosis in domestic cattle from the United States. The presence of brucellosis in wildlife creates a conflict with this goal as well as it impacts the production of these wild populations. *Brucella abortus* Strain RB51 vaccine is a laboratory-derived rough mutant of virulent *B. abortus* Strain 2308. Its advantage over other *Brucella* vaccines is that it does not give false positives on standard tests. One potential problem with any *Brucella* vaccine is the possibility of inducing abortion. The purpose of this study was to determine if Strain RB51 vaccine induced abortion and then protected against abortion upon challenge during the time period that free-ranging elk would normally be vaccinated and subsequently exposed to virulent *Brucella.* Fourteen captive adult elk were diagnosed pregnant using pregnancy-specific protein analysis. The elk were randomly and equally divided into Test and Control Groups. On February 26, 1998 the Test Group was vaccinated intramuscularly with $10^{10}$ cfu of Strain RB51 vaccine. None of the elk aborted due to vaccination. On April 7, 1998, all elk were challenged with $10^{7}$ cfu of *B. abortus* Strain 2308 administered conjunctivally in a split dose in both eyes. The efficacy portion of this study will be completed by June 1998.**

**(32) BRUCELLOSIS IN EUROPEAN BROWN HARES (*LEPUS EUROPAEUS*) IN DENMARK. A RESERVOIR FOR PORCINE BRUCELLOSIS?**

**HANS H. DIETZ, ERIK RATTENBORG, THOMAS H. ANDERSEN, Danish Veterinary Laboratory, Department of Poultry, Fish and Fur Animals, Section for Small Animal and Wildlife Pathology, 2 Hangovej, DK-8200 Aarhus N, Denmark; and STEEN B. GIESE, Danish Veterinary Laboratory, Department of Microbiology, 27 Bülowsvej, DK-1790 Copenhagen V, Denmark.**

***Brucella suis, biotype 2* is a gram negative bacteria characterized by a narrow host spectre i.e. only infecting pigs and brown hares (*Lepus europaeus*). Brucellosis is a rare disease in Danish pigs. Eight outbreaks have been reported between 1929 and 1965. The ninth outbreak occurred in November 1994. This outbreak among outdoor pigs led to increasing public awareness of the possible role of hares as carriers and farmers and hunters were urged to refer hares from this area of low population density for laboratory examination. Brucellosis in Danish brown hares is also a rare disease and the first case was confirmed in 1951. Experimental studies showed that pigs eating internal organs from infected brown hares were easily infected. *Brucella suis, biotype 2* was isolated infrequently from brown hares until 1985. Serological surveys in the nineteen fifties showed a prevalence of less than 4 percent. The next case was seen in October 1995 in a hare shot approximately 30 kilometers from the point of outbreak among outdoor pigs in October 1994. In November 1997 another hare shot approximately 10 kilometers from the point of outbreak in 1994 was found to be infected with *Brucella suis, biotype 2*. No more cases among pigs have been seen. Serological surveys in hares from the area showed no positive cases. These findings indicate that brucellosis among hares has been present for at least 50 years and although keeping pigs outdoor has been discouraged for decades, the potential for exchange of disease between wildlife and domestic animals still exists even with a sporadic infection like brucellosis in hares. Subsequently the Danish State Veterinary Service has implemented new regulations implicating fencing of all outdoor pig herds.**

**(33) DISSEMINATED *ACTINOMYCES PYOGENES* INFECTION IN A WILD FREE-RANGING WHITE-TAILED DEER, AND POSSIBLE EMERGENCE OF A NEW DISEASE SYNDROME IN DEER; FOOTWARTS (PAPILLOMATOUS DIGITAL DERMATITIS/INTERDIGITAL DERMATITIS).**

**KERRY BEHELER-AMASS, Wildlife Health Program, Wisconsin Department of Natural Resources (WDNR), P.O. Box 7921, Madison WI 53707; KATHY STRELOW, Wisconsin Animal Health Laboratory (WAHL), Madison, WI 53711; KAREN WOODS, WAHL, Madison, WI 53711; ANDY NELSON, WDNR, Horicon, WI 53032; NEIL WISELEY, Mayville Animal Clinic, Mayville, WI 53050.**

**The aerobic bacterium *Actinomyces pyogenes*, normally found as a commensal organism on exposed mucosal surfaces of cattle, sheep, goats, and deer, can invade a variety of tissues. It is associated with mastitis, metritis, septicemia, and mammary, pulmonary, or foot abcesses, and has recently been associated with intercranial abcessation/suppurative meningoencephalitis infections in white-tailed deer (*Odocoileus virginianus*). Few reports, however, identify *A. pyogenes* as a primary pathogen of deer. It is more commonly identified as a secondary or end-stage infection, and its isolation may mask other disease symptoms. Six dead adult deer, observed in a localized area of Wisconsin (WI) within a four month period, had no apparent external anomalies except abnormal gross foot lesions. Hoof rot was grossly diagnosed from two deer feet, and *A. pyogenes* was isolated from multiple tissues of a severely emaciated adult buck carcass. Localized outbreaks of hoof rot in WI deer are quite rare. Differential diagnosis included infectious virulent foot rot, bovine interdigital necrobacillosis, deer footworm, infectious bulbar necrosis, bluetongue, ovine interdigital dermatitis, and papillomatous digital dermatitis (PDD), a new disease entity in dairy cattle known as footwarts. PDD prevalence approaches 60% among dairy herds that surround this localized WI deer area. With the high number of deer around these dairy herds, comingling in pastures or at feed bunks possibly allowed PDD transmission to deer. The unusually warm, wet, snowless WI 1997-98 winter likely enhanced disease transmission. Further investigations of possible PDD in these localized deer are underway. Aspects of PDD will be discussed, including disease epidemiology, clinical and histologic signs, causative agents, possible transmission to deer, and PDD diagnosis in captive or free-ranging deer known to date. One objective of this presentation is an open discussion of the implications and potential of this disease for deer.**

**(34) PREVALENCE AND DISTRIBUTION OF TOXOPLASMOSIS IN URBAN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*).**

**KARMEN M. HOLLIS, University of Illinois, College of Veterinary Medicine, 2001 S. Lincoln Ave., Urbana, IL 61801; LAURA L. HUNGERFORD, University of Nebraska, Great Plains Veterinary Education Center, Box 187, Clay Center, NB, 68933; J.P. DUBEY, Parasite Biology and Epidemiology Laboratory, Agriculture Research Service, U.S. Department of Agriculture, Beltsville, MD 20705; CHRIS ANCHOR and JAMES CHELSVIG, Forest Preserve District of Cook County, Route 4 Box 178, Elgin, IL 60120.**

**White-tailed deer (*Odocoileus virginianus*) can harbor the protozoan parasite known to cause toxoplasmosis in humans. Data on seroprevalence in deer may serve as a useful biomonitoring tool. Sera were collected from 380 deer in Cook County, Illinois from November through April, in 1995-96 and 1996-97. Samples were tested by serum agglutination for *Toxoplasma gondii* at dilutions of 1:25, 1:50, 1:500, and $\geq$1:500. One-hundred eighty-seven (49.2%) deer serum samples tested positive for *Toxoplasma gondii*. There was no significant difference between the sexes (Female Prev 52.6%, Male Prev 49.6%, PR=1.2, p=0.70). There was a significant trend of increasing prevalence with age (Fawn Prev 25.8%, Yearling Prev 44.1%, Adult Prev 61.4%). A significant difference in seroprevalence was detected among the 12 sampling locations (range 17.8% to 80%, $p<0.001$) and between the 2 live-capture sites (Des Plaines Prev=68.8%, Palos Prev=33.3%, PR=2.1, $p<0.001$). The difference between the two capture locations remained the same when adjusted for sex and age. Locations of seropositive and seronegative deer were mapped using a geographical information system (GIS) to analyze demographics, spatial, and habitat differences. Information from this study will allow wildlife biologists to include public health considerations in urban deer management as well as provide health professionals with specific data on the prevalence of toxoplasmosis in different types of preserves in Cook County, IL.**

**(35) CYCLIC PATTERNS OF HEMORRHAGIC DISEASE IN GEORGIA WHITE-TAILED DEER.**

**D.E. STALLKNECHT and V.F. NETTLES, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, Georgia, 30602.**

**Hemorrhagic disease in white-tailed deer, which is caused by viruses in the epizootic hemorrhagic disease virus and bluetongue virus serogroups, has been reported to occur on a 2 to 3 year cycle in enzootic areas of the southeastern United States. This pattern was confirmed by examining clinical and serological data from white-tailed deer in Georgia collected from 1981 to 1997. The observed cycles could be attributed to infection with EHDV-1, EHDV-2, BTV-10, BTV-11, and BTV-13, but only EHDV-2 was represented on every outbreak year. To better understand these cycles, we constructed a simple model based on concurrent 3 and 8 year cycles. Predicted changes in antibody prevalence based on this model follow observed changes in the antibody prevalence and may explain why outbreaks can occur at 2 as well as 3 year intervals.**

**(36) SELECTIN REGULATION IN WHITE-TAILED DEER INFECTED WITH EPIZOOTIC HEMORRHAGIC DISEASE VIRUS.**

**ELIZABETH W. HOWERTH, Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602; MOLLY MURPHY, Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602; and DAVID E. STALLKNECHT, Southeastern Cooperative Wildlife Disease Study, University of Georgia, Athens, GA 30602.**

**The epizootic hemorrhagic disease viruses (EHDV) cause devastating disease in white-tailed deer (WTD) which is commonly referred to as hemorrhagic disease. These viruses infect endothelial cells resulting in cell death and activation of the coagulation/fibrinolytic systems. Infection of the endothelium no doubt triggers other events that either potentiate or ameliorate the disease caused by these viruses. P and E selectin are proteins that are expressed on the surface of the endothelium (P and E) and platelets (P) when these cells are activated by various insults. When expressed these proteins direct the migration of cells involved in cellular immunity, inflammation, and hemostasis to sites of vascular injury. Because both endothelium and platelets are involved in the pathogenesis of EHDV infection, selectin expression may be involved in the pathogenesis and clinical outcome of EHDV infection. The goal of this study was to characterize P and E selectin expression in WTD and WTD endothelium infected with EHDV. Selectins were up-regulated in both platelets and endothelium during EHDV infection. However, up-regulation was low in EHDV infected WTD endothelium when compared to up-regulation in EHDV-infected endothelium from other species and to up-regulation as the result of non-viral activators. It is speculated that relatively poor up-regulation of selectins during EHDV infection may be partially responsible for the extreme susceptibility of WTD to EHDV.**

**(37) MODEL HEALTH PROTOCOL FOR IMPORTATION OF WILD ELK FOR RESTORATION.**

**JOSEPH L. CORN and VICTOR F. NETTLES, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602.**

**Historically, elk were indigenous to much of the eastern United States, and there currently is considerable interest in restoration of elk into suitable remaining habitat. Michigan, Wisconsin, Pennsylvania, Arkansas and Kentucky have made elk introductions, and several other states are considering restoration programs. Public interest in this concept is increasing, and wildlife managers are in the process of evaluating the possible positive and negative elements of elk reintroduction. Among the negative elements is the concern of the potential for introduction of diseases and parasites that would be injurious to wildlife, domestic animals, or humans. Prior relocations of elk or red deer have been attributed as the vehicle for introduction of *Fascioloides magna* to Italy, the abomasal parasites *Spiculopteragia spiculoptera, S. asymmetrica,* and *Ostertagia leptospicularis* to Argentina, New Zealand, and/or Texas, *Onchocerca cervipedis* to Poland, and *Elaphostrongylus cervi* to Canada.**

**Although it is impossible to create a "sterile animal" without accompanying organisms, it is feasible to reduce the risks by not moving animals that are infected with known pathogenic agents. This Model Protocol was prepared to give an overview of health considerations associated with reintroduction of elk, with an emphasis on the southeastern United States. Diseases and parasites reported in elk worldwide were evaluated from two perspectives. First, a qualitative estimate was made of the ability of the agent to be introduced and to become established in the release environment. Second, if there was a good possibility that the organism could become established in the release area, the potential pathological consequences for elk and other wildlife, domestic animals, and human beings were assessed. The results of these evaluations were used to classify disease agents and parasites as low risk, unknown risk, and high risk. One hundred seventy-five disease agents and parasites were classified as low risk and eight as unknown risk. High risk diseases and parasites were chronic wasting disease, brucellosis, bovine tuberculosis, *Dermacentor andersoni, Ixodes pacificus,* and psoroptic mange. A five-stage process to reduce the risk of introduction of high risk disease agents and parasites was developed and includes (1) evaluation of the health status of source populations, (2) quarantines, (3) physical examination and diagnostic testing, (4) restrictions on translocation of animals from certain geographic areas or populations, and (5) prophylactic treatment.**

**(38) HEALTH MONITORING OF FLORIDA'S ENDANGERED KEY DEER.**

**CHARLOTTE F. QUIST,.Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602 ;VICTOR F. NETTLES, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602; and THOMAS J. WILMERS; National Key Deer Refuge, Big Pine Key, Florida.**

**After paratuberculosis was diagnosed in an emaciated 2.5-year-old Key doe, a field investigation was initiated to determine whether the disease had become established in Florida's herd of endangered Key deer. Disease surveillance methods used included obtaining serology and feces from live deer and necropsy examination of Key deer killed by automobiles or found dead of unknown causes. Thus far, total of 61 deer have been necropsied, and 32 live deer have captured and sampled. To date, surveillance has failed to give any indication that paratuberculosis is established in the Key deer herd. Serum samples have been uniformly negative for antibodies to *Mycobacterium paratuberculosis* via agar gel immunodiffusion and cultured fecal samples have found no trace of the organism.**

**Additional mortality factors have been recognized in the herd. The leading cause of mortality for Key deer remains automobile fatalities with 43 of 61 deer (70.5%) determined to have been hit-by-automobiles prior to death. The most significant secondary mortality factor in Key deer is the cranial abscess syndrome that was identified in 12 of 16 mature bucks over 2 year of age which equates to 46% of that sex and age group. Five of these 16 bucks had also been hit by automobiles. *Actinomyces pyogenes* was isolated out of 14 of 16 of these animals. Three bucks with brain abscesses had apparently drowned in canals. Haemonchosis was identified as a significant factor in deer <1 year of age. Eight of 15 animals <1 year of age were diagnosed with clinical haemonchosis. Haemonchosis had not previously been considered a threat to Key deer. Other conditions diagnosed included salmonellosis, mandibular osteomyelitis, congestive heart failure, and triplets.**

**(39) CASE REPORT: FIRST REPORT OF *LEYOGONIMUS POLYOON* (TREMATODA:STOMYLOTREMATIDAE) IN AMERICAN COOT (*FULICA AMERICANA*) IN NORTH AMERICA.**

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**On September 30, 1997, an avian mortality event was reported at the 6000 acre Shawano Lake in Shawano, WI. By November 13 over 11,000 American coot and more than 1,000 waterfowl were dead. Examinations of carcasses at the USGS, BRD, National Wildlife Health Center in Madison, WI, found that the waterfowl were infected with *Sphaeridiotrema globulus*, an intestinal trematode which causes intestinal hemorrhage and death. The coot mortality, however, was caused by *Leyogonimus polyoon*, an exotic trematode never reported from North America. This parasite has been reported from Eurasian coot (*Fulica atra*) and common moorhen (*Gallinula chloropus*) in Europe and Russia. Retrospective examination of coot tissues from a 1996 mortality event (>1400 coot) at Shawano Lake revealed that *L. polyoon* was present. It is not known how extensive this parasite's range is within North America; however, it may possibly spread to other locations with the migration of coot, providing a suitable snail intermediate host is present.**

**(40) ECTOPIC PREGNANCY IN THE BOBCAT.**

**MILLER, D.L., Division of Comparative Pathology, University of Miami School of Medicine, Miami Florida 33136, B.J. WOODY, Animal Health Center, Franklin, Tennessee 37064, B.D. LEOPOLD, Department of Wildlife and Fisheries, Mississippi State, Mississippi 39762 and E.D. STYS, Florida Fish and Game, Tallahassee, Florida 32399.**

**A bobcat (*Felis rufus*) from the Mississippi State University Captive Bobcat Research Facility was found to have an ectopic pregnancy during a routine pregnancy check. The fetus was approximately 3 cm in diameter and adjacent to the right horn at about mid-point on the medial side. The fetus was encased in a vascular membrane which was continuous with the uterine vasculature. An ultra sound examination performed 2 weeks later revealed a reabsorbing embryo with the vasculature present but no fetal bulge. Total resorption of the fetus and associated membranes was verified by laparoscopic examination during a subsequent pregnancy check later in that breeding season.**

**Prior to and after this occurrence, the female had successfully given birth to litters ranging from 1-5 kittens each. Greater than 100 laparoscopic pregnancy checks had been performed on the bobcats at the captive facility since 1983 with no prior ectopic pregnancy occurring.**

**(41) VACCINE-INDUCED CANINE DISTEMPER IN A GRAY FOX.**

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**A captive juvenile gray fox (*Urocyon cinereoargenteus*) presented semi-comatose with tonic and clonic seizures and respiratory distress 19 days post vaccination with a commercial modified-live virus canine 5-way vaccine. The animal was previously seronegative for canine distemper as verified from blood collected on the day of vaccination. Necropsy revealed a mild to severe, multifocal, nonsuppurative encephalitis and severe, acute suppurative bronchopneumonia. The encephalitis was most pronounced in the mid-brain region, namely the pons and thalamus. Cytoplasmic or glassy eosinophillic intranuclear inclusion bodies were observed in the neurons. Many neurons displayed central chromatolysis. Additionally, gliosis and satellitosis were observed around neurons. Meningeal involvement was not noted. Incidentally, *Pasteurella multocida* was cultured from the lung; however, the pneumonia most likely represented a secondary finding. The clinical and microscopic neurological findings are consistent with vaccine-induced canine distemper. An interesting aspect of this case is the somewhat unusual location (i.e. they were limited to the midbrain) for the lesions.**

**(42) OCULAR ABNORMALITIES IN DUCKS EXPOSED TO HYPERSALINE WATER FROM THE PLAYA LAKES REGION OF SOUTHEASTERN NEW MEXICO.**

**P.E. MILLER, School of Veterinary Medicine, University of Wisconsin; L.A. BAETEN, National Wildlife Health Center, Madison, Wisconsin; S. SISSLER, School of Veterinary Medicine, University of Wisconsin; F.J. DEIN, National Wildlife Health Center, Madison, Wisconsin; R.R. DUBIELZIG, School of Veterinary Medicine, University of Wisconsin; J. PAUL-MURPHY, School of Veterinary Medicine, University of Wisconsin; C.J. MURPHY School of Veterinary Medicine, University of Wisconsin.**

**Purpose. Conjunctivitis and cataracts have been observed in dead wild ducks naturally exposed to hypersaline water (dissolved ion content 10X sea water) in the playa lakes of southeastern New Mexico. These lakes serve as discharge sites for water used in potash mining. We sought to determine whether visual impairment could have caused affected birds to be reluctant leave these lakes and hence contributed to their death. Methods. Detailed ophthalmic and serum/ocular electrolyte investigations were performed on 4 groups of living mallard ducks exposed to either tap water, or 100, 80 and 70% of normal concentration of playa lakes water. Additionally, the eyes of 20 anesthestized or dead mallard ducks were exposed to hypersaline and/or tap water to determine the cause and reversibility of the lens changes. Results. Severe chemosis and conjunctivitis occurred in birds exposed to all concentrations of playa lakes water but cataracts were noted only in anesthetized, moribund or dead birds in which the eye was immersed in hypersaline water. Cataracts appeared to be the result of fluid/electrolyte shifts within the eye and were at least partially reversible upon immersion in tap water. Conclusions. Hypersaline water from these lakes causes substantial ocular surface irritation in exposed birds but this alone is insufficient to substantially interfere with flight. Cataracts observed in ducks collected on mortality surveys appear to be attributable to terminal immersion of the eye in hypersaline water rather than antemortem events. Salt poisoning/dehydration (fresh water deprivation) is the most likely cause of mortality on these lakes.**

**(Funding for this project provided by USGS Biological Resources Division and Hewitt Ophthalmic Research Fund.)**

**(43) MITIGATING FOR BREEDING BIRDS THAT NEST AT SELENIUM CONTAMINATED EVAPORATION BASINS.**

**ANDREW G. GORDUS, JEFF L. SEAY, and SCOTT TERRILL, H. T. Harvey & Associates, 423 W. Fallbrook St., Suite 206, Fresno, CA 93711 USA.**

**Selenium contaminated tile-drainage agricultural evaporation basins have caused reproductive impacts to recurvirostrids that feed and nest at these sites. The operators of these basins have modified the design and management of the basins in an effort to discourage shorebird use and nesting on these sites. Modified designs include: deep water depths, steep banks with 3:1 slopes, no islands or windbreaks, level bottoms, and plastic lined or "stacked" rip-rap banks for erosion control. Management strategies include rapid filling and drawdowns of the ponds, intensive hazing during the pre-breeding and breeding seasons, vegetation control on the banks and ponds, and clean, smooth, scraped dikes and banks. Hazing efforts include: propane cannons, cracker shells, bird bombs, mylar flagging, airboat, and vehicles. Other management strategies include modifying sites adjacent to the basin that attracted birds to the basin area or by providing a freshwater mitigation wetland adjacent to the evaporation basin. The following presents nest numbers and egg selenium results from two evaporation basins that have implemented the previously stated mitigation efforts.**

**(44) GRANULOCYTIC EHRLICHIOSIS IN A CAPTIVE REINDEER AND STRAY CAT FROM WISCONSIN.**

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**Granulocytic ehrlichiosis (GE) is caused by rickettsiae of the *Ehrlichia phagocytophila* genogroup. In the midwestern United States, GE is transmitted by the deer tick, *Ixodes scapularis*, which probably serves as the primary vector. GE has been reported in humans, horses, dogs, ruminants, and small mammals. However, the complete range of susceptible mammalian hosts is unknown. We report two cases of severe GE involving a captive reindeer and stray cat from northwestern Wisconsin. Both animals presented with high fever and extreme lethargy and the reindeer had marked thrombocytopenia. Peripheral blood smears showed characteristic ehrlichial morulae within granulocytes. Both animals recovered promptly after therapy with tetracycline. Immunoserologic testing of convalescent sera by IFA, using *E. equi* as substrate, revealed titers of 1:256 and 1:512 for the cat and reindeer, respectively. GE was confirmed by PCR amplification of ehrlichial 16S rDNA from the peripheral blood of both animals. Sequencing of the 1420 base-pair PCR products revealed the cat-*Ehrlichia* to be 100% homologous to the sequence reported for the agent of human granulocytic ehrlichiosis (HGE). The reindeer-*Ehrlichia* sequence was 99.8% homologous to the agent of HGE and had the same base substitutions seen in a putative GE variant found in asymptomatic white-tailed deer from Wisconsin, Maryland, and Rhode Island. To our knowledge these are the first reported cases of naturally acquired GE in a cat and reindeer and demonstrates that the GE variant is capable of causing severe illness.**

**(45) GENDER VARIATION IN ELEPHANT SEAL (*MIROUNGA ANGUSTIROSTRIS)* BLOOD PARAMETERS DURING THE BREEDING SEASON.**

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**The ability to withstand repetitive, prolonged breathhold dives is a critical element of foraging success in pinnipeds. Physical health of individuals therefore may have a direct impact on a seal's survival and reproductive success by its effects on diving performance. To evaluate blood parameters as indicators of health and performance, we compiled standard veterinary medical profiles (hematological and serum biochemical analyses) for 52 free-ranging northern elephant seals (*Mirounga angustirostris*) at the California Channel Islands. We chemically immobilized 39 adults (29 males, 10 females), 7 juveniles (4 males, 3 females), and 6 weaned pups (4 males, 2 females) during the breeding season (1993-1996) for attachment of telemetry instruments and collection of blood samples. Most blood values were within reference ranges published for phocid seals. However, adult males differed significantly ($p<0.05$) from adult females for a suite of parameters associated with inflammation, infection or other stressors. Adult males had significantly higher white blood cell and neutrophil counts than females, lower numbers of lymphocytes and lower serum albumin and iron. No significant differences were found between male and female juveniles or weaned pups for these parameters. These results suggest that adult males were suffering from subclinical inflammatory or infectious conditions, probably associated with intrasexual competition. Creatine kinase (CK) levels were also significantly higher in adult males, evidently a further reflection of the stress associated with aggression among breeding males. These results suggest that the relatively poorer post-breeding season dive performance of adult males compared with adult females may be related to sexual differences in their health as a result of intrasexual combat and competition for mates during the breeding season.**

## (46) THE RACCOON DOG IS A POSSIBLE RESERVOIR FOR CANINE HEARTWORM?

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***Dirofilaria immitis*** **is known to be one of the canine parasites, but also to infect naturally several species. The parasite has been found in the heart of carcasses of the raccoon dog, *Nyctereutes procynoides*, which is one of the Canoidea and distributed in East Asia. The animals have been spreading their habitat into human-dwelling spheres, because of land exploitation at the suburbs of the big cities in Japan. This suggests the possibility that the animal may be a reservoir for *D. immitis*. The prevalences of the parasite in raccoon dogs were 6.9 and 13 % at Nishi-Tama and Kanagawa, respectively. These values were obviously lower than those that have been reported in dogs. Thus, to examine the susceptibility to the infection, 3 raccoon dogs and 2 control dogs were experimentally infected 4 times with 25 infective larvae and necropsied at 180 days after infection. The averages of the worm burden in the raccoon dogs and the control dogs were 2.3 and 24.5 %, respectively. Antibody titers in sera from the animals were determined by ELISA using excretory and secretory antigen from adult worm culture. The raccoon dogs showed relatively earlier immune response to *D. immitis* infection than the dogs did. Furthermore, two raccoon dogs were infected with 100 L3. Microfilaremia began at 187 and 201 days after infection and disappeared in Winter on the basis of seasonal periodicity. However, in next Spring and Summer, microfilaremia did not occur. These findings indicate that the raccoon dog is hardly thought to be an appropriate reservoir for this parasite, but plays the role of a transient reservoir.**

**(47) MONITORING HEALTH OF TROPICAL PELAGIC SEABIRDS IN HAWAII.**

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**The Honolulu Field Station (HFS) was established in 1992 to assist Federal and state agencies in wildlife health related matters. Since then, the station has used clinical and microscopic pathology, toxicology, microbiology and epizootiology to evaluate seabird health. From 1992-1997, the HFS examined tissues from >1800 individuals comprising 15 species of seabirds. Initial surveys involved establishing baseline physiologic values for representative species. Subsequent efforts included mortality surveys and applied research to address management needs. Bacterial infections and dehydration were responsible for a die-off of wedge-tailed shearwater (*Puffinus pacificus*) chicks in 1994. Unusually large numbers of Laysan albatross (*Diomedea immutabilis*) adults died on Midway in 1995 from acute anemia of unknown origin. Lead poisoning from paint chips and necrotizing enteritis are major causes of mortality in Laysan albatross chicks on Midway Atoll. In 1997, lead mitigation efforts on Midway were evaluated and management guidelines provided to the US Fish and Wildlife Service. Other diagnostic findings were incidental and included the first documentation of endemic hemoparasites in Hawaii.**

**(48) NECROPSY FINDINGS IN GREAT HORNED OWLS (*BUBO VIRGINIANUS*) FROM ONTARIO, 1990-98.**

**G. DOUGLAS CAMPBELL, Canadian Cooperative Wildlife Health Centre, Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada N1G 2W1. and CHRISTINE BISHOP, Canadian Wildlife Service, Environment Canada, PO Box 5050, 867 Lakeshore Rd., Burlington, Ontario, Canada L7R 4A6.**

**A retrospective analysis was done of necropsy findings in great horned owls (*Bubo virginianus*) found sick or dead in Ontario and necropsied by the Ontario Veterinary College or Canadian Cooperative Wildlife Health Centre during the period 1990-98. Fifty-six birds were in emaciated body condition and 38 were in fair to good body condition. In non-emaciated birds, principal lesions identified included trauma (28), Herpesvirus (3), inflammatory and degenerative conditions (2) while no final diagnosis was made in 3 cases. In emaciated birds, significant findings included chronic traumatic lesions (18), bumblefoot (10), lymphosarcoma (3), tuberculosis (1), miscellaneous conditions (9), and no diagnosis (14). Sarcocystis-like protozoan tissue cysts were identified in the myocardium of 16 of 42 emaciated birds and in 4 of 23 non-emaciated birds for which histology was available. Tissue cysts were occasionally found in skeletal muscle but in no other tissues. Lead levels were measured in bone and/or liver and kidney in 19 emaciated and 14 non-emaciated birds. One emaciated bird had liver lead levels indicative of lead poisoning (54.64 ppm dry weight) and one emaciated bird had liver (9.78 ppm dw) and kidney (6.62 ppm dw) lead levels indicative of recent exposure. Nine owls had bone lead levels between 1.01 and 2.81 ppm dw indicative of low-level chronic exposure.**

**(49) EXPOSURE OF EMPEROR GEESE TO SELENIUM IN WESTERN ALASKA.**

**J. CHRISTIAN FRANSON and LYNN H. CREEKMORE, U.S. Geological Survey, Biological Resources Division, National Wildlife Health Center, 6006 Schroeder Road, Madison, WI 53711; JOEL A. SCHMUTZ and ADA C. FOWLER, U.S. Geological Survey, Biological Resources Division, Alaska Biological Science Center, 1011 East Tudor Road, Anchorage, AK 99503.**

**We determined selenium concentrations in blood samples collected from 124 emperor geese (*Chen canagica*) on their breeding grounds on the Yukon-Kuskokwim Delta in western Alaska. Concentrations of selenium in the blood of adults ranged up to 10 ppm wet weight, which is nearly as high as the concentrations associated with death in experimental studies with mallards (*Anas platyrhynchos*). Incubating adult female geese captured in late May through mid-June had significantly higher concentrations of selenium in their blood than adult females captured in late July during wing molt. Analysis of sequential samples from two adult geese suggests that the half-time for selenium in the blood of emperor geese is greater than the half-time reported for mallards. The fact that selenium concentrations declined in the blood of adult females from May to July supports a previous hypothesis that exposure of emperor geese to selenium is greater on the wintering and staging areas than on the breeding grounds. High selenium concentrations in the blood of adult female emperor geese point to a need for the analysis of eggs to more thoroughly evaluate the potential effects of selenium on this population.**

**(50) LEAD POISONING AND CONCENTRATIONS OF SELECTED TRACE ELEMENTS IN COMMON EIDERS FROM FINLAND.**

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**The breeding population of common eiders *(Somateria mollissima)* in the Finnish archipelago increased from the 1940s to the mid-1980s. However, concern regarding the health of the eider population arose in the late 1980s, when duckling survival at many breeding areas dropped to as low as 1-5%. During 1994-1996, we evaluated tissues of 63 common eiders from four locations in the Gulf of Finland to identify potential problems related to exposure to selected trace elements. Four of 36 adults (three females and one male) had high liver lead concentrations (> 47 ppm dry weight). Two of these females had acid-fast intranuclear inclusion bodies in their kidneys. Two other adults (one female and one male) had liver lead concentrations of 8 and 14 ppm, which are considered to be elevated for waterfowl. The mean lead concentration in the blood of 11 eider hens was 0.37 ppm wet weight. Lead was found in the liver of one of 16 ducklings tested, at a concentration of 2 ppm dry weight. Arsenic concentrations of up to 39 ppm dry weight were detected in livers of four of 36 adult eiders. Selenium residues of > 60 ppm dry weight were found in livers of five adults and the mean selenium concentration in the blood of hens was 1.98 ppm wet weight. Mercury concetrations in livers were less than 10 ppm dry weight. These results raise concern about exposure of Finnish eiders to lead, selenium, and arsenic.**

**(51) NORTHERN FUR SEAL (*CALLORHINUS URSINUS*) STRANDINGS ALONG THE CENTRAL CALIFORNIA COAST OVER TWENTY-THREE YEARS, 1975-1997.**

**DEBORAH FAUQUIER, School of Veterinary Medicine, University of California at Davis, Davis, CA 95616; FRANCES GULLAND and MARTIN HAULENA, The Marine Mammal Center, Marin Headlands, Sausalito, CA 94965; LINDA LOWENSTINE, Department of Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California at Davis, Davis, CA 95616.**

**Between January 1, 1975 to December 31, 1997, 114 northern fur seals (*Callorhinus ursinus*) stranded live along the central California coast were transported to a rehabilitation center. Of these 114 animals, 80 died during rehabilitation. There were 92 pups (<1 year), 5 juveniles (1-4 years) and 17 adults (>4 years) admitted. The majority of these animals were stranded during the months of September to December (n=95). Most of the animals were stranded in San Luis Obispo, Monterey, Santa Cruz and San Mateo counties (n=90). The average number of animals admitted per year was less than 5. The peak years for strandings were during El Nino events in 1992 (n=22) and 1997 (n=31). The major cause of stranding for the pups was emaciation and malnutrition. Infectious diseases and parasitism were rare. Several pups were anemic on admittance, with decreased hematocrit and hemoglobin values. The average weight for the pups, which were at least 2 months old, was 5.45 kg and the average length was 68.2 cm. Northern fur seal pups are born on average at 60 cm and between 4.5 -5.4 kg.**

**(52) HUMAN INTERACTION RELATED INJURIES OBSERVED IN PINNIPEDS AT A REHABILITATION CENTER IN CENTRAL CALIFORNIA 1986-1996.**

**TRACEY GOLDSTEIN, SHAWN JOHNSON, KRISTA HANNI, DEBORAH FAUQUIER, and FRANCES GULLAND, The Marine Mammal Center, Marin Headlands, GGNRA, Sausalito, CA 94965.**

**From 1986-1996, 348 cases of human interaction related injuries were observed in pinnipeds admitted to a rehabilitation center in central California. Of these animals, 84 had lesions caused by entanglement with marine debris, 25 with fishing tackle, and 3 from boat related trauma. Although cases were observed throughout the year, the month in which the most cases were seen was June. Typical lesions caused by entanglements were seen around the neck and head. The material was usually embedded in granulation tissue and muscle, causing swelling and deep suppurative wounds. An obvious scar developed once the debris was removed and the wound healed. Fish hooks were most often found embedded in the mouths, esophagus, stomachs and flippers of the animals and were most commonly diagnosed with radiographs. In the cases where trauma was suspected due to boat propellers, diagnosis was made based on the presence of multiple deep smooth edged lacerations. Of the animals in this group, 77 were released, 29 died and six were euthanized.**

**Wounds caused by gunshots were the most common, accounting for 236 cases, with the majority occurring in California sea lions (*Zalophus californianus*). A total of 204 gunshot cases was seen from 1992-1996. All were California sea lions except for two Pacific harbor seals (Phoca vitulina), four northern elephant seals (*Mirounga angustirostris*) and one Stellar sea lion (*Eumetopias jubatus*). In 1992, the prevalence was the highest (16%), with the mean for the last five years of the study (1992-1996) being 11% in contrast to 5% for the first five years (1986-1991). Yearling sea lions stranding in the Monterey area were the most affected age class, with June being the peak month for admission of these animals. Clinical presentation of animals suspected of gunshot included paralysis or paresis, signs of central nervous system damage such as blindness or seizures, ocular lesions, lesions typical of bullet entry or exit wounds, deep draining abscesses from puncture wounds, or multiple small dermal wounds from shotgun pellets. The majority of wounds caused by gunshots were found in the head (139), then less frequently in the spine (39), and fewer in the thorax (24), shoulders (17), and abdomen (2). The most effective way of diagnosing these gunshot cases was with radiographs. The common types of ammunition found were shot gun pellets (66), followed by bullets (63)-mostly 0.22 in caliber. Of these 204 gunshot animals, 101 were euthanized, 92 died, 42 were released, and one was placed in a captive facility as it was deemed unreleasable. This data suggests that extensive pinniped-human interactions do occur in central California, and that the predominant interaction is shooting of California sea lions.**

**(53) ISOLATION OF MHC CLASS II BETA GENES AND ITS APPLICATION TO WILDLIFE DISEASE RESEARCH.**

**ANDREW J. PACEJKA, Department of Biology, University of Utah, Salt Lake City, Utah 84112; WAYNE K. POTTS, Department of Biology, University of Utah, Salt Lake City, Utah 84112.**

**Major Histocompatibility Complex (MHC) molecules are expressed on the surface of most vertebrate cells and play an active role in vertebrate disease resistance and activation of immune response by presenting antigenic peptides to T-lymphocytes. The genes that code for the antigen binding site of MHC molecules are some of the most polymorphic genes known. It has been hypothesized that a higher level of polymorphism in these genes enables the MHC to recognize and alert the immune system to a greater array of pathogens and parasites. Thus, MHC gene polymorphism in a population should be sensitive to selective pressure from pathogens encountered by the population. Indeed, the level of polymorphism may be a gauge of the disease history of the population as well as an indicator of the relative vigor of the population. Using primers normally used to isolate passerine MHC, we have isolated what appears to be the MHC Class II Beta genes of Mallards (*Anas platyrhynchos*). By analyzing Mallard MHC polymorphism we hope to use Mallards as a model system for studying wildlife disease resistance.**

**(54) THE HAWAIIAN MONK SEAL (*MONACHUS SCHAUINSLANDI*) EPIDEMIOLOGY PLAN: HEALTH AND DISEASE CONSIDERATIONS IN THE MANAGEMENT OF AN ENDANGERED SPECIES.**

**A. ALONSO AGUIRRE, Joint Institute for Marine and Atmospheric Research, University of Hawaii, 2570 Dole St., Honolulu, Hawaii, 96822.**

**The Hawaiian monk seal (*Monachus schauinslandi*) is considered the most endangered pinniped in North America. As a result of a severe population decline, the species has been protected since 1972. In 1984 a rehabilitation program was initiated consisting of taking undersized, weaned female pups and holding them in captivity on Oahu. Rehabilitated seal pups were then released into the Northwestern Hawaiian Islands after 8-10 months. An eye disease of unknown etiology has been documented in 11 of 12 female seal pups brought into captivity for rehabilitation during 1995. The seals have not been released because of the risk of spreading the disease to the wild population. It is difficult to evaluate that risk and compare it to the benefits of translocation when our understanding of disease in this species is limited. Studies were initiated in the fall of 1996 as part of an epidemiology plan to characterize the role of health and disease in monk seal population dynamics. This plan identifies surveillance, rehabilitation and translocation efforts as part of a comprehensive program to enhance the recovery of the monk seal population. The objectives of this project include evaluation of clinically healthy 'normal' values for wild monk seals; collection and interpretation of biomedical and pathologic data; assessment of prevalence, incidence and geographic distribution of potential pathogens; implementation of a die-off response plan; development of diagnostic, preventive and treatment methods as required; and development of a computer data base and biological specimen (serum and tissue banks) collection.**

**(55) EXPOSURE TO FISHING TACKLE IN SELECTED AVIAN SPECIES.**

**SCOTT P. HANSEN, J. CHRISTIAN FRANSON, and TERRY E. CREEKMORE. U.S. Geological Survey, Biological Resources Division, National Wildlife Health Center, 6006 Schroeder Road, Madison, WI 53711.**

**From March 1996 through early 1998, we studied the prevalence of exposure to fishing sinkers and other tackle in swans and 31 species of fish-eating birds, including common loons. We recorded visual observations of sinkers and other tackle from x-rays of live birds, and recovered sinkers and tackle from necropsy specimens. Radiographs and necropsy findings were gathered with the assistance of rehabilitators and natural resource managers. We collected data from 31 locations in the United States; 42% of the specimens were from Florida. Of 1,272 birds examined, radiographic evidence or direct observation of one or more pieces of fishing tackle was noted in 139 birds of seven species. Common loons and brown pelicans which had been brought into rehabilitation centers accounted for 85% of the specimens in which sinkers and tackle were found. Ingested fishing sinkers were found in four of 142 common loons and seven of 260 brown pelicans. Fishing tackle, other than sinkers, including hooks, line, and other materials, was also found in 14 common loons, 99 brown pelicans, eight double-crested cormorants, four Brandt's cormorants, one bald eagle, one great blue heron, and one Pacific loon.**

**(56) SELECTED BLOOD PARAMETERS COLLECTED FROM WILD MANATEES IN FLORIDA.**

**SCOTT D. WRIGHT and J. MARK SWEAT, Marine Mammal Pathobiology Laboratory, Florida Marine Research Institute, Department of Environmental Protection, St. Petersburg, Florida; CATHY PERRY, Sea World of Florida, Orlando, Florida.**

**Whenever possible, blood samples were collected from wild manatees captured for research purposes since 1991. Whole blood was collected, stored on wet ice and then centrifugated, usually within eight hours of collection. Laboratory processing of blood was conducted at the clinical laboratory of Sea World in Orlando. Hematological and serum chemistry values were measured from many samples. There are several subsets of data. The first set of values (identified as: West Coast, Red Tide Matlacha, Red Tide Marco) represent blood samples collected one time. The other subset (South West Study) represents the pooled blood samples collected from several captures of the same animals over several months. Also, the results of serial bleeding from two manatees captured during the SW Study are presented. For comparison, similar published blood parameters collected from captive and a few wild manatees are included. Blood values reported in this work are represented by the mean, range, and standard deviation of each paramenter.**

**Overall, the values did not differ appreciably between groups or among individual samples collected from the same manatee. When compared to published data, there were some differences in specific parameters. When evaluated against the whole panel of parameters for an individual, these differences were not biologically important. Nevertheless, this information is important as it represents baseline data previously unavailable and provides insight into "normal" values for wild manatees. This type of information does have limitations and does not alone represent disease processes.**

**An advantage of this type of data is that, considered with body weight, blubber measurements, telemetry movement data, and behavioral observations, there can be clearer assessment of the ability of the animal to survive in the wild. This is especially important for rehabilitated manatees. An important disadvantage is that unlike all the other data types mentioned above, blood values are not available at the capture site and therefore do not contribute to the immediate decision about the condition of the animal.**

**(57) OVERVIEW OF ISSUES SURROUNDING AMPHIBIAN DECLINE AND THE ROLE OF THE DECLINING AMPHIBIAN POPULATIONS TASK FORCE.**

**GARY S. CASPER, Vertebrate Zoology Section, Milwaukee Public Museum, 800 W. Wells St., Milwaukee, WI 53233.**

**Over the last 50 years, many species of amphibians throughout the world have declined markedly in numbers. Some species have become extinct. In many cases the declines are a direct response to the impact of human activities (such as habitat destruction or pollution) acting at a local level. Towards the late 1980s, biologists from many parts of the world reported declines in amphibian populations in apparently pristine habitats, such as national parks and nature reserves, where local effects could not be implicated. This led to the suggestion that there may be one or more global factors that are adversely affecting amphibians. Rates of deformities in amphibians are also increasing. Possible candidates for the causes of these problems are:**

**An increase in ionizing radiation (UV-B) resulting from ozone layer depletion.**
**Chemical contamination (including pesticides, herbicides, acid precipitation, fertilizers).**
**Introduction of exotic competitors and predators.**
**Pathogens.**
**Habitat losses and degradation.**

**The Declining Amphibian Populations Task Force (DAPTF) was established in 1991. The DAPTF consists of a network of over 3,000 scientists and conservationists belonging to national and regional working groups which now cover more than 90 countries around the world. The mission of the DAPTF is to determine the nature, extent and causes of declines of amphibians throughout the world, and to promote means by which declines can be halted or reversed. In addition to research into amphibian decline and deformities, a need to establish a monitoring program to adequately track species trends and status has been identified. Two options currently lie before policy makers on the issue of amphibian declines - wait and see if the problem goes away or act now. The ecological return on the first approach will be ignorance and further extinctions, while the second could well prevent biodiversity losses and head off serious repercussions for human as well as amphibian health.**

**(58) GLOBAL AMPHIBIAN POPULATION DECLINES & THE ROLE OF INFECTIOUS DISEASES.**

**D. EARL GREEN, National Institutes of Health, Building 28A, 28 Library Drive, Bethesda, Maryland 20892-5230.**

**The most troubling aspect of amphibian population declines, and multiple probable extinctions, is that many have occurred in the last 10-12 years in remote, pristine and protected sites, such as national parks and biosphere reserves. Documented amphibian declines which spread from site to site, like an epizootic, have been documented in Southwestern USA, Lower Central America, and Queensland. Where amphibian populations have been monitored, these declines are usually preceded by massive die-offs, and the decline is completed in 1-3 years. Most declines involve riparian amphibians. Nearly simultaneous discoveries in Queensland and western Panama that dying frogs were infected by a novel fungal infection (chytridiomycosis) is strong evidence that sudden amphibian population declines are epizootics. Multiple recent reports of spontaneous and experimental lethal infections by iridoviruses in fish and amphibians suggests that multiple or concurrent epizootics may be involved in amphibian population declines. The role of viruses in disease, mortality, and population declines in amphibians will be reviewed; recently detected amphibian viral diseases will be presented. Recent discoveries from amphibian mass casualty sites will emphasize the need for a rapid response to reports of mass casualties and the need for comprehensive diagnostic examinations of all 3 amphibian lifestages (eggs, larvae, adults).**

## (59) ARE CONTAMINANTS IMPACTING AMPHIBIAN POPULATIONS?

**ROBIN E. JUNG, USGS, Patuxent Wildlife Research Center, 12100 Beech Forest Rd., Laurel, MD 20708.**

**During the past decade, increasing reports of amphibian declines and deformities have instigated researchers to investigate potential underlying causes for these problems. Exposure of amphibians to toxicants has been hypothesized to be related to amphibian declines and deformities. While laboratory studies have shown that a variety of contaminants can cause amphibian deformities, mortality, and increased susceptibility to diseases, few field studies have provided direct cause-effect relationships between contaminants and amphibian population effects. I will provide an overview of the information currently available on this topic.**

**(60) FROG MALFORMATIONS: MORE QUESTIONS THAN ANSWERS.**

**CAROL U. METEYER, Biological Resources Division-U.S. Geological Survey, National Wildlife Health Center, 6006 Schroeder Road, Madison, WI 53711.**

**Frog malformations have been reported in 38 states and 3 Canadian provinces. The wide geographic distribution of malformations, the difficulty in determining environmental characteristics unique to the habitat of malformed frogs and the variation in malformation types seen in the frogs confound the search for an underlying cause. Malformations in frogs are the result of errors in tadpole development. Tadpoles progress through their embryonic and larval stages with little protection from the environment. Chemical agents, predation, UV-B radiation, host factors and infectious agents have all been postulated as causes of malformed frogs.**

**A general review of teratogenic mechanisms can provide insight into the processes that result in malformations. Extrapolation of previous work done in other species, primarily mammals, can also provide information as to classes of agents that are known to produce similar malformations. But conclusions cannot be drawn from circumstantial evidence or extrapolation. Efforts to identify factors that occur in the wetland environment and that consistently reproduce characteristic malformations under both laboratory and field conditions need to continue.**

**The impact that malformations have on the physical, behavioral and physiological condition of affected tadpoles and frog has not been assessed and the impact malformations are having at the population level has not been determined.**

**(61) EPIDEMIC EPIDERMAL CHYTRIDIOMYCOSIS IS A CAUSE OF AMPHIBIAN POPULATION DECLINES IN CENTRAL AMERICA.**

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**Unexplained sudden amphibian population declines have occurred for at least 12 years in Costa Rica and Panama. Examinations of sick and dead-in-the-field frogs from remote protected sites in Costa Rica (1993) and Panama (1996-7) revealed the presence of a novel fungal pathogen not previously known to infect vertebrates. Based on histologic, ultrastructural and DNA sequencing, the fungus was identified as belonging to the phylum, Chytridiomycota. These fungi are principally saprobes on plant detritus, chitin and keratin. Histologically, the fungal infection is restricted to keratinized epidermis of tadpoles and post-metamorphic amphibians. There is an affinity for the pelvic patch and tarso-phalangeal skin. Histologically, the organism is identified as irregularly spherical, single, intracellular, non-hyphal sporangia (thalli) from 5-20 microns in diameter. Various stages of development occur in the cells of the stratum corneum, and mature sporangia produce zoospores (1-2 microns) which are released through a characteristic discharge pore. Ultrastructurally, the zoospores have a single flagellum with a non-functional centriole at an angle to the kinetosome, membrane-bound clustered ribosomes, 2-3 mitochondria with discoidal cristae, and an extensive microbody-lipid complex. These features suggest this chytrid is a new genus. Chytridiomycosis of tadpoles is a restricted infection which probably is clinically silent.**

**The mechanism of lethality for such a superficial infection is unknown but several theories will be presented. Examinations of preserved amphibians in museum collections from these two sites support the conclusion that chytridiomycosis is a recently introduced pathogen in a naive and highly susceptible amphibian assemblage.**

**(62) A DIE-OFF OF TIGER SALAMANDERS, *AMBYSTOMA TIGRINUM*, CAUSED BY A NEW IRIDOVIRUS.**

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**Numerous dead adult tiger salamanders were observed during late May and early June, 1997, in 2 farm dugouts near Regina, Saskatchewan. Necropsy examination of 2 salamander carcasses revealed mild generalized edema and congestion with scattered etechial hemorrhages. *Aeromonas* sp., *Aeromonas hydrophila* and *Enterobacter* sp. were isolated from lung, liver, kidney, spleen and intestine. Histological lesions consisted of acute hepatic necrosis, multifocal skin ulcers and multifocal necrosis of gastric and intestinal mucosa. Cells containing large basophilic intracytoplasmic inclusions and marginated chromatin were associated with areas of necrosis. Transmission electron microscopy identified the cytoplasmic inclusions as virus particles resembling iridoviruses. Over 50 additional tiger salamanders affected with the disease were examined, some of which originated from a laboratory colony which became infected after tiger salamanders from disease sites in the wild were introduced. Detailed histopathology of 20 of these tiger salamanders revealed viral inclusions and necrosis may occur in all organs except nervous system and skeletal muscle but was most common and severe in the epidermis, liver and gastrointestinal mucosa. Epithelioma papulosum cyprini (EPC) cells inoculated with frozen tissue from diseased salamanders showed cytopathic effects after 9 days when grown at room temperature (approx. 20$^{o}$ C). Intraperitoneal inoculation of 2 tiger salamanders with a 0.5 ml solution containing 5 x $10^{6}$ $TCID_{50}$ of SSV1 resulted in death of both salamanders 13 days post-inoculation. Virus was isolated from both salamanders and lesions were identical to those observed in salamanders from the initial outbreak. Analysis of the amino acid sequences of the major capsid protein, the SDS-PAGE profile of labeled viral proteins in infected FHM cells and the restriction enzyme profile of HindIII and XbaI cut radiolabeled viral DNA confirmed SSV1 as an iridovirus and that it is closely related, but distinct from frog virus 3 (FV3). Iridoviruses are increasingly being recognized as an important cause of disease in ectothermic vertebrates.**

**(63) HELMINTH COMMUNITIES IN SIX SPECIES OF AMPHIBIA FROM SOUTHEASTERN WISCONSIN.**

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**A total of 317 representatives of six amphibian species including: eastern american toads, blue-spotted salamanders, tiger salamanders, wood frogs, spring peepers, and central newts were collected from three ephemeral ponds at, or near, the University of Wisconsin-Milwaukee Field Station during the 1994 breeding migration and inspected for helminth parasites. Parasite infracommunities were isolationist and depauperate. The component communities of toads, wood frogs, and spring peepers consisted of seven, seven, and nine helminth species, respectively. Component parasite communities of blue-spotted salamanders, tiger salamanders, and newts consisted of two, two, and three species, respectively. Feeding habits (range of prey species and foraging behavior), vagility and habitat preference were identified as aspects of host ecology important in helminth community structure. Host gender did not appear to be important in helminth community structure. In terms of parasite life histories, several nematodes were identified as core species, with skin penetrators being common but considered dominant only in toad infracommunities. Larval and adult platyhelminths were considered satellite species. Relationships between size and infection varied considerably at the level of individual host and parasite species. However, clear, positive relationships were evident between size and measures of parasitism when comparisons were made among host species.**

**(64) A SEASONAL AND COMPARATIVE STUDY OF HELMINTH PARASITES IN WISCONSIN AMPHIBIANS.**

**MATTHEW G. BOLEK, Department of Biological Sciences, University of Wisconsin-Milwaukee, Milwaukee, Wisconsin 53201.**

**Three hundred fifty amphibians of 9 species including green frogs, northern leopard frogs, American toads, Cope's gray treefrogs, western chorus frogs, spring peepers, blue-spotted salamanders, spotted salamanders, and red-backed salamanders were collected during 1996 and 1997, from 4 ponds in Waukesha and Bayfield Counties, Wisconsin and examined for helminth parasites. Two of these species, the green frog and American toad were surveyed seasonally. In general adult trematode prevalence and intensity was highest in late summer-early fall while overall nematode prevalence and abundance was highest in midsummer. Of the 9 amphibian species examined, a total of 4,105 nematodes, approximately 3,352 larval trematodes, approximately 3,323 larval cestodes, 784 adult trematodes, 13 monogeneans, 3 nematomorphs, and 1 adult cestode were recovered. Of these, nematode abundance was highest in toads while larval trematode and cestode abundance was highest in green frogs, Cope's gray treefrogs and leopard frogs. Salamanders harbored fewer helminth species than anurans, with nematodes and larval trematodes being most common. Stomach content analysis revealed that toads were search forager specialists which ate many small prey items primarily ants and mites while Cope's gray treefrogs, leopard frogs, and green frogs were considered sit and wait predators with a broader diet of invertebrates. Accordingly, parasites with direct life cycles were common in toads, making up 68%-94% of the helminth abundance while helminths which utilize intermediate hosts were most common in green frogs, leopard frogs and Cope's gray treefrogs, making up 65%-90% of the helminth abundance. Although site characteristics played a role in producing variation in type and prevalence of helminths observed, diet and feeding patterns are important in determining helminth parasites in amphibian hosts.**

**(65) DEVELOPMENT AND DISTRIBUTION OF IMMUNOGLOBULIN-CONTAINING CELLS IN THE AMERICAN LEOPARD FROG, *RANA PIPIENS*: EMBRYOGENESIS THROUGH ADULTHOOD.**

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**Recently, investigators have suggested that: (i) embryonic hematopoietic stem cells (HSCs) develop from precursor cells which are formed in the ventral marginal zone of the early gastrula, and that (ii) during neurulation two HSC compartments -- one dorsal - one ventral -- are derived from the original marginal zone cells. HSCs in the dorsal compartment, which includes the aorta, gonads and nephros (AGN), are sites for enrichment of developing immunoglobulin-producing (Ig+) B lineage cells. In Rana, cells in various B lineages appear just prior to the completion of embryogenesis. By the time tadpoles begin to eat: (i) pre-B cells, which are defined by a lack of detectable amounts of surface IgM, yet contain detectable amounts of cytoplasmic IgM (sIgM-cIgM+), and (ii) sIgM+ B cells are present in the AGN, liver and circulating blood. Later, B lineage cells are observed in thymus and lymph glands. Plasma cells, which contain abundant quantities of cIgM, are observed first in tadpole nephric tissues by Taylor and Kollros (T&K) stage II, while plasma cells containing only low molecular weight Ig are observed in nephric tissues at T&K stage V. The proportion of pre-B to B cells decreases with the approach of metamorphosis, while B and plasma cell numbers approach peak levels. Coincident and consistent with the vast systemic changes associated with metamorphosis, the cells and tissues of the tadpole immune system are also radically changed -- even deleted. After metamorphosis, newly emergent lymphoid organs of froglets are formed and populated with various blood cell lineages. By adulthood, cells in B lineages are present in male and female lymphoid tissues, which include jugular bodies, bone marrow, intestine, kidneys, spleen, liver and circulating blood.** **(Supported, in part, by an award to LDZ from NIEHS and by NIEHS Biomedical Research Laboratories, Great Lakes WATER Institute).**

**(66) FIBROPAPILLOMATOSIS IN OLIVE RIDLEY TURTLES (*LEPIDOCHELYS OLIVACEA*).**

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**Fibropapillomatosis (FP) is a neoplastic disease that primarily affects green turtles (*Chelonia mydas*) in epidemic proportions worldwide. Although several infectious agents (herpesvirus, retrovirus and papillomavirus) have been associated with the condition, the etiologic agent has not been isolated or characterized. FP has been recently reported in other sea turtle species including confirmed cases in loggerhead turtles (*Caretta caretta*) in Florida and field observations in olive ridley turtles (*Lepidochelys olivacea*) in the Pacific coasts of Mexico and Costa Rica. Normal skin (6) and tumor (41) biopsies were collected from 25 adult female olive ridley turtles in Ostional, Costa Rica, between July and September 1997. Grossly, biopsies were small, white to grey, smooth to verruciform, raised masses on the integument of the neck and flippers. All 41 masses were 25 mm or less in diameter and histologically, 8/41 masses were small foci of chronic active dermatitis and not tumors; and 33/41 were diagnosed as fibropapillomas. Twelve of 33 tumors were regressing and 9 of the remaining 21 tumors had early histological changes that suggested degeneration within the tumor. During field surveys based on gross lesions, prevalences of 1-10% have been reported in this nesting population. This is considered the first diagnostic confirmation of FP in olive ridley turtles.**

## (67) FIBROPAPILLOMATOSIS OF GREEN TURTLES IN HAWAII...WHAT'S NEW?

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**Since 1995, the Honolulu Field Station (HFS) has worked closely with the National Marine Fisheries Service to systematically evaluate causes of morbidity and mortality in stranded marine turtles in Hawaii. To date, >80 turtles have been examined using gross and microscopic pathology. The most prevalent disease is fibropapillomatosis (FP) and vascular fluke infections. Internal fibromas, fibrosarcomas of low grade malignancy and myxofibromas were seen in ~20% of animals examined with the lungs, kidney and heart most commonly affected. White blood cell morphology of green turtles was recently characterized thus allowing comparisons of healthy versus diseased turtles. Turtles afflicted with FP exhibited hypoproteinemia, anemia, lymphopenia, heterophilia, eosinopenia and monocytosis. A retrovirus was found in turtles afflicted with FP and a tumor-associated herpesvirus was recently discovered. The latter was found only in skin and internal tumors but not in unaffected skin, internal organs or blood. Cell lines from green turtle embryos were established and are being used to try and isolate potential viruses associated with FP.**

**(68) IMPACTS OF PCB EXPOSURE ON SNAPPING TURTLE REPRODUCTION, HATCHLING DEVELOPMENT, AND BEHAVIOR.**

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**Snapping turtles (*Chelydra serpentina*) from contaminated rivers accumulate high body burdens of PCBs which are transferred to the eggs. Our objective was to incubate snapping turtle egg clutches from PCB-contaminated and reference sites under controlled conditions to determine if reproduction is impacted. Clutches were collected from 10 females from contaminated sites and 4 from reference sites. Two to 8 egg composites from each clutch were analyzed for 69 PCB congeners. Half of each clutch was incubated at male-inducing and the other half at female-inducing temperatures. Congener sums ranged from 0.07 to 27.98 ppm with variable composition between contaminated sites. Hatching success was reduced in clutches with PCBs greater than 15 ppm incubated at male-inducing, but not female-inducing temperatures. Gross deformities were observed in 2 turtles and bent tails were observed in 9 out of 353 hatchlings. Hatching weight was positively correlated with adult female weight, but not PCB concentration. Hatchling growth and motor skills were monitored for 33 weeks. In 1996, growth curves diverged at 16-24 weeks resulting in significantly lower final weights for reference hatchlings. Righting response was significantly slower in turtles from contaminated sites in 1996. Growth and righting response for 1997 turtles will be analyzed and presented. Ninety-four turtles were necropsied at 15 days or 8 months to determine the sex of gonads morphologically and histologically, analyze blood samples for circulating hormone levels, and test liver enzyme activity for the effects of PCBs. Hepatic ethoxyresorufin-O-deethylase was significantly depressed in 8 month turtles. Analysis of a partial dataset indicates that progesterone/testosterone ratio may be inversely related to PCB exposure. Remaining turtles were marked with microchips and released where the female was captured. We intend to resample released turtles through periodic trapping to monitor PCB accumulation, growth, reproductive maturation, and survival to determine if PCBs are having a long-term impact on snapping turtle populations.**

**(69) THE EFFECT OF NEWCASTLE DISEASE AND OTHER CAUSES OF MORTALITY ON THE REPRODUCTIVE SUCCESS OF DOUBLE-CRESTED CORMORANTS.**

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**Newcastle disease may cause high mortality in juvenile double-crested cormorants, but it is not known how important this is compared to other causes. To determine cause-specific mortality rates of juveniles in a breeding colony of double-crested cormorants on Doré Lake (Saskatchewan, Canada), morbidity and mortality were monitored every third day during the breeding season from 1994 to 1996 from inside an above-ground tunnel-and-blind system. When possible, affected birds (n = 1571) were collected for examination and diagnosis. The main causes of mortality were avian predation, displacement from the nest, starvation from sibling competition, Newcastle disease, and coyote predation. In 47% of the cases, avian predation and displacement from the nest were associated with human disturbance. Other diseases included beak malformation, abnormal rotation of the carpal joint, hypopigmentation, and eye loss. Overall mortality of juvenile cormorants between hatching and the end of the breeding season varied from 25 to 48%. The most important causes of mortality were Newcastle disease, which killed 21% of hatched chicks in 1995, sibling competition (maximum 12% in 1994), and coyote predation (2% in 1994).**

**(70) NEWCASTLE DISEASE VIRUS IN DOUBLE CRESTED CORMORANTS; CULTURE CHARACTERISTICS AND SEROLOGY.**

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**Morbidity and mortality, associated with Newcastle Disease Virus (NDV), occurred in 1992 and 1997 among double crested cormorants (DCC) in the U.S. The 1992 event occurred on islands in Lakes Michigan, Superior, Huron and Ontario, and at locations in Minnesota, Nebraska, and South Dakota. The 1997 event occurred at the Salton Sea in California and at locations in Oregon and Utah. The 1997 DCC NDV isolates were similar in culture characteristics and presented a diagnostic challenge on initial isolation unlike the 1992 isolates. The culture characteristics of these isolates and the methods used for identification will be discussed. These methods include attempts to increase the sensitivity of the test system by using indicator red blood cells from different avian species and to slow the apparent rapid elution rate of the 1997 isolate. The results of serology tests, to determine antigenic relationships between a reference (LaSota) lentogenic NDV and the DCC NDV isolates from 1992 and 1997, will also be discussed.**

**(71) *MYCOPLASMA GALLISEPTICUM* IN HOUSE FINCHES AND OTHER PASSERINE SPECIES IN GEORGIA 1997-1998.**

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**The apparent adaptation of *Mycoplasma gallisepticum* (MG) to free-living avian species presents many potential problems for control of this disease in both domestic poultry and wildlife populations. The objectives of this study were: 1) to determine the prevalence of MG infection in house finches and other passerine species commonly associated with backyard feeders and poultry production facilities; and 2) to evaluate existing MG diagnostic techniques for use in passerine species other than house finches. To date, 679 birds representing three avian orders and 13 families have been sampled. Of these, positive serum plate agglutination (SPA) test results were detected in eight species including house finches. Antibody prevalence in house finches was extremely low (1.8%) but infection was confirmed by MG culture from two birds. Most SPA positive species were included in two avian families, the Paridae (tufted titmouse and carolina chickadee) and the Muscicapidae (hermit thrush, eastern bluebird, and American robin). Although some of these birds also tested seropositive by hemagglutination inhibition (HI), none were culture positive. Results indicate that MG was present in house finches in Georgia two years after it was first documented in house finches in this state. The low prevalence of infection observed in this study, however, suggests that MG may be difficult to detect. Infection in other species has not been confirmed by culture or PCR, suggesting that future surveillance directed at these species may be complicated by non-specific serologic reactions.**

**(72) MOLECULAR EPIDEMIOLOGY OF SONGBIRD CONJUNCTIVITIS ASSOCIATED WITH *MYCOPLASMA GALLISEPTICUM* AND *M. STURNI.***

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**Reports of house finches with conjunctivitis were first made in February 1994, and since that time affected birds have been observed throughout its eastern range. Numerous isolates of *Mycoplasma gallisepticum* have been made from clinically affected birds, and its etiological role confirmed.**

**In 1996 *M. gallisepticum* was isolated from American goldfinches with conjunctivitis. Random amplification of polymorphic DNA (RAPD), a polymerase chain reaction-based method of DNA fingerprinting, was used to examine the molecular epidemiological relationships of *M. gallisepticum* isolates. All of the isolates from songbirds tested had essentially identical RAPD patterns, indicating that this epidemic is caused by a single strain of *M. gallisepticum,* probably first involving house finches and more recently goldfinches. Additionally, the finch *M. gallisepticum* RAPD pattern differed from patterns of reference strains, vaccine strains, and isolates from commercial poultry. Therefore, the *M. gallisepticum* epidemic in songbirds is not caused by vaccine strains, and has not been linked to the reference strains or poultry isolates tested. In 1996-97, a new mycoplasmal species, *M. sturni,* was isolated in Connecticut from a European starling with bilateral conjunctivitis. Archived isolates from blue jays and northern mockingbirds with conjunctivitis in Florida have also been identified as *M. sturni*. Therefore, *M. sturni* as well as *M. gallisepticum* should be considered in the differential diagnosis of songbirds with conjunctivitis.**

## (73) RESERVOIRS FOR AVIAN CHOLERA: WETLANDS OR WATERFOWL?

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**Avian cholera is an infectious bacterial disease that causes the death of thousands of water birds throughout North America each year. Since the initial reports of this disease in the 1940s, the distribution of avian cholera epizootics have spread throughout the country. Despite the importance of this disease for water bird populations, little is known about the reservoirs for the bacteria (*Pasteurella multocida* serotype 1) or how this disease is spread. Because snow geese have been linked to previous avian cholera epizootics, we collected swab samples and conduced serological surveys on breeding colonies in the western arctic to look for potential disease carriers. In the fall, we attempted to recover *P. multocida* bacteria from wetlands where avian cholera epizootics had occurred the previous winter or spring. We recovered a pathogenic *P. multocida* culture from a healthy snow goose and the prevalence of antibodies to *P. multocida* increased following avian cholera epizootics on snow goose nesting colonies, indicating that healthy geese had been exposed to the disease. In contrast, we have been unsuccessful in recovering *P. multocida* from wetlands 6-9 months following avian cholera mortality. Snow geese and other waterfowl species may serve as an important reservoir in the epizootiology and spread of this disease.**

**(74) HABITAT MANAGEMENT AND VECTOR CONTROL: PROSPECTS FOR MANAGING AVIAN DISEASE IN HAWAIIAN FOREST BIRDS.**

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**Wet and mesic forests on the southwestern slopes of the island of Hawaii are home to remnant populations of four species of endangered forest birds, including Alala (*Corvus hawaiiensis*), Akiapola'au (*Hemignathus munroi*), Hawaii Akepa (*Loxops coccineus*), and Hawaii Creeper (*Oreomystis mana*). While a number of things threaten these endemic species, high susceptibility to avian pox and malaria (*Plasmodium relictum*) is believed to be the primary factor preventing recovery of populations at elevations below 5,000 ft. We conducted a detailed study of the seasonal fluctuations, distribution, breeding sites, and infection rates of the primary vector of these diseases, *Culex quinquefasciatus*, along elevational transects through critical forest bird habitat. This information was used to test the effectiveness of source reduction in reducing *Culex* populations. We found that primary oviposition sites were concentrated between elevations of 3,000 and 5,000 ft. in hollowed tree ferns that had been damaged by feral ungulates. Approximately 12,000 of these sites were manually drained within an 800 hectare plot over a four month period. In spite of this effort, up to 30% of the oviposition sites were missed because of thick vegetation and difficult terrain. Reductions in adult Culex populations were not detected before and after treatment or when comparisons were made between treatment and control plots. While initial results were discouraging, source reduction is potentially feasible if it is coupled with fencing and feral ungulate control and practiced over a wide enough area.**

## (75) IMMUNIZATION OF DUCKS FOR TYPE C BOTULISM.

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**Sick birds treated during botulism outbreaks do not develop resistance to the toxin and are likely to become reintoxicated if they return to the outbreak site. We investigated the potential of immunizing ducks as part of treatment. A single subcutaneous immunization with a commercial vaccine used for ranch mink protected mallard and northern pintail ducks against challenge with $4.5 \times 10^4$ and $2.25 \times 10^4$ mouse lethal doses ($MLD_{50}$), respectively, of type C botulinum toxin at 10 and 15 days post-immunization (pi). There was no protection at 5 days pi. Protection persisted for at least 90 days pi. To simulate use of vaccine in treating sick birds, mallards were exposed to toxin and when clinical signs were evident, all birds were treated by giving antitoxin by intraperitoneal injection and half of the birds were immunized. Immunization did not effect recovery from intoxication. At 10 days post-treatment, all birds were re-challenged with toxin. Clinical signs and mortality were less frequent among the immunized birds than among the non-immunized birds. Immunization would not contribute materially to the cost of treatment and may be a useful part of the treatment regimen in botulism outbreaks.**

**(76) MARINE BIRDS AS POTENTIAL MONITORS OF MARINE ECOSYSTEM HEALTH.**

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**Marine ecosystem health is a relatively new and ill defined concept, but the extent to which mortality events involving different species at various trophic levels may serve as a measure of health, is now being explored. In 1997-98 we experienced three marine bird dieoffs that have implications for the health of the marine environment. 1) In August of 1997, approximately 400 common murres (*Uria aalge*) were found dead in a relatively confined area of the southern end of Monterey Bay. Serosanguinous rhinitis and severe pulmonary edema were the most prominent gross lesions and imunohistochemical staining for brevetoxin was positive. This is the first report of inhaled bevetoxin killing birds on the Pacific coast, but in retrospect, brevetoxicosis is suspected in several recent common murre dieoffs in California, each involving hundreds of murres and each occurring during months with warmer ocean temperatures. 2) Over an approximately one week period in late October of 1997 five hundred marine birds, predominately western grebes (*Aechmophorus occidentalis*), common loons (*Gavia imer*), and surf scoters (*Melanitta perspicillata*) became fouled with a fish/vegetable oil. This oil caused water saturation, hypothermia and associated debilitation, but many birds also suffered enteritis and septicemia due to salmonellosis. The combination of physical fouling and acute stress due to oil, bacteremia, and migration related debilitation resulted in relatively high mortality. 3) Over a three month period in the winter of 1997-98 a significant percentage of the common murre population off California's central coast died as the result of oil and tar contamination. Over 600 live birds (94% of which were murres) and over 1200 dead birds were recovered. Although no point source for the petroleum was found, it appears to be a refined product and several lines of investigation are being followed. Events of this type have occurred repeatedly in California over the last 5 - 10 years and may have serious population level effects.**

**Each of these dieoffs occurred in a National Marine Sanctuary where baseline mortality information is collected regularly, thus comparison with previous years and inference as to whether such events are "normal" can be made. These events and processes may be of various durations, cyclic, or periodically repeated, but to the extent they result in significant mortality events and reflect human impacts, may be an indicator of marine ecosystem health.**

## (77) CAUSES OF MORBIDITY AND MORTALITY IN BALD EAGLES FROM FLORIDA.

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**The most common cause-of-death for Bald Eagles (*Haliaeetus leucocephalus*) in Florida is trauma due to collisions with moving motor vehicles, gunshot, intraspecific aggression, flying into power lines, wires, etc. These factors accounted for 59% of the deaths of 309 Bald Eagles from Florida examined over the 30-year period from 1963 to 1994. Other significant factors were electrocution (16%), poisoning (10%), infectious diseases (7%), and emaciation (of unknown causes) (5%). Inclement weather (especially hurricanes) is responsible for some mortality of eggs and young. One hundred and ten disease agents and parasites have been identified in Bald Eagles from Florida. These include organochlorines (14), organophosphates (1), carbamates (1), barbiturates (1), metals (10), viruses (2), bacteria (28), fungi (6), protozoans (9), trematodes (8), cestodes (1), acanthocephalans (3), nematodes (11), ticks (2), mites (3), dipterans (4), and chewing lice (6). Several of these disease agents are known to have caused direct mortality and include dieldrin, cumulative organochlorines, pentobarbitol, lead, poxvirus, *Aspergillus fumigatus*, and an undescribed species of *Hamatospiculum.* In addition DDT and its metabolites (especially DDE) have caused reproductive impairment due to eggshell thinning. There is evidence that the latter problem has diminished since the banning of DDT sales in the U.S. and currently Bald Eagle populations in Florida are increasing.**

## (78) SCHISTOSOMIASIS IN A COLLECTION OF CAPTIVE CHILEAN FLAMINGOS.

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**Schistosome ova were detected in the histological sections of several captive Chilean flamingos (*Phoenicopterus chilensis*) that died at the Calgary Zoo from 1989 to 1998. In only one case could the cause of death be partly attributed to the presence of trematode ova. Ova were most consistently found in the pancreas, ventricular muscle, and proventriculus; other sites included the cerebellum, liver, kidney, spleen, lungs and skeletal muscle. Within hepatic parenchyma, there was moderate inflammation associated with the presence of ova; other sites did not exhibit inflammatory response. Adult schistosomes were not found in any sections, nor in blood vessels at gross necropsy, and the genus of the trematode remains undetermined. Flamingos are most likely aberrant hosts; definitive hosts may include wild Mallard ducks (*Anas platyrhynchos*), domestic ducks (*Anas platyrhynchos*), wild Canada Geese (*Branta canadensis*), or Red-eared Sliders (*Trachemys scripta elegans*) which all shared the summer pond with the flamingos. Work is underway to locate and identify snails which may be acting as intermediate hosts.**

**(79) PARASITIC MITES OF NORTH AMERICAN OWLS.**

**JAMES R. PHILIPS, Math/Science Division, Babson College, Babson Park, Massachusetts, 02157.**

**Parasitic mites of owls include those which feed on blood, feather oils, feather tissue, skin, and tissue fluid. Host relationships range from monoxenous to polyxenous, and mite geographic distribution ranges from endemic to cosmopolitan. Of 41 species of North American owls, mites are known from 18 species, but records from the Holarctic species are mainly from the Palaearctic region. The mite fauna of *Asio otus* is most well known, 16 species, but only 2 species are known from North American longeared owls. The North American mite fauna of *Bubo virginianus* is the best known, 11 species, followed by that of *Speotyto cunicularia* with 9 species, *Strix varia* with 5 species, and *Otus asio* with 4 species. Three or fewer mite species are known from other owls in North America. Data on occurrence on different regions of the host's body, mite populations on healthy and diseased owls, and pathology are largely lacking.**

**(80) SALMONELLOSIS IN WILD AND CAPTIVE BIRDS: PATHOLOGY AND CHARACTERIZATION OF ISOLATES.**

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**A recent large scale epizootic of salmonellosis in songbirds in the northeastern and midwestern United States has raised concerns regarding the strains of *Salmonella* that cause disease in passerine species as little is known of non-domestic bird isolates. Here, 12 *Salmonella* group B isolates obtained from nine wild bird species and three captive non-domestic species were characterized based on lesions, antibiotic resistance patterns, invasiveness, and virulence. Birds were submitted from five southeastern states. Five of the cases in wild birds were epizootics that resulted in morbidity and mortality of numerous birds. The four additional cases involving wild birds were individual animal submissions. Species examined included brown-headed cowbirds, goldfinches, laughing gulls, wild turkey, coturnix quail, and an unidentified owl. Captive bird isolates came from a parakeet, an African grey parrot, and an emu. Gross lesions were seen >50% of the birds; histologic lesions were seen in all cases. All *Salmonella* isolates were genetically related. Based on the presence of integrons and disc diffusion assays, only two of the isolates examined were resistant to antibiotics. Eleven of twelve isolates contained genes for invasion, and nine of twelve contained virulence plasmids. Seven of the twelve isolates contained genes for the fimbria seen in isolates that cause human disease. The degree of invasiveness seen on avian cells ranged from 10% to 400% when compared to the human control strain. Based on the genetic characterization, these *Salmonella* strains are invasive and highly virulent, as evidenced by the gross and histologic lesions, and mortality seen in a number of bird species. Characterization patterns indicate these isolates may be capable of causing disease in humans and domestic animals suggesting epizootics in wild birds may be public health concerns.**

**(81) IDENTIFICATION OF DUCK PLAGUE VIRUS BY POLYMERASE CHAIN REACTION.**

**WALLACE R. HANSEN, National Wildlife Health Center, 6006 Schroeder Rd., Madison, Wisconsin 53711; SUSAN E. BROWN, Department of Bioagricultural Sciences and Pest Management, College of Agricultural Sciences, Colorado State University, Fort Collins, Colorado 06523; SEAN W. NASHOLD, National Wildlife Health Center, 6006 Schroeder Rd., Madison, Wisconsin 53711; and DENNIS L. KNUDSON Department of Bioagricultural Sciences and Pest Management, College of Agricultural Sciences, Colorado State University, Fort Collins, Colorado 06523.**

**Duck plague (DP) is a herpesvirus disease of Anseriformes (ducks, geese and swans) that causes annual mortality in waterfowl of the United States. To effectively manage this disease virus infected waterfowl need to be identified quickly to prevent additional disease spread. Traditional diagnostic virology methods are too slow for detecting and identifying DP virus under these circumstances. Molecular approaches offer the best solution. A polymerase chain reaction (PCR) assay was developed that is rapid, specific and sensitive for DP-DNA. Primer sets were constructed for the PCR assay using DNA sequence information from a cloned fragment of the DP vaccine virus genome coding for the DNA-polymerase. The PCR was negative, did not produce product of expected molecular size, for test DNA from nine other avian herpesviruses or normal tissue extracts. The assay can detect as little as 1 fg of DP-DNA, which is equivalent to five virus particles. The PCR clearly identified all seven historical field isolates of DP virus tested and easily detected viral DNA in tissues from diagnostic cases submitted to the National Wildlife Health Center, even in the absence of virus isolation. Positive PCR results were confirmed by comparing PCR product sequences to the known sequence of the target genome segment. The present PCR methods can provide results in 36-48 hours after sample collection. This PCR assay provides a new rapid diagnostic method for detecting DP infected waterfowl and a new tool for epizootiological studies.**

**(82) CHARACTERIZATION OF DUCK PLAGUE ISOLATES BY AMPLIFIED RESTRICTION FRAGMENT POLYMORPHISM (AFLP) ANALYSIS.**

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**Duck plague (DP) is a disease of waterfowl caused by a herpesvirus that annually causes mortality in the United States. While field isolates of virus are known to vary in virulence for some waterfowl species, suggesting that there is variation in the genomes of these DP isolates, no genetic markers have been looked for to identify the different strains. Serologically the field isolates are all identical, but restriction fragment profiles of some field isolates show that DNA sequence differences do exist. A polymerase chain reaction (PCR) based amplified restriction fragment polymorphism (AFLP) analysis method was used to compare DP field isolates to the vaccine strain of virus to find genetic markers. Field isolates of DP virus from 1973 to 1994 representing different geographical locations in the U.S. were compared by AFLP analysis. The DP vaccine virus was easily distinguishable from other field isolates by analysis of electrophoresis patterns of AFLP products in agarose gels. All field isolates examined had a similar basic AFLP electrophoresis pattern, but minor differences were also seen between them. While more work on this assay is required, it shows promise for grouping DP field isolates based on these DNA markers and using these markers for epizootiological studies.**

**(83) MOLECULAR AND IMMUNOLOGICAL EVIDENCE FOR DIVERSITY OF *PLASMODIUM RELICTUM* IN HAWAII.**

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**Periodic, epidemic outbreaks of avian malaria (*P. relictum*) threaten the survival of many species of Hawaiian honeycreepers (*Drepanidinae*). Susceptibility differs among and within honeycreeper species and their current elevational distribution can be explained by relative susceptibility or resistance to this disease. Although genetic factors of the host may play a major role in susceptibility, genetic variability of the parasite may also be important. Using immunological and PCR-based techniques, we are examining genetic diversity among geographic isolates of *P. relictum* in Hawaii. Hawaii Amakihi (*Hemignathus virens*) were infected by mosquito bite with either Hawaii or Kauai isolates of *P. relictum.* After the birds recovered from acute infections, they were challenged with the reciprocal isolate and monitored by blood smear to detect increases in parasitemia. Infected Amakihi displayed concomitant immunity to the reciprocal isolate, suggesting that the isolates were identical. Partial SSU rRNA (small subunit ribosomal RNA) genes and nearly full-length TRAP (thrombospondin-related anonymous protein) genes have been PCR (polymerase chain reaction) amplified, cloned and sequenced from the isolates used in challenge experiments and from blood from infected wild birds. DNA sequence comparisons, RFLP (restriction fragment length polymorphism) and SSCP (single stranded conformational polymorphism) are being used to evaluate diversity. Phylogenetic trees constructed from cloned partial SSU rRNA gene sequences reveal no distinct groupings based on geographic origin. However, when PCR-generated fragments from partial SSU rRNA genes (from either Hawaii or Kauai origin) are subjected to SSCP analyses, patterns appear very similar within groups (suggesting invariance within groups) but are distinct between groups. Since estimates of the nucleotide substitution rate for SSU rRNA are so low (estimated at 2-4% per 100 million years), evidence of diversity within these genes may eventually help to determine whether multiple introductions of *P. relictum* to Hawaii have occurred.**

**(84) PASSERINE AND WATERFOWL REPRODUCTIVE AND IMMUNE STATUS ON RECLAIMED WETLANDS ON OIL SANDS MINING SITES.**

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**Wild, nestling tree swallows (*Tachycineta bicolor*) and semi-captive, juvenile, mallard ducks (*Anas platyrhynchos*) inhabiting constructed and reclaimed wetlands receiving run off from mine tailings ponds on Suncor and Syncrude Oil Sands mining sites in Fort McMurray, AB, are the subjects of an ongoing study to determine the ecological viability of these areas. Immunological and reproductive variables were studied in adult and nestling tree swallows, while immune response and pathology were the focus in young-of-the-year waterfowl on these areas. Cell mediated immune function was assessed *in vivo* based upon a T lymphocyte proliferative response to intradermal mitogen challenge. Pilot studies showed a decreased T-lymphocyte responsive in pharmacologically immunosuppressed birds of both species. Adult female tree swallows from the principal reference area showed a stronger T cell response than those from the Suncor reclamation site, but due to the high standard error, this was not statistically significant. There was no difference in the T cell proliferative response of nestlings from any of the five sites under investigation. The reproductive variables examined included the number and weight of eggs per clutch, hatching success, growth rate of the chicks, and survival to 12 days of age. Although there were differences detected among some of the variables, no site was consistently higher or lower that the other sites, and the reference sites did not show greater numbers or size of eggs produced, and there was no greater growth rate or survival to fledging seen on the reference sites relative to the other four test sites. Nestling growth rates were determined using principal component analysis to represent the five actual measurements taken on each bird. Overall body sizes and growth rates did not differ among the sites.**

**No difference in the T lymphocyte proliferative response was detected in mallard ducklings from either of the two sites being studied. However, on both sites an unexpectedly high level of disease was seen in the ducks. During routine mid-study sampling 40% of the ducks from the "impacted" site, and 15% of the ducks from the reference site showed gross and/or histopathological changes in various tissues. By the final sampling time, 60% of study site and 30% of reference site birds had grossly visible pathology. In spite of NSD in the T cell proliferative response, a possible compromise in immune function in these ducks was expressed as increased morbidity and mortality during the study period.**

**This abstract describes results from year 1 of an ongoing study, designed to complement "on site" studies in lower animal and plant communities, working towards a means of detecting subtle and early contaminant related changes in vertebrates at higher trophic levels living on reclaimed, rehabilitated wetlands on large scale Oil Sands mining sites.**

**(85) ECOLOGICAL RISK ASSESSMENT AND CLEAN-UP GOALS FOR THE MOTHER LODE MERCURY MINE, PRINEVILLE, OR.**

**ANNE FAIRBROTHER and RICHARD S. BENNETT, ecological planning and toxicology, inc., 5010 SW Hout St., Corvallis, OR 97333.**

**The Mother Lode is a small abandoned mercury mine in the Ochoco Mountains 45 miles east of Prineville, OR, with soil containing up to 23,500 mg/kg (dry wt.) mercury. The mine is on a steep slope at the bottom of which is Canyon Creek, a small stream that flows five miles to the Ochoco River. An ecological risk assessment (ERA) was conducted to determine ecologically relevant clean-up goals, and was one of the first ERAs to follow the new Oregon Department of Environmental Quality guidelines. The site is within the Douglas fir climax forest ecotype, with both terrestrial and aquatic exposure pathways. Contaminants of Interest (CoI) include mercury and other heavy metals. Site-specific trophic transfer factors for the CoI were developed and the percent methyl to total mercury was characterized. No evidence of toxicity to vegetation is evident; omnivorous birds are potentially at risk from mercury. Piscivorous wildlife are not at risk, but the level of methyl mercury in fish is close to the screening benchmark values. Fish are not at risk from the CoI, but benthic invertebrates in the upper portion of the creek may be affected. Removal of the small areas of soil with mercury concentrations greater than 2,000 mg/kg (dry wt.) would eliminate the source of mercury that poses a risk to the environment.**

**(86) HEAVY METAL, RADIONUCLIDE AND ORGANOCHLORINE CONTAMINANT LEVELS IN ESKIMO HARVESTED BOWHEAD WHALES OF ARCTIC ALASKA.**

**TODD O'HARA, Department of Wildlife Management, North Slope Borough, Barrow, Alaska, 99723; GERALD BRATTON, Department of Veterinary Anatomy and Public Health, College of Veterinary Medicine, Texas A&M University, College Station, Texas; PEGGY KRAHN, National Marine Fisheries Service, National Marine Mammal Laboratory, Seattle, Washington; VICTORIA WOSHNER, Department of Veterinary Biosciences, College of Veterinary Medicine, University of Illinois, Urbana, Illinois; LEE COOPER, Environmental Sciences Division, Oak Ridge National Laboratories, Oak Ridge Tennessee.**

**Levels of contaminants in bowhead whale tissues are presented. Mean levels of metals (As, Cd, Co, Cu, Pb, Mg, Mn, Hg, Mo, Se, Ag, and Zn) studied in these bowhead whales in liver and kidney are considered low or normal for most mysticete species. However, from a consumption or subsistence perspective the kidney represents a significant source of cadmium (Cd), while other elements are not at levels of concern. Mercury (Hg) is at very low levels and is of little concern, unlike what has been detected in some odontocetes. Mean levels of metals for blubber, epidermis, and muscle in bowhead whales would be considered low or normal for most mysticete species. From a consumption or subsistence perspective these tissues are not a significant source of heavy metals. There was a significant correlation between body length and both liver and kidney Cd levels. Mercury, even at these relatively low levels, correlated with selenium (Se), Cd, and length. The mean cesium-137 (Cs-137) levels (Bq/kg w.w.) indicate that the increasing rank order for tissues is blubber < kidney/liver < skin/muscle; and levels are considered very low. All other gamma emitters were not detected. Preliminary data indicates that strontium-90 (Sr-90), and plutonium (Pu-239/240) are mostly below detection levels and thus very low. Polonium-210 (Po-210) was above detection level at approximately 5 Bq/kg and considered low. Most (7 of 11) of the organochlorines measured are at higher levels in longer (older) male whales than younger males as they were noted to be at higher concentrations with increasing length. In females, hexachlorobenzene appeared to decrease with increasing length and others did not change. In conclusion, tissues of the bowhead whale are low in most natural and anthropogenic contaminants of the inorganic, organochlorine, and radionuclide classes. We expect no adverse effects in the whales related to these contaminants, and only renal Cd requires further investigation for subsistence users and is under investigation.**

**(87) EXPOSURE OF GREAT EGRET NESTLINGS TO MERCURY THROUGH DIET IN THE EVERGLADES ECOSYSTEM.**

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**We estimated exposure of great egret (*Ardea albus*) nestlings in the Everglades to mercury in food by collecting regurgitated food samples during the 1993 - 1996 breeding seasons, and also measured concentrations of mercury in individual prey items from those samples. Great egret nestlings had a diet composed predominantly of fish (>95% of biomass), though the species composition of fishes in the diet fluctuated considerably among years. Great egrets concentrated on the larger fishes available in the marsh, especially members of the Centrarchidae. The importance of non-native fishes fluctuated from 0 - 30% of the diet by biomass. Mercury concentrations in prey fishes ranged from 0.04 - 1.40 mg/kg wet weight, and we found a significant relationship between mass of individual fish and mercury concentration. We estimated concentration of mercury in the diet as a whole by weighting fish species-specific mercury concentrations by the proportion in the diet. We estimate that mercury concentrations in the diets ranged annually from 0.37 - 0.47 mg/kg fish among years (mean = 0.412 mg/kg). We estimated mercury exposure in great egret nestlings by combining these mercury concentrations with measurements of food intake rate, as measured over the course of the nestling period in both lab and field situations. We estimate that at the 0.412 mg/kg level, nestlings would in aggregate ingest 6.49 mg mercury during an 80-day nestling period. Captive feeding studies indicate that these levels of exposure could be associated with reduced fledging mass, increased lethargy, decreased appetite, and possibly, poor health and juvenile survival.**

**(88) LEAD POISONING IN BIRDS IN SWEDEN.**

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**Lead toxicosis and high prevalence of ingested lead pellets have been observed previously in Anseriform birds in Sweden. During the period 1986 to 1996 several different species of birds, sent in for post-mortem examination to the National Veterinary Institute, were investigated for lead levels in liver and kidney. Lead toxicosis (liver value > 5 mg/kg) was observed in 44 Anseriform birds and was mainly observed in whooper swan (*Cygnus cygnus*), Canada goose (*Branta canadensis*), mute swan (*Cygnus olor*) and mallards (*Anas platyrhynchos*). The origin of the lead could not always be identified, but the most frequent found source was gunshot pellets. Among birds of prey lead toxicosis has been observed in golden eagle (*Aquila chrysaetos*) and white tailed sea eagle (*Haliaetus albicilla*) while lead levels in other birds of prey such as goshawk (*Accipiter gentilis*), common buzzard (*Buteo buteo*), tawny owl (*Strix aluco*), eagle owl (*Bubo bubo*) and long eared owl (*Asio otus*) were low with a mean value below 1 mg/kg in liver. Lead toxicosis was also observed in one grey-headed woodpecker (*Picus canus*) and one white-backed woodpecker (*Dendrocopus leucotos*). No other cases of lead poisoning were observed. The use of lead pellets is today banned for waterfowl hunting and a total ban is expected to be in force by the year 2000.**

**(89) EFFECTS OF DIET AND SOIL INGESTION ON THE TOXICITY OF ZINC TO GAME-FARM MALLARDS, *ANAS PLATYRHYNCHOS*: HEMATOLOGY AND SERUM CHEMISTRY.**

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**We conducted a 30-day acute toxicity test using female farm-raised mallards to examine the effects of diet, including soil ingestion, on the toxicity of zinc. Sixty ducks received an average dose of 0.97 g zinc in the form of 8 No. 4 shot pellets containing 98% Zn and 2% Sn, and another 60 ducks were sham-dosed. Fifteen ducks from each of the 2 dose treatment-groups were assigned to 1 of 4 dietary treatments: corn only, corn with soil, pelletized ration only, or pelletized ration with soil. The zinc dose resulted in high mortality, incoordination, paralysis and anorexia, changes in body and organ weights, and macroscopic lesions. Differences in erythrocyte and leucocyte parameters, serum enzyme activities, and metabolite concentrations were primarily associated with main (e.g dose or diet) effects. In zinc-dosed ducks, there were trends towards heavier organs, higher enzyme activities and metabolite concentrations, and reduced severity of gross lesions in ducks fed the commercial pelletized ration. Although the results suggested that a more nutritionally-complete diet may provide some protection from zinc intoxication, these effects were neither dramatic nor conclusive.**

**(90) WILDLIFE POISONING IN THE UNITED STATES: A LAW ENFORCEMENT PERSPECTIVE.**

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**Intentional poisoning of protected wildlife is illegal. Yet the intentional poisoning of species such as the Bald and Golden Eagle is a common law enforcement issue. The National Fish and Wildlife Forensics Laboratory receives and analyzes carcasses of various wildlife species suspected of being poisoned primarily through the illegal use of pesticides. Information on how to poison predator species as well as the poisons themselves are available through the illegal or "underground" market.**

**From 1990 through 1998, we have diagnosed over 200 cases of poisoning or suspected poisoning of protected species submitted by law enforcement agents. Primary (direct consumption of the poison) and secondary (consumption by a scavenger species of a victim dead from poison) poisoning are differentiated. Carbofuran is the most commonly detected pesticide used to directly poison wildlife. Aldicarb is the second most commonly misused pesticide. Both of these are commonly available carbamate pesticides and are used on baits and carcasses to target predator species such as wolves, coyotes, hawks and eagles. Famphur, an organophosphate pesticide used widely in the livestock industry, may be responsible for accidental poisoning of scavenger species, particularly eagles, because of the long term residual effect. It has also been used illegally to control pest birds which has resulted in secondary poisoning of eagles. Diazinon, a commonly used lawn chemical, has been found frequently in waterfowl. Fenthion, legally used to control pest birds, is increasingly being found responsible for secondary poisoning of bird eating raptor species. Strychnine, used legally for rodent and pigeon control, also causes secondary poisoning in raptors. Euthanasia solutions containing pentobarbital used by veterinarians is responsible for an increasing number of eagle deaths due to improper disposal of animal carcasses. A summary of eight years of diagnostic data on poisoning cases submitted by law enforcement agents along with methods used to poison wildlife will be presented.**

**(91) PATHOGENESIS OF TUBERCULOSIS IN FERRETS (*MUSTELA FURO*) EXPOSED TO LOW AND HIGH DOSES OF *MYCOBACTERIUM BOVIS* INFECTION.**

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**Tuberculosis (Tb) caused by *Mycobacterium bovis* is an important zoonotic disease affecting livestock in New Zealand. The brushtailed possum (*Trichosurus vulpecula*) has been recognized as the major wildlife reservoir of Tb in New Zealand, but recent data indicates that ferrets (*Mustela furo*) may be as well. The objectives of this study were to describe the pathogenesis of tuberculosis in ferrets (*Mustella furo*), and study humoral and cellular immune responses to develop a model for further studies. Juvenile ferrets of either gender were randomly allocated to two treatment groups (n= 25) challenged orally with either 5 X $10^6$ colony forming units (cfu) or 5 X $10^2$ cfu, and a control group (n=10) fed a sham inoculation. Five ferrets from each treatment group and 2 from the control group were necropsied at 4, 12, 20, 30, and 37 weeks post-infection (PI). Blood samples were collected for development of the enzyme linked immunosorbent test (ELISA), and the lymphocyte transformation (LT) test. Development of disease was described by gross, histological and bacteriological examinations. Histological lesions were first visible at 4 weeks post-inoculation (PI) in the high dose group as a suspicious lesion, which was confirmed on bacterial culture. Lesions as previously described were seen after 12 weeks in most ferrets in this group. Gross lesions were first visible only in the high dose group, as small granulomas in the mesenteric lymph node at 12 weeks PI. After 20 weeks PI they were typical of ferret tuberculosis. Bacterial culture identified infections only in the high dose group in 21 of 25 ferrets. ELISA was considered a highly specific test able to diagnose infections at 4 weeks and correlated to severity of disease. The LT test was unreliable, the response to purified protein derivatives of bovine (PPD-b) and avian (PPD-a) were too low or absent, and to ConA were good but absent as the disease progressed. However, ferrets with severe disease had positive and detectable responses. This model was developed to study disease transmission, immune responses and vaccination of ferrets against Tb.**

**(92) VACCINATION OF BLACK-FOOTED FERRET X SIBERIAN POLECAT HYBRIDS AGAINST CANINE DISTEMPER WITH RECOMBINANT AND MODIFIED-LIVE VIRUS VACCINES.**

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**Canine distemper continues to be a threat to captive and reintroduced black-footed ferret (*Mustela nigripes*) populations. Black-footed ferrets are currently vaccinated with an inactivated canine distemper virus vaccine; however, a safe efficacious vaccine providing long-term protection would improve management of these animals. We studied a modified-live virus canine distemper vaccine (Galaxy D) and an experimental recombinant vaccine in hybrid black-footed ferret X Siberian polecats (*Mustela eversmanni*). Eight ferrets were vaccinated subcutaneously with Galaxy D vaccine, eight ferrets were vaccinated intramuscularly with a recombinant vaccine, and eight ferrets received diluent subcutaneously as a control group. All ferrets remained clinically normal following vaccination. Animals were bled periodically postvaccination for serology, hematology, and lymphocyte blastogenesis. By day 17 postvaccination all vaccinated ferrets had seroconverted and on day 38 geometric mean titers were 2,371 and 89 for the Galaxy D and recombinant vaccine groups, respectively. The group receiving the recombinant vaccine was boostered on this day. On day 162 postvaccination geometric mean titers were 2,818 in the Galaxy D group and 251 in the recombinant group. All control ferrets remained seronegative except for one animal that was housed with a ferret vaccinated with Galaxy D. All vaccinated and four control ferrets were each challenged intranasally, intraconjunctivally, and orally with a total of 0.5 ml tissue suspension containing canine distemper virus originally derived from black-footed ferrets. None of the vaccinated ferrets but all of the control ferrets developed fatal canine distemper. Both Galaxy D and the recombinant vaccine were safe and efficacious in hybrid ferrets warranting additional study of these vaccines in black-footed ferrets.**

**(93) SUSCEPTIBILITY OF RED FOXES (*VULPES VULPES*) AND GRAY FOXES (*UROCYON CINEREOARGENTEUS*) TO INFECTION BY *EHRLICHIA CHAFFEENSIS*.**

**WILLIAM R. DAVIDSON, J. MITCHELL LOCKHART, DAVID E. STALLKNECHT, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602 and ELIZABETH W. HOWERTH, Department of Pathology, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602.**

**Red foxes (*Vulpes vulpes*) and gray foxes (*Urocyon cinereoargenteus*) were evaluated for their susceptibility to experimental infection with *Ehrlichia chaffeensis*, the causative agent of human monocytotropic ehrlichiosis. Two red foxes and three gray foxes were inoculated intravenously with *E. chaffeensis* and were monitored at 7, 14, 21, and 28 days post inoculation (DPI) for evidence of infection using an indirect fluorescent antibody (IFA) assay, light microscopy, polymerase chain reaction (PCR), and cell culture methods. One red fox and one gray fox served as negative controls. Red foxes were susceptible to infection based on reisolation of *E. chaffeensis* from blood at 7 and 14 DPI, seroconversion by 7 DPI, and positive PCR assays on spleen and lymph nodes at 28 DPI. Morulae were not found in circulating leukocytes, and clinical signs or lesions of ehrlichiosis were not observed. In contrast, gray foxes were refractory to infection based on negative results on all culture, PCR, serologic, and microscopic examinations. These findings imply that red foxes, but not gray foxes, are potential vertebrate reservoirs for *E. chaffeensis*.**

**(94) HEMATOLOGIC VALUES IN *CYTAUXZOON FELIS* INFECTED FLORIDA PANTHERS (*FELIS CONCOLOR CORYI*) AND TEXAS COUGARS (*FELIS CONCOLOR STANLEYANA*).**

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***Cytauxzoon felis*** **has been a long-recognized hemoparasite of free-ranging Florida panthers (*Puma concolor coryi*) but its potential effect on the population has not been assessed. The presence or absence of infection was determined by examination of stained blood smears for red blood cells containing piroplasms. Red blood cell indices and white blood cell counts were compared between *C. felis* positive and negative Florida panthers from south Florida and Texas cougars (*Puma concolor stanleyana*) living throughout Florida. Parameters compared were RBC, PCV, MCV, MCHC, Hb, and MCH for the erytrocytes and neutrophils, lymphocytes, eosinophils, basophils, and monocytes for the leukocyte indicies. The overall infection rate for both populations was 36.3% (33/91 with the infection rates for Texas cougars and Florida panthers are 32% (11/28) and 35% (22/63), respectively. PCV values and RBC counts were slightly, but significantly (P<.05), lower in positive compared to negative felids, but no significant differences were measured between positive and negative animals when the analysis was stratified by subspecies. Pre and post-infection samples were available for some animals. Mean pre-infection PCV, RBC and Hb values were slightly higher than respective mean post-infection values, but differences were not significant when analyzed using a paired t-test. Statistically significant differences (p<.05) were measured for the MCHC, eosinophils, neutrophils, monocytes, basophils and absolute white blood cell count from Florida panthers compared to values from Texas cougars, without regard to infection status. Although some statistically significant differences were measured, biologically significant differences were not likely, because differences were small and values were generally within expected reference ranges for healthy animals.**

**(95) SEROLOGICAL SURVEY OF INFECTIOUS DISEASE AGENTS OF BLACK BEARS, *URSUS AMERICANUS*, IN NORTHERN CALIFORNIA, OREGON AND WASHINGTON.**

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**The causes of natural mortality and disease in free-ranging black bears, *Ursus americanus*, in northern California, Oregon and Washington are poorly known. One hundred and ninety-nine black bear serum samples were collected between 1993 and 1997 and tested for selected viral and bacterial disease agents. Antibody prevalence was 0% for bluetongue virus, 12.6% (24/190) for *Borrelia burgdorferi* (Lyme disease), 0% for *Brucella spp*., 0% for *Dirofilaria immitis* (heartworm), 4.8% (8/165) for distemper virus, 4.5% (9/199) for *Ehrlichia equi*, 0% for epizootic hemorrhagic disease virus, 9% (8/88) for *Francisella tularensis* (tularemia), 1.8% (3/165) for canine infectious hepatitis virus, 2.5% (5/198) for *Trichinella spiralis*, 45% (89/198) for *Toxoplasma gondii* and 5.5% (11/198) for *Yersinia pestis* (plague). Prevalence differences were observed between study sites. Lyme disease and plague antibodies were mainly detected in black bears from northern California and Oregon. *E. equi* antibody detection was highest from northern California. Prevalence rates for *T. gondii* antibodies were greatest in females and in 1 to 4 year old bears of both sexes. Five of the eight distemper positive samples were from bears that had overlapping home ranges with one being a translocated urban nuisance bear. *B. burgdorferi* antibody prevalence rates increased during the sampling period. This is the first report of *E. equi* in the family Ursidae.**

**(96) THE ROLE OF AN ENDEMIC VIRAL INFECTION ON INDIVIDUALS AND ON POPULATION DYNAMICS IN EUROPEAN WOOD MICE, *APODEMUS SYLVATICUS* AND BANK VOLES, *CLETHRIONYMUS GLAREOLUS*.**

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**Cowpox is an endemic viral infection of European wood mice, *Apodemus sylvaticus* and bank voles, *Clethrionymus glareolus*. There are no apparent clinical signs of cowpox in these wild rodents, so the incidence of infection has been monitered, in two woodland populations, by monthly serosurveys, for the past 3 years. All animals in these populations are individually identified and so times of seroconversion to the virus, subsequent rodent survival rates and preferentially infected groups, within these populations are becoming established. Results show that infection delays onset of breeding in captive rodents and in the wild, some infected rodents fail to achieve adult body weights. Poxviral DNA has been demonstrated by PCR, from rodent blood samples and the emerging epidemiology of this endemic virus will be discussed.**

## (97) PATHOLOGY OF HEPATITIS B IN ARCTIC GROUND SQUIRRELS.

**JOHN BLAKE, CHRISTINE TERZI, and KIMBERLEE BECKMEN. Institute of Arctic Biology, PO Box 757000, University of Alaska Fairbanks, Fairbanks, AK 99775-7000.**

**Since 1987, the Institute of Arctic Biology has used Arctic ground squirrels (AGS) in a wide variety of research programs including: hibernation, reproductive biology, neurobiology, stroke, fat metabolism, and non-shivering thermogenesis. In 1990, during necropsies conducted as part of the Institute's animal care program, we identified hepatic tumors in these experimental animals. The etiology of these tumors was not identified until 1995 when archived tissues were analyzed at the Pasteur Institute and a new hepadnavirus was identified [(1)]. To date, Arctic ground squirrels infected with hepatitis B virus have been obtained from 2 very distinct sites in Alaska: the northern foothills of the Brooks Range (Toolik Lake) and the southern foothills of the Alaska Range (along the Denali Highway). Using dot blots, prevalence rates ranging from 0-17% have been observed in groups of squirrels brought into captivity. Lesions associated with the AGS hepadnavirus infection are varied. Liver tumors are single to multiple, discrete, expansile nodules ranging in diameter from a few millimeters to a centimeter. Tumor cells are disorganized and contain abundant lipid. There is also an irregular pattern of lipid accumulation in hepatocytes. Nuclei are often large and vesicular. Hepatitis B surface antigen (HBsAg) is detectable in hepatocytes surrounding the tumors but not in cells within the tumor (using a polyclonal anti-woodchuck-HBsAg antibody). Active hepatitis with hepatic swelling, necrosis and accumulation of inflammatory cells is periodically seen with or without tumors. Similar to human hepatitis B, we have observed high morbidity/mortality from severe glomerulonephritis in juvenile (approximately 4-months-old) AGS born to hepatitis B infected mothers. If confirmed, a causal relationship would augment the woodchuck hepatitis B model since the juvenile glomerulonephritis does not occur with high frequency in this animal. Transmission of the organism is thought to be through bite wounds and sexual transmission, similar to woodchuck hepatitis B. A combined population biology - epidemiology study is currently underway.**

**[(1)] Testut, P., C-A. Renard, O. Terradillos, L. Vitvitski-Trepo, F. Tekaia, C. Degott, J. Blake, B. Boyer, M.A. Buendia. 1996. A new hepadnavirus endemic in Arctic Ground Squirrels in Alaska. Journal of Virology. 70(7):4210-4219.**

**(98) PROBABLE MALIGNANT CERVICAL NONCHROMAFFIN PARAGANGLIOMA WITH PULMONARY AND CARDIAC METASTASES IN A SOUTHERN SEA OTTER (*ENHYDRA LUTRIS*).**

**HOWARD STEINBERG, The Department of Pathobiological Sciences, School of Veterinary Medicine, University of Wisconsin - Madison, Madison, Wisconsin, 53706, CAROL U. METEYER, U.S. Geologic Survey, National Wildlife Health Center, Madison, Wisconsin, 53711, ELIZABETH J. GALBRAITH, Bristol-Meyers Squibb Pharmaceutical Research Institute, Princeton, New Jersey, 08543, and DOUG M. ENGLAND, Meriter Hospital, Madison, Wisconsin, 53715.**

**An emaciated adult female Sea Otter (*Enhydra lutris*) found dead off the California coast had a large soft tissue tumor attached to, but separate from, the right thyroid gland with metastases to the right atrioventricular heart valve and lung. Microscopically the neoplastic cells were arranged in an organoid pattern outlined by a fine reticular fiber network. The tumor cells stained positively for antibody to neuron specific enolase. Ultrastructurally, the tumor consisted of light and dark epithelial cells with abundant cytoplasm containing numerous polyribosomes, moderate numbers of mitochondria, sparse rER, occasional lipid droplets, rare small dense granules, and large nuclei with peripheral heterochromatin and prominent nucleoli, when present. A diagnosis of malignant cervical nonchromaffin paraganglioma was made based on morphologic, immunohistochemical and ultrastructural features.**

## (99) FORENSIC EVALUATIONS OF WOLF CARCASSES: IS MS RED RIDING HOOD GUILTY?

**RICHARD K. STROUD, US Fish and Wildlife Service, Fish and Wildlife Forensics Laboratory, 1490 E. Main, Ashland, Oregon 97530.**

**Reestablishment of the gray, red and Mexican wolf in various native habitats in the lower 48 states has been a controversial issue. Circumstances surrounding the deaths of these wolves are investigated by the Special Agents of the Fish and Wildlife Service. Since 1992, 54 carcasses of wolves killed under suspicious circumstances were submitted to the National Fish and Wildlife Forensics Laboratory, in Ashland, Oregon, to determine the cause, manner and mechanism of death and to collect trace evidence useful in the pursuit of legal cases. Data on general causes of death including gunshot, poisonings and natural causes will be presented and significant pathological lesions illustrated. Interesting cases will be discussed which demonstrate the process of medicolegal investigation.**

**(100) IMPLANTATION OF INTRAPERITONEAL RADIOTRANSMITTERS IN BROWN BEARS (*URSUS ARCTOS*), WOLVERINES (*GULO GULO*) AND LYNX (*LYNX LYNX*): ANESTHETIC AND SURGICAL PROCEDURES FOR FIELD USE.**

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**Traditionally, collars have been used for radiotagging of wild carnivores. However, in neonates, juveniles and certain species, radiocollars may cause problems, and implantable transmitters have been introduced in several ecological studies on carnivores in Scandinavia. From 1995 to 1997, surgical implantation of intraperitoneal radiotransmitters was performed in 19 brown bears (*Ursus arctos*), 49 wolverines (*Gulo gulo*) and 13 lynx (*Lynx lynx*). Eleven wolverines and 3 lynx underwent a second surgical procedure within 3-12 months in order to change the implant. The animals were chemically immobilized with medetomidine-tiletamine/zolazepam (brown bears), medetomidine-ketamine (wolverines and lynx) or xylazine-ketamine (wolverines and lynx). Implantations were performed in the field using standard surgical principles and techniques. A ventral midline approach for access to the peritoneal cavity was used in all species. After prewarming to body temperature, disinfection with benzalkonium chloride and washing with sterile saline, the transmitter was placed intraperitoneally. The wound was closed in two layers with absorbable sutures, using a simple interrupted pattern in the linea alba and a horizontal interrupted mattress pattern in the skin. The peritoneum and subcutaneous tissue were not sutured. All animals were given a prophylactic injection of procaine benzylpenicillin and dihydrostreptomycin. No mortalities that can be related to the surgical procedures or to the implant, have occurred. Implantations have been performed in neonatal lynx, juvenile brown bears and wolverines, lactating brown bears and wolverines, and in adult brown bears and wolverines. Several female wolverines with implants have whelped and successfully raised cubs. In conclusion, implantable transmitters can be recommended for radiotelemetry studies in brown bears, wolverines and lynx. The method may be applicable in other wild carnivores, especially in neonates, juveniles and in species with an anatomy that complicates the use of radiocollars.**

**(101) PORCINE ZONA PELLUCIDA IMMUNOCONTRACEPTION IN COYOTES, *CANIS LATRANS*.**

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**We conducted studies of porcine zona pellucida (PZP) immunocontraception in coyotes. Our objectives were to determine if PZP was effective at reducing or eliminating litters in coyotes and, if so, to determine the mechanism of action of PZP. Fifteen female coyotes were assigned to a Control, Peptide, or PZP group (n = 5/group). In early December 1997, all animals were administered vaccines by subcutaneous injection. The Control group received 0.5 ml of saline in 0.5ml of complete Freund's adjuvant (CFA), the Peptide group received 300 g (0.5 ml) of three distinct peptides synthesized from two of the proteins which comprise PZP in 0.5 ml CFA, and the PZP group received 300 g (0.5 ml) of PZP in 0.5 ml of CFA. Additional booster vaccinations were administered 30 and 44 days after the initial injection. Vaccinations of PZP resulted in absolute infertility. However, because there were no differences (P > 0.05) in pregnancy rate between the Control and Peptide groups, our initial attempt to isolate the immunogenic portion of the PZP molecule responsible for infertility failed. Post-mortem examination of reproductive tissues failed to provide evidence that PZP affects reproductive tract morphology. There were not treatment effects on ovarian weight (P = 0.232), oviduct weight (P = 0.249), number of corpra lutea (P = 0.114), or the number of resorptions (P = 0.585). We provide additional data on antibody titer levels and histology in an effort to localize the mechanism of action of PZP antibodies within the reproductive tract.**

## (102) SHOULD TRADITIONAL RABIES DIAGNOSIS BE ATTEMPTED ON FIXED TISSUE SAMPLES?

**CATHLEEN A. HANLON, JOHN SHADDOCK, and CHARLES E. RUPPRECHT, Centers for Disease Control and Prevention, 1600 Clifton Road, Atlanta, GA 30333.**

**Wildlife rabies is endemic throughout the United States, with the exception of Hawaii. The direct fluorescent antibody (dFA) test is the preferred diagnostic procedure for rabies evaluation. The optimal sample consists of acetone-fixed tissue impressions or brain smears from the brainstem, cerebellum and hippocampus of suspect mammals. In some cases, despite the possibility of rabies as a differential diagnosis and the need for fresh brain tissue, samples may inadvertently during necropsy be placed in another fixative, such as formalin, for histopathologic evaluation. Specimens mishandled in this fashion are generally considered non-testable by the majority of rabies diagnostic laboratories. To evaluate the effect of formalin upon subsequent diagnosis by dFA, brain tissue from uninfected, normal animals and experimentally- and naturally-infected rabid animals were evaluated by dFA and then placed in formalin for varying lengths of time. After removal from formalin and soaking in PBS, the material was then subjected to dFA. Application of dFA was useful in confirming rabies in 100% of samples from experimental animals which were subjected to formalin for approximately 10 minutes but sensitivity declined when samples remained in formalin for an hour or more. In addition, 100% of samples from naturally-infected mammals (such as raccoons) were still positive following 1 hour in formalin. The specificity of dFA was unaffected by placing the tissue in formalin; all rabies-negative tissue remained dFA-negative. The sensitivity of dFA was diminished and correlated with the length of time that tissue remained in formalin and the relative size of the brain tissue. Thus, it appears reasonable to attempt dFA on mishandled specimens in formalin, especially if the interval is short and the tissue sample is large. Additional testing on a variety of species and rabies variants under common laboratory conditions is warranted to demonstrate the utility of this approach. Alternatively, samples may be subject to further testing in reference laboratories using techniques such dFA, specially adapted for formalin-fixed tissue, immunohistochemical techniques, or the reverse transcriptase polymerase chain reaction assay.**

**(103) WHEN SIZE MAY MATTER: RABIES IN THE BEAVER (*CASTOR CANADENSIS*).**

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**Despite their ubiquity in distribution and abundance, and their diversity as the largest mammalian Order, documented cases of rabies in rodents are uncommon. To date, there are no known rodent reservoirs nor identified human rabies cases attributable to contact with rabid rodents in the United States. Detection of rabies in rodents depends in part on several variables, such as the probability of rodent contact with reservoir species; the ability of the individual rodent to survive an initial, presumably traumatic, encounter with a naturally infected animal, typically a carnivore; the likelihood that the infected rodent can avoid terminal predation, particularly during the debilitating stages of encephalomyelitis; and the opportunity for human interaction, and successful submission and testing at the diagnostic laboratory. Thus, it is not surprising that when rabies is diagnosed, large-bodied (>1 kg) rodents predominate, and significant long-term spatial-temporal patterns may gradually appear when particular hosts or viral variants emerge, especially in areas of high human population density. For example, from 1953 to 1976, during a period when nonspecific results based on inclusion bodies could have led to false-positive diagnoses, only 3 rabid beavers were recorded, despite their occurrence throughout the continental United States. In contrast, from 1977 through 1996, 16 rabid beavers were recorded, all within the eastern raccoon rabies enzootic, at a time when laboratory test specificity had dramatically improved. Little is known concerning the pathogenesis and epizootiology of such cases in beaver. Retrieval of the head of a single naturally infected animal from Cabarrus County, North Carolina, during April 1998 permitted additional analysis beyond primary rabies diagnosis. Examination of the head revealed no gross lesions. Immunofluorescent and immunohistochemical tests for rabies virus antigen were positive for all tissues examined, including skin, tonsil, tongue, lymph nodes, cranial nerves, and salivary glands. By monoclonal antibody typing, spillover infection resulted from the rabies virus variant associated with raccoons in the eastern United States. As the largest rodent in North America, with prominent mandibles and incisors, beavers may inflict severe bites. On the basis of these very preliminary results suggesting vector competence, laboratory submission of suspect beavers and other rodents should continue to be evaluated on a case-by-case basis, particularly in situations with unprovoked human or domestic animal exposure by an ill animal.**

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