<table>
<thead>
<tr>
<th>Animal Group(s) Affected</th>
<th>Transmission</th>
<th>Clinical Signs</th>
<th>Severity</th>
<th>Treatment</th>
<th>Prevention and Control</th>
<th>Zoonotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captive psittacine birds</td>
<td>Primarily direct transmission via urofeco-oral route.</td>
<td>“Proventricular dilatation disease” (PDD); infection may range from asymptomatic to severe gastrointestinal signs with or without neurological signs.</td>
<td>Birds infected with ABV may or may not show clinical disease. Once clinical signs develop, PDD is generally considered a progressive disease which ultimately becomes fatal. Acute outbreaks with high mortality have been described in psittacine avaries.</td>
<td>No specific treatment. Supportive and symptomatic treatment and good husbandry can prolong life. Possibility of complete cure is not certain.</td>
<td>No vaccine. Avoid introducing ABV into new flocks. Excellent husbandry practices, strict quarantine protocols including determining the disease and ABV status of all newly introduced birds. Isolate infected or exposed birds.</td>
<td>No.</td>
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<td>Canaries</td>
<td>Viral shedding in urine, feces, choanal secretions and feathers. Increasing evidence for vertical transmission</td>
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<td>Wild free-ranging goose, swan, duck and gull species.</td>
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**Fact Sheet compiled by:** Pauline Delnatte and Dale A. Smith  
**Sheet completed on:** 11 August 2013  
**Fact Sheet Reviewed by:** Ady Gancz; Ian Tizard

**Susceptible animal groups:**  
Psittacines: Avian bornavirus (ABV) infection has been reported in more than 80 species of captive psittacine birds in at least 33 different genera. Certain species, such as African grey parrots, blue and gold macaws, cockatoos, and Amazon parrots, seem affected most frequently.  
Passerines: ABV infection and disease have been described in captive canaries (*Serinus canaria*).  
Anseriformes and Laridae: ABV infection has been identified in wild Canada geese, snow geese, Ross’s geese, trumpeter swans and mute swans. ABV has been detected in a small percentage of northern shovelers, northern pintails, gadwalls, mallards, American widgeons, redheads, herring gulls, ring-billed gulls, and laughing gulls. The list of families and species of birds from which ABV has been recovered has expanded rapidly. One case has been reported in a bald eagle. It is likely that additional susceptible species will be described. Variety of species have been reported with consistent pathologic lesions.

**Causative organism:** ABV is an enveloped RNA virus belonging to the family *Bornaviridae*. It was first identified in 2008 as the cause of PDD in psittacine birds. Since then, 12 ABV genotypes have been identified to date based on sequence identity, including seven ABV genotypes from psittacine birds (ABV-1 to ABV-7), four genotypes from canaries (ABV-C1 to ABV-C4) and one genotype from free-ranging waterfowl (ABV-
### Zoonotic potential
None reported.

### Distribution

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<th>Psittacines:</th>
<th>The clinical syndrome of ABV infection in psittacine birds (“PDD”) was first identified in the late 1970’s in the United States. ABV infection and associated disease have been described in captive psittacines in North America, Europe, Australia, the Middle East, South America, South Africa and Japan. Worldwide dissemination is assumed to have resulted from the trade in captive birds. In 2013, a preliminary report described ABV infection in free-ranging parrots from Brazil. The significance of this report is still to be determined; large-scale active surveillance for ABV has not yet been carried out in the wild.</th>
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<tr>
<td>Passerines:</td>
<td>ABV infection in canaries has been reported from Europe.</td>
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<tr>
<td>Anseriformes and Laridae:</td>
<td>To date, ABV infection in wild birds only has been reported in North America. However, given the migratory nature of the host species, ABV-CG infection is unlikely to be restricted to North America.</td>
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### Incubation period
This aspect has been poorly investigated but appears extremely variable. Reports suggest a minimum of 11 days under experimental conditions up to months or years under natural conditions. Suggestions have been made of an acute form with birds dying within days or weeks after acute onset of clinical signs and a persistent form where birds are able to live for years without clinical impairment.

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

### Post mortem, gross, or histologic findings
Consistency among clinical signs and gross and histologic findings is not always present. Gross lesions include mild to severe emaciation, atrophy of the pectoral, proventricular and ventricular muscles, dilation of inguillum, proventriculus or ventriculus, ventricular muscle atrophy and duodenal distension. Proventricular rupture and resulting peritonitis have been rarely reported. Accumulation of fluid in the subarachnoid space has been reported in birds. Occasionally, no gross lesions are observed.

Microscopic lesions consist of non-suppurative inflammation in peripheral, central and autonomic nervous tissues. Similar infiltrates may also be present in adrenal glands and myocardium. Cerebellar Purkinje cell necrosis, neuronophagia, myelin degeneration, gliosis and axonal swelling can accompany the inflammatory lesions.

### Diagnosis
PDD as a clinical entity and infection with ABV are not synonymous. Histological lesions in biopsy or post-mortem samples remain the gold standard for diagnosis of PDD. Detection of virus/viral RNA/viral antigen or antibodies against ABV provides evidence of infection or exposure, but does not differentiate among patients with clinical PDD, asymptomatic shedders and previously exposed birds. Optimal screening protocols for PDD and/or ABV infection in psittacine flocks are yet to be determined but would probably involve a combination of repeated RT-PCR assessment of choanal and/or cloacal swabs or feathers and serology.
AVIAN BORNAVIRUSES

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

Detection of virus, RNA or antigen:
Reverse Transcriptase Polymerase Chain Reaction (RT-PCR): Both gel-based RT-PCR and real time RT-PCR have been developed using primers for various segments of the ABV genome.
Immunohistochemistry (IHC): IHC can be used to identify ABV antigen in formalin fixed biopsy or necropsy specimens. Moderately intense, diffuse, intracytoplasmic staining accompanied by intranuclear staining in neurons and certain epithelial cells is considered positive for viral antigen presence.

Virus culture: Various genotypes of ABV have been successfully grown in duck and quail cell lines. Cytopathic effects do not occur, thus virus must be demonstrated by Western blot, immunohistochemistry, indirect immunofluorescence or RT-PCR.

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

Detection of antibodies: Indirect immunofluorescence, Western blot, and indirect ELISA assays using various sources of primary antigen have been used in psittacine birds and waterfowl. Test specificity and sensitivity are difficult to determine and compare due to our poor understanding of the epidemiology of the disease, the absence of a gold standard for diagnosis, and the relatively poor correlation between the presence of antibodies, fecal shedding of ABV, and the presence of pathologic lesions and/or clinical disease.

Material required for laboratory analysis:
Antemortem: crop tissue (histology +/- IHC and RT-PCR); choanal and cloacal swabs, feces, and calami of plucked chest contour feathers (RT-PCR); serum (serology). Pooling multiple droppings from a single bird over several days or samples from multiple birds in an aviary increases test sensitivity as shedding of virus can be intermittent.
Post-mortem: Submission of brain, proventriculus, ventriculus, adrenals and vitreous of the eye are recommended as the most consistently infected tissues for RT-PCR +/- sequencing; brain, proventriculus, ventriculus can be submitted for histology +/- IHC. RT-PCR can also be performed from formalin-fixed paraffin embedded (FFPE) tissues in some laboratories.

Relevant diagnostic laboratories:
Histopathologic assessment: can be performed by any veterinary pathology diagnostic service.
RT-PCR testing: can be carried out by any molecular laboratory with appropriate primers. Information on test validation and primer selection should be requested.

Diagnostic Services with a Research Focus:
- Avian bornavirus PCR Testing Services, Department of Veterinary Pathobiology, Texas AgriLife Research - Texas A&M University, Texas, USA. http://vetmed.tamu.edu/schubot/services/
- Animal Health Laboratory, University of Guelph, Guelph, Ontario, Canada. http://www.guelphlabservices.com/ahl/

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

Treatment: No specific treatment exists. Supportive and symptomatic therapy may prolong life for months to years. Inconsistent results with the use of various NSAIDs, immuno-suppressive drugs and antiviral drugs. Non-steroidal anti-inflammatory drugs: Celecoxib, tepoxalin and meloxicam have been recommended but evidence of effectiveness has been inconsistent. A recent study in cockatiels suggested that the use of
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Meloxicam would actually be detrimental to birds affected with PDD.

**Immunosuppressive protocols:** These treatments may be of therapeutic benefit, especially selective T-cell elimination. Research on the efficacy of cyclosporine is currently underway.

**Antiviral drugs:** These drugs (e.g., amantadine) have been described as beneficial by some authors, but have been reported as having no apparent effect on fecal shedding of virus by others. Ribavirin readily kills ABV in tissue culture but does not appear to have a measurable effect on viral shedding *in vivo.*

**Prevention and control:** Preventive measures are intended to prevent introduction of ABV into new flocks and include excellent husbandry and sanitation practices and strict quarantine protocols. As no gold standard diagnostic test exists and interpretation of the result of diagnostic tests can be challenging with intermittent shedding, asymptomatic carriers, and so on, determination of the ABV status of birds in quarantine can be problematic. The possibility of vertical transmission of ABV complicates the management of infected aviaries. Pairing ABV positive birds, incubating their eggs artificially, and hand-raising the chicks separately until their ABV status is determined may be a viable option for critically endangered species. Currently no vaccine against ABV infection exists. Furthermore recent studies have suggested that serum antibodies are not protective and that ABV escapes recognition by the innate immune system; immune responses may actually increase the severity of disease so vaccination may be contraindicated.

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

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

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

**Measures required for introducing animals to infected animal:** The mixing of infected and non-infected birds is not recommended. No known strategy to prevent ABV transmission between in-contact birds is available.

**Conditions for restoring disease-free status after an outbreak:** No system of “ABV-free” certification is possible. When ABV infection is identified in an aviary, efforts should be made to determine the disease and ABV infection status of all birds, and to separately manage birds showing evidence of infection or disease from those that do not. As described above, reliable identification of infected birds this may be difficult.

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