Avian bornavirus and proventricular dilatation disease

Fact sheet

August 2023

Key points

- Avian bornaviruses, discovered in 2008, can infect a wide range of parrots, as well as waterbirds, canaries, and a number of other avian species.
- Infection can result in nervous system and intestinal disease, although some birds have no clinical signs.
- Disease in parrots, known as proventricular dilatation disease (PDD), is chronic, generally fatal and has had devastating impacts on aviary birds overseas.
- Multiple genotypes of parrot bornavirus have disseminated globally with the trade in aviary birds.
- Other avian bornaviruses cause disease and mortality in some wild bird species overseas.
- Parrot bornavirus and PDD have been diagnosed in a small number of aviary parrots in Australia but avian bornaviruses have **not** been detected in wild birds in Australia.

Aetiology

Order (Mononegavirals), family *(Bornaviridae)*, genus (*orthobornavirus*).

Avian bornaviruses are RNA enveloped viruses that replicate in the nucleus of infected cells. At least 15 genetically diverse viruses have been described [1].

Parrots: eight parrot bornaviruses (PaBV-1 to -8) have been detected. PaBV-2 and PaBV-4 are the most common; the other PaBV are very rarely detected [1].

Waterbirds: two aquatic bird bornaviruses (AABV-1 and -2) have been described from wild birds in the orders Anseriformes (ducks and geese) and Charadriiformes (shore birds) [1, 2].

Passerines: five bornaviruses (CnBV-1, -2 and -3, MuBV-1 and EsBV-1) have been described from canaries (*Serinus canaria*) [3] and finches [1, 4].

One Health implications

Wildlife and the environment: avian bornaviruses have **not** been reported in wild birds in Australia. The potential movement of PaBV from aviary birds into wild birds could pose a risk to native parrot species in Australia [5]. Parrot bornavirus is on the National Priority List of Exotic Environmental Pests, Weeds and Diseases (www.agriculture.gov.au/biosecurity-

trade/policy/environmental/priority-list) as a high-priory exotic disease that may have biosecurity impacts on Australian wildlife.

Domestic animals: there is no evidence that avian bornaviruses can infect non-avian species but the risk to domestic species is not well understood.

Humans: there have been no reports of ABV in humans and it is not considered zoonotic.

Natural hosts

The natural reservoirs for ABVs are not known [6].

Parrots (Psittaciformes)

Parrot bornaviruses have either been detected, or suspected (based on histological lesions), in over 70 species of parrot globally. It is likely that most if not all psittacines are susceptible to infection although avian bornaviruses have **not** been detected in wild parrots [1]. PaBVs have been detected in captive species whose original range would have included the Indo-pacific, Asia, Africa and Central and South America [7].

Australian species known to develop PDD or have been detected with ABV infection in captivity (overseas) include:

- sulphur-crested cockatoo (*Cacatua galareta*)
- palm cockatoo (*Probosciger atterimus*),
- red-tailed black cockatoo (*Calyptorhynchus magnificus*)
- galah (*Eolophus roseicapillus*)
- gang gang (*Callocephalon fimbriatum*)
- cockatiel (*Nymphicus hollandicus*)
- red-capped parrot (*Purpureicephalus spurius*)
- eclectus parrot (*Eclectus roratus*) [7]-.

It is highly likely that most native Australian species of parrots are susceptible to infection.

Passerines

Passerine bornaviruses have been described overseas in canaries and other finches [1]. Disease and microscopic lesions resembling PDD have described in canaries [4]. Proventricular dilatation disease has been described in a greenfinch (*Carduelis chloris*), a long-wattled umbrella bird (*Cephalopterus penduliger*), a bearded barbet (*Lybius dubius*), honey creeper, and weaver finch [all species exotic to Australia] [8].

Waterbirds

Proventricular dilatation disease-like lesions were first reported in Canada geese in 1991 [9]. **ABBV-1** has been detected in wild Canada geese and trumpeter swans (*Cygnus buccinator*) and mute swans (*C. olor*) in North America [2, 10-12] and has been detected by PCR from the brains of snow geese (*Chen caerulescens*) and Ross's geese (*C. rossii*) in the USA [13].

ABBV-1 was isolated from fibroblasts derived from Pekin duck eggs in North America and viral RNA has been detected in Pekin duck eggs from commercial sources, suggesting that the virus is present

in some commercial flocks [13]. Pekin ducks were inadvertently infected with ABV genotype 4 when exposed to infected cockatiels [14].

ABBV-2 has been identified in one study from the brains of mallards *(Anas platyrhynchos)* and a wood duck *(Aix sponsa)* in the USA but its significance and prevalence are not known [15].

Other species

ABBV-1 has been detected:

- in the brain of a wild bald eagle (*Haliaeetus leucocephalus*) with encephalitis and from a wild Mississippi kite (*Ictinia mississippiensis*) [13]
- from the brain of a neurological, euthanased zoo emu in Canada. Exposure to the virus was presumed to be as a result of environmental contamination from grazing Canada geese [16]
- in three species of North American gull; herring gull (*Larus argentatus)*, ring-billed gull (*L. delawarensis)* and laughing gull (*L. atricilla)* [13].

Lesions resembling PDD have been described in other avian species [17, 18].

World distribution

PaBV have been found in captive psittacines worldwide. It is likely that the virus has spread as the result of the international trade in wild-caught and domestically raised parrots [1, 7, 19-23]. There is **no** strong evidence for ABV in wild psittacine species [1]

ABBV-1 has been identified in waterfowl and other bird species in North America and Europe [1, 2, 11, 24, 25]. **ABBV-2** has been found only in North America [15].

Canary bornaviruses have been described in canaries from Germany. The finch bornavirus was identified in a common avicultural species of finch in Japan [4].

Occurrences in Australia

There have been **no** confirmed reports of ABV in Australian wild birds.

Proventricular dilatation disease was first reported in Australia in 1997 in a captive green-winged macaw (*Ara chloroptera*) imported into Australia in 1993 [26]. PDD has since been described in at least five other non-native parrots including a red-sided eclectus parrot (*Eclectus roratus polychloros*), Moluccan cockatoo (*Cacatua moluccenensis*), sun conure (*Aratinga solstitialis*), African grey parrot (*Psittacus erithacus erithacus*) [27] and blue and gold macaw [28]. The most recent report is from 2023. An informal survey of a group of Australian avian practitioners in late 2008 indicated that 7 of 35 had cases of PDD documented by histopathology (D Phalen pers. comm. 2013).

PaBV-2 was sequenced from tissues from a Moluccan cockatoo from a Qld aviary [22]. PaBV-4 has been detected in the tissues of a psittacine bird with PDD. Uncharacterized genotypes of PaBV have also been detected in droppings from two other subclinically infected psittacine birds in aviaries on the east coast of Australia (D Phalen pers. com. 2013).

Wild cockatoos in Vic were tested for parrot bornaviruses with an indirect immunofluorescence serology, PCR and histopathology. Anti-PaBV antibodies were detected in one of twenty-four birds,

but PCR and histopathology did not detect PaBV [29]. The significance of this single seropositive finding is not clear.

Epidemiology

Proventricular dilatation disease of psittacine birds was first described in the late 1970s in Europe and the USA, however avian bornavirus was only identified as the cause in 2008 [19, 30, 31]. The epidemiology of PaBV in parrots is complex. Infected birds may or may not develop disease [32]. Incubation periods between infection and the development of disease may range from a few weeks to many years.

Subclinically infected birds may shed virus for years, possibly their entire life [10]. Birds may shed virus continuously, intermittently, or rarely, and it is possible that some may never shed virus. Virus may be shed in oral secretions, faeces, urine, feathers and feather dander [33].

The routes and methods of natural bornavirus transmission are not well understood [1]. It is assumed spread of the virus is primarily through direct contact or contamination of the environment with shed virus. Virus ingestion may be one route of infection. Inhalation of aerosolised particles contaminated with virus is another possible but unconfirmed route of infection.

It is possible that some but not all ABV infected parrots can vertically infect their offspring [13, 34, 35]. Horizontal transmission after hatch is also likely [35].

Viral transmission appears to be ineffective as parrots and canaries shedding virus can live with other birds for long periods without spreading infection. The factors that affect the infectivity of shed virus and the susceptibility of a new host are not known [4, 36].

Infection prevalence within flocks of avicultural species can vary substantially from 0-28% of birds infected [7]. Disease prevalence can also vary substantially with no known history of disease in some infected collections to the loss of the majority of a collection over a period of a few years in others.

The pathogenesis (manner in which disease develops) of bornavirus infection is not known. It seems likely that disease results in part from the host's immune response to infection [6]. There are probably multiple factors directly or indirectly influencing the immune response, including virus and host genetics, the host´s immune status, co-infections with other pathogens and stress [1].

Little is known about significance of ABV infection in waterbirds and passerines. Infection with ABBV-1 appears widespread in free-ranging waterfowl (and gulls) in North America, but is associated with disease only in a small subset of cases [11, 37, 38].

Clinical signs

Parrots: many PaBV infections do not result in disease. Disease is the result damage to the nervous system that may be viral induced or the result of the host response to infection. Signs correspond to damage to the central nervous system and to the nerves controlling the motility of the digestive tract.

Central nervous system signs are typically slow to develop, progressive and include mentation changes, ataxia (loss of balance) and progressive weakness developing into paralysis. Seizures and blindness, although rare, can occur [33, 39].

Damage to digestive system nerves results in alterations in gut motility and even paralysis of the gut, which impacts the bird's ability to digest food. Signs include passage of whole seeds in the droppings, diarrhoea, regurgitation, delayed crop emptying and weight loss. Many birds are emaciated on presentation [1].

Waterbirds: Canada geese affected with AABV-1 had similar clinical signs to parrots with PDD [12].

Passerines: canaries infected with canary bornaviruses can show PDD-like clinical signs and pathologic lesions [4].

Diagnosis and laboratory procedures

Testing sensitivity for individual birds is improved when both PCR and antibody tests are run concurrently [40].

PCR is used to detect ABVs in samples collected from live birds. The tests need to be designed to detect the specific virus that is expected to be found in the species of bird tested [35, 40]. Avian bornaviruses have been detected in oral and tracheal swabs, cloacal swabs, crop biopsies, blood and feathers [35]. In the live bird, blood, faeces, feathers, urine and crop, choanal and cloacal swabs can be submitted for PCR.

Virus shedding can be extremely variable in frequency and amount. Recommendations for testing include repeated testing of cloacal swabs or droppings (a minimum of three samples), possibly in conjunction with PCR analysis of feathers [40, 41].

Some birds infected with ABV develop **antibodies**. Others go long periods without developing antibodies and then will suddenly develop them [33]. The sudden onset of antibody production may indicate the onset of clinical signs. However, many seropositive birds do not show signs of disease. Western blot assays, enzyme-linked immunoassays and immunofluorescent assays can be used [42].

Clinical signs of PDD are not sufficient to make a diagnosis. In advanced cases, plain and contrast **radiographs** demonstrate distension (sometimes massive) of the proventriculus and ventriculus. Dilatation of the intestines may occur less frequently. The presence of gas in any part of the digestive tract is an indication of altered gastrointestinal motility [33, 41].

A confirmed diagnosis of PDD can only be made by demonstrating specific inflammatory lesions in affected nerves. Crop biopsies are the easiest and safest means of obtaining diagnostic tissue from the live bird, but only around 50% of birds with PDD will have lesions on crop biopsy [41, 43, 44]. Biopsy of a nerve on the serosal surface of the proventriculus or ventriculus is a more sensitive, but riskier procedure [45].

At post-mortem, a complete set of tissues should be submitted for histopathology, including transverse sections of all levels of the digestive tract.

Avian bornaviruses are readily grown in cell culture [33, 46].

Clinical pathology

There are no consistent changes in clinical pathology profiles [6]. Birds with advanced PDD are typically anaemic and hypoproteinaemic.

Pathology

Typical necropsy findings in PaBV include an emaciated bird with a massively distended proventriculus and ventriculus containing ingesta. Myenteric nerves to the proventriculus and ventriculus may be grossly enlarged. Dilatation of some or all of the intestines may also occur, however dilatation of the gastrointestinal tract is not always present.

Microscopic lesions include a non-suppurative encephalomyelitis, enlargement and lymphoplasmacytic infiltration of the myenteric nerves of ventriculus, proventriculus, crop and intestines in decreasing order of frequency with an associated lymphoplasmacytic infiltration. Similar lesions are found in peripheral nerves [47]. Inflammation of the nerves of the heart and lymphoplasmacytic infiltration of the adrenals occurs infrequently. ABVs can be identified in brain sections and many other tissues, using immunohistochemical testing [36, 48-50].

Canaries infected with canary bornaviruses can show PDD-like pathologic lesions [3]. **Canada geese** infected with ABBV-1 had gross and histopathologic findings lesions similar to those of PDD [12].

Differential diagnoses

Many chronic diseases can resemble PDD, including bacterial and fungal infections of the gut, cancer, heavy metal intoxication, vitamin E, thiamine, and vitamin A deficiencies and intestinal obstructions. Microscopically, paramyxovirus 1, 2 or 3, and arboviruses, including West Nile virus can produce brain lesions similar to those caused by ABV [40, 41].

Treatment

Treatments are generally limited to symptomatic measures as there are no options to cure a bird of infection. Nonsteroidal anti-inflammatory drugs have been used to reverse the clinical signs and microscopic lesions in birds with PDD [51]. There is anecdotal evidence that the antiviral drug amantadine hydrochloride may also contribute to the recovery of birds with PDD [41].

Prevention and control

Effective quarantine protocols and separation of infected and uninfected birds remain the only known methods for preventing the horizontal spread of bornavirus infection [1]. Preventing the spread of PaBV infection in captive birds requires all birds entering a collection to be repeatedly tested by PCR and serology. As infected birds may be negative on both assays, introduction of positive birds into a collection may still occur. It is recommended to have at least two sequential negative results before the individual bird can be presumed negative [6].

Collection of eggs for artificial incubation and hand-raising chicks have been proposed as components of conservation programs [52].

There is experimental development of vaccines however they are not available in practice [53, 54].

The ability of ABV to survive in the environment is not known. The viruses are assumed to be susceptible to commonly used disinfectants including chlorhexidine, phenolics, quaternary ammonium products and bleach [40].

Research

There are many knowledge gaps associated with avian bornaviruses, including

- the extent that PaBV has spread in aviculture in Australia
- the potential presence and impact of PaBV in Australian wild parrots
- the significance of seropositive detections in wild birds
- the potential risk of PaBV and other ABV to Australian native bird species
- the routes of transmission and the factors influencing infection, development and progression of disease
- the pathogenic significance of canary and aquatic bird bornaviruses.

Further investigation is also necessary to develop reliable and commercially viable serologic tests with wide applicability [6].

Surveillance and management

Parrot bornaviruses are listed on National Priority List of Exotic Environmental Pests, Weeds and Diseases, however there are no formal surveillance programs in place in Australia for ABV. Cases of ABV in wildlife would fit within the general surveillance category of "Interesting or unusual cases" and should therefore be captured by as part of Australia's general wildlife surveillance system.

WHA is interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. Negative data are also valuable. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia administers Australia's general wildlife health surveillance system, in partnership with government and non-government agencies. Wildlife health data is collected into a national database, the electronic Wildlife Health Information System (eWHIS). Information is reported by a variety of sources including government agencies, zoo based wildlife hospitals, sentinel veterinary clinics, universities, wildlife rehabilitators, and a range of other organisations and individuals. Targeted surveillance data is also collected by WHA. See the WHA website for more information https://wildlifehealthaustralia.com.au/Our-Work/Surveillance and https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/eWHIS-Wildlife-Health-Information-System.

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Wildlife Health Australia recognises the Traditional Custodians of Country throughout Australia. We respectfully acknowledge Aboriginal and Torres Strait Islander peoples' continuing connection to land, sea, wildlife and community. We pay our respects to them and their cultures, and to their Elders past and present.

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