Beak and feather disease virus in Australian birds

Introductory statement

Beak and feather disease virus (BFDV) is the causative agent of psittacine beak and feather disease (PBFD), an endemic disease in Australia’s wild parrot populations. Descriptions of parrots with feather loss consistent with the disease date back to the late 1800s (Ashby 1907). The virus is believed to have originated in Australia sometime following the separation of the continent from Gondwanaland, with spread to other parts of the world with modern movement of parrots as pet and aviary species. It has the potential to impact on several endangered Australian and non-Australian parrot populations and is listed as a key threatening process by the Australian government. Of late, the virus also has been identified in various non-psittacine species.

Aetiology

Beak and feather disease virus is a 14 to 16 nm non-enveloped icosahedral DNA virus belonging to the family Circoviridae. Formerly, it was believed that the circoviruses recovered from a diverse range of psittacines were all antigenically similar. Doubt was cast on this theory when a virus that appeared to be serologically and genetically different was isolated from cockatiels (Nymphicus hollandicus) (Shearer et al. 2008).

More recent research appears to indicate that psittacine circoviruses can be divided into two species and multiple viral strains. Based on work by Varsani et al. (2011), BFDV contains 14 strains, while budgerigar circovirus (BCV), a newly defined species to date only found in budgerigars (Melopsittacus undulates), contains three strains. However, it is likely that this number will continue to increase as shown by the discovery of two new distinct BFDV lineages in orange-bellied parrots (Neophema chrysogaster) (Peters et al. 2014) and further strains in budgerigars in China (Ma et al. 2019).

Natural hosts

Seemingly all members of the psittacine superfamilies Psittacoidea (true parrots) and Cacatuoidae (cockatoos) are susceptible, but to date no susceptibility has been detected in the ancient New Zealand superfamily Strigopoidea, which includes the kakapo (Strigops habroptila) and kaka (Nestor meridionalis) (Raidal et al. 2015). Various non-psittacine birds have tested positive for the virus, with associated disease in some cases including Gouldian finches (Erythura gouldiae), rainbow bee-eaters (Merops ornatus) and a powerful owl (Ninox strenua) (Circella et al. 2014; Sarker et al. 2015; Sarker et al. 2016).
World distribution

The disease is enzootic in wild Australian and other South Pacific psittacines but has been introduced to free ranging and captive psittacines throughout the world via the live bird trade (Bassami et al. 2001; Ha et al. 2007; Harkins et al. 2014; Fogell et al. 2018; González-Hein et al. 2019; Ma et al. 2019). Potential impact of the virus on survival of endangered species is recognised both in Australia and South Africa (Downs et al. 2015).

Occurrences in Australia

The disease occurs Australia wide with reports dating back to the 1880s (Ashby 1907; Raidal and Peters 2018).

Epidemiology

Variation in the manifestation of the disease appears to be the result of the age and species of the host, with little variation resulting from the strain of virus (Raidal et al. 2015). More severe disease with peracute to acute death is seen in neonate and fledgling birds and in some species, such as African grey parrots (*Psittacus erithacus erithacus*), rapid death and marked immunosuppression is a feature (Doneley 2003). The chronic form of the disease varies from subtle feather colour changes, such as in the smaller *Neophasa* species of parrots to more severe, progressive feather dystrophy and beak malformation typical of the cockatoos (Raidal and Peters 2018). Anecdotally subclinical infections are recognised in rainbow and scaly-breasted lorikeets (*Trichoglossus moluccanus* and *T. chlorolepidotus*) but in other cases these species demonstrate susceptibility (Raidal et al. 2015). Recent work indicates that chronically affected crimson rosellas (*Platycercus elegans*) can clear the infection following a viraemic period of several months (Martens et al. 2019).

Large amounts of virus are found in feather dust and faeces, resulting in opportunities for direct and indirect transmission (Ritchie et al. 1991; Raidal et al. 2015). Extensive environmental contamination with this very stable organism particularly promotes indirect transmission through competition for nesting hollows, (Raidal and Peters 2018). Shedding of the virus in crop epithelium facilitates transfer from adults to chicks during feeding (Ritchie et al. 1991) and vertical transmission through eggs can also occur (Rahaus et al. 2008). However, a large scale study of wild breeding crimson rosellas did not find a correlation between parental and offspring infection status, indicating parental to offspring transmission may be less important than expected (Eastwood et al. 2019).

Transmission to non-psittacine species is still unclear. Competition for nesting hollows occurs and can explain transmission for some but not all species. Predatory birds may become infected when preying or scavenging on infected psittacines (Raidal and Peters 2018). BFDV has been identified in the gut content of *Knemidocoptes* mites found on a BFDV-infected sulphur-crested cockatoo (*Cacatua galerita*), raising the possibility of insects acting as vectors (Portas et al. 2017).

Clinical signs

Peracute disease can be seen in neonates and juveniles of certain species, and has been best described in African grey parrots. These birds present fluffed, lethargic, anorexic and weak with crop stasis and vomiting followed by death (Schoemaker et al. 2000; Doneley 2003). Feather changes are not a feature of the disease in these species (Schoemaker et al. 2000).
Acute disease of other psittacine species, especially cockatoos, is usually seen in young or fledgling birds during their first feather formation. It is characterised by depression, diarrhoea and crop stasis, with feather abnormalities appearing in 1-2 days and death in 1-2 weeks (Doneley 2003).

Chronic PBFD usually occurs in psittacine birds aged six to 12 months undergoing their first adult moult but can also be seen in older individuals (Raidal et al. 2015). The result is progressive appearance of abnormally developed feathers during each successive moult. Changes include retention of feather sheaths, haemorrhage within the pulp, fractures of the rachis, deformed curled feathers and constrictions at the base of the feathers (Pass and Perry 1984; Gill 2001). In older birds one of the first signs is a loss of powder down and white birds will appear dirty. Beaks and feet can appear shiny due to the lack of powder. Variation in presentation includes lorikeets, which often only lose primary flight and tail feathers, and other species in which feathers exhibit a colour change (green to yellow and blue to white) (Gill 2001). Beak changes may also occur, particularly in cockatoos. These include elongation, fractures, palatine necrosis and oral ulceration (McOrist et al. 1984; Pass and Perry 1984). Claw abnormalities can also develop. Most affected birds eventually die as a result of impaired eating and/or secondary infections due to the immunosuppressive nature of the infection (Raidal et al. 2015).

Diagnosis

In chronic disease, a diagnosis of PBFD can often be reliably made based on clinical signs of feather dystrophy and beak deformity (Raidal et al. 2015).

Three BFDV diagnostic assays, haemagglutination (HA), haemagglutination inhibition (HI) and PCR, can be used individually or in combination to describe the BFDV infection status of an individual bird and to aid in determining the epidemiology of BFDV in a flock. Each of these tests, when used on specific tissue samples, provides information that can inform the disease course, prognosis and history of exposure in that individual. HA on feather material is a sensitive and highly specific indicator of viral shedding in an infected bird. Because it is not an amplification procedure (unlike PCR) it is not susceptible to environmental contamination with BFDV. HI on blood measures BFDV-directed antibodies and thus is an indicator of both previous exposure and the relative magnitude of the humoral immune response to BFDV infection. PCR on blood is highly sensitive and specific for BFDV viraemia and indicates current or very recent infection with BFDV. Birds that recover from BFDV infection will typically mount a strong antibody response (i.e. high HI titres) and occasionally transient low level viral shedding (i.e. no to low HA titre). Birds that exhibit latent infection will typically exhibit a waxing and waning viraemia (by PCR) with a waxing and waning low level antibody response and intermittent viral shedding. Birds that succumb to PBFD will typically have persistent viraemia (by PCR) with no antibody response and high levels of shedding (A Peters and S Raidal, pers comm Mar 2020).

Table 1 compares currently available testing modalities. Biopsies of feathered skin can be attempted but are often not rewarding for diagnosis (Raidal et al. 2015). Highly sensitive techniques, such as PCR, may produce false positive results when applied to environmentally exposed samples (feathers and blood from toenail clippings) (A Peters, pers comm Mar 2020).
Table 1. Comparison of testing modalities for BFDV (based on (Khalesi et al. 2005; Sarker et al. 2014; Raidal et al. 2015; Chae et al. 2020).

<table>
<thead>
<tr>
<th>Test</th>
<th>Component detected</th>
<th>Sample required</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemagglutination</td>
<td>Virus</td>
<td>Feathers</td>
<td>Slightly less sensitive than PCR for detection of virus in feathers</td>
</tr>
<tr>
<td>Haemagglutination inhibition</td>
<td>Antibody</td>
<td>Serum; plasma; blood dried on filter paper</td>
<td>Gold standard for antibody detection</td>
</tr>
<tr>
<td>ELISA-based tests</td>
<td>Antibody</td>
<td>Serum</td>
<td>Validity not guaranteed due to unknown cross-reactivity of IgY between avian species</td>
</tr>
<tr>
<td>PCR</td>
<td>Viral DNA</td>
<td>Blood; cloacal swab; tissue; feathers</td>
<td>Currently the main technique for diagnosing BFDV; sequencing valuable for tracing origin of infection in a flock</td>
</tr>
<tr>
<td>PCR-HRM curve analysis</td>
<td>Viral DNA</td>
<td>Blood; cloacal swab; tissue; feathers</td>
<td>Rapid method for differentiating viral genotypes; valuable in epidemiological studies</td>
</tr>
<tr>
<td>Swarm loop-mediated isothermal amplification (sLAMP)</td>
<td>Viral DNA</td>
<td>Blood; cloacal swab; tissue; feathers</td>
<td>Recently developed test with equivalent detection to PCR but faster results</td>
</tr>
<tr>
<td>Immunohistochemistry</td>
<td>Antigen</td>
<td>Formalin fixed tissue</td>
<td>Apart from biopsy of feathered skin, requires post mortem samples; sensitivity on skin biopsies is low</td>
</tr>
<tr>
<td>In-situ hybridisation</td>
<td>Viral DNA</td>
<td>Formalin fixed tissue</td>
<td>Apart from biopsy of feathered skin, requires post mortem samples; sensitivity on skin biopsies is low</td>
</tr>
</tbody>
</table>

Clinical Pathology

Acutely affected juvenile birds, particularly African grey parrots, often present with severe leucopenia (Schoemaker et al. 2000; Doneley 2003; Raidal et al. 2015).

Chronically affected birds exhibit low serum protein, characterised by low prealbumin and gamma globulin concentrations (Jacobson et al. 1986; Raidal et al. 2015).

Pathology

In peracute to acutely affected birds, few gross changes are noted, but weight loss, hepatomegaly with necrosis and splenomegaly may be seen (Schoemaker et al. 2000; Doneley 2003). Lesions associated with secondary infections due to immunosuppression may be seen (Schoemaker et al. 2000). Histologically, inclusion bodies consistent with circovirus are found in the bursa of Fabricius with associated lymphoid atrophy. Coagulative necrosis of liver and demonstrate hyperplasia of the periarteriolar sheaths and lymphoid atrophy of spleen are seen (Raidal and Cross 1995; Schoemaker et al. 2000).

In the chronic form of the disease, gross pathology consists of feather changes, often with profound emaciation at the point of death. Histologically, epithelial cells within affected feather shafts and beak may be necrotic and there is evidence of a predominantly heterophilic perivascular infiltrate within the feather pulp (McOrist et al. 1984; Pass and Perry 1984). Necrosis and atrophy of the bursa of Fabricius and thymus is also frequently present (Raidal et al. 2015). Large intranuclear and/or intracytoplasmic basophilic inclusion bodies
occur most commonly in the bursa and pulp and epidermal layers of affected feathers but can also be found in the beak, thymus and Kupffer cells (McOrist et al. 1984; Pass and Perry 1984; Raidal et al. 2015).

Immunohistochemistry and in-situ hybridisation are most reliable when performed on bursa of Fabricius, feather follicles, spleen, oesophagus and crop (Ramis et al. 1994; Raidal et al. 2015). Due to the high prevalence of BFDV, sampling of the bursa of Fabricius from all juvenile psittacine birds for histopathology is recommended.

### Differential diagnoses

The main differential diagnoses are infection with avian polyomavirus (Gill 2001) and self or conspecific trauma i.e. feather picking. Rarely, endocrine disease such as hypothyroidism can mimic the bilaterally symmetrical loss of feathers (Raidal et al. 2015).

### Laboratory diagnostic specimens

Submit one or two blood feathers and a drop of blood on filter paper.

### Laboratory procedures

There are three main diagnostic assays available for detecting evidence of PBFD infection, but new testing modalities are arising (see Table 1). The three main assays are:

- **PCR** - can be used to detect the presence of virus in affected feathers or blood.
- The haemagglutination assay (HA) - will also detect virus in feathers and blood. It is not as sensitive as PCR but provides a quantitative result. HA titres in excess of 640 HAU/50 µl usually confirm PBFD infection.
- The haemagglutination inhibition assay (HI) - measures PBFD antibodies in the blood and is inversely related to the HA result i.e. a bird that has mounted a strong immune response will tend to have a low HA result while a bird with clinical disease will have a high HA result but a low level of circulating antibodies (Khalesi et al. 2005).

### Treatment

There is no treatment, but birds of many species, such as lorikeets (Trichoglossus sp.) and Eclectus parrots (Eclectus sp.) will make a full recovery (Raidal et al. 2015). Supportive therapy can assist in recovery. Other species, such as cockatoos (Cacatua sp.) are more susceptible and usually succumb to secondary infections.

### Prevention and control

All new birds entering an aviary should be quarantined and undergo testing using a combination of testing modalities assessing antibody production and viral presence. If the aviary is located in an environment where free-ranging species are potentially infected with the virus, measures should be put in place to prevent exposure of the captive birds (Raidal et al. 2015).

No commercially produced vaccine is available, but research indicates vaccination could be effective in preventing disease. Long-billed corellas were vaccinated and then challenged with psittacine circovirus. Only four of 97 samples taken from vaccinated birds tested positive for virus using PCR, whereas 17 of 35 samples
taken from non-vaccinated controls tested positive. Vaccinated birds did not develop feather lesions, had only transient PCR-detectable viraemia and had no evidence of persistent infection 270 days post-challenge using PCR, histopathology and immunohistochemistry. Non-vaccinated control corellas developed transient feather lesions and had PCR, HI and HA test results consistent with PBFD. They were circovirus PCR-positive for up to 41 days post-challenge (Bonne et al. 2009). This vaccination study does not appear to prevent viral replication and it is unclear whether shedding could still occur (Bonne et al. 2009; Raidal et al. 2015).

The virus is extremely stable in the environment. Incubation at 80 C for thirty minutes failed to inactivate it. The only disinfectant that has been shown to be effective is the peroxygen compound, Virkon-S, if in contact with the virus for a minimum of 10 minutes (Cross 2006).

**Surveillance and management**

BFDV is endemic in Australia’s parrots. Table 2 lists published prevalence data for Australian parrots.

**Table 2.** Prevalence of BFDV infection in free-ranging Australian parrots, by species.

<table>
<thead>
<tr>
<th>Common name</th>
<th>Scientific name</th>
<th>No. positive/ no. tested</th>
<th>Location</th>
<th>Publication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulphur-crested cockatoo</td>
<td><em>Cacatua galerita</em></td>
<td>10-20% (estimate)</td>
<td>Victoria</td>
<td>McOrist et al. (1984)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>95/135</td>
<td>Camden, NSW</td>
<td>Raidal et al. (1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15/17</td>
<td>Yeoval, NSW</td>
<td>Raidal et al. (1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12/17</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12/13</td>
<td>Victoria</td>
<td>Sutherland et al. (2019)</td>
</tr>
<tr>
<td>Little corella</td>
<td><em>Cacatua sanguinea</em></td>
<td>4/6</td>
<td>Camden, NSW</td>
<td>Raidal et al. (1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1/1</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3/3</td>
<td>Victoria</td>
<td>Sutherland et al. (2019)</td>
</tr>
<tr>
<td>Long-billed corella</td>
<td><em>Cacatua tenuirostris</em></td>
<td>10/19</td>
<td>Camden</td>
<td>Raidal et al. (1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0/1</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>17/30</td>
<td>Victoria</td>
<td>Sutherland et al. (2019)</td>
</tr>
<tr>
<td>Galah</td>
<td><em>Eolophus roseicapilla</em></td>
<td>13/23</td>
<td>Camden</td>
<td>Raidal et al. (1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>32/79</td>
<td>Yeoval, NSW</td>
<td>Raidal et al. (1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4/7</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td>Gang gang</td>
<td><em>Callocephalon fimbriatum</em></td>
<td>3/3</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td>Yellow-tailed black cockatoo</td>
<td><em>Zanda funereal</em></td>
<td>0/1</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td>Crimson rosella</td>
<td><em>Platycercus elegans</em></td>
<td>5/18</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>29/84</td>
<td>South-eastern Australia</td>
<td>Eastwood et al. (2015)</td>
</tr>
<tr>
<td>Australian king parrot</td>
<td><em>Aisterus scapularis</em></td>
<td>15/28</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td>Rainbow lorikeet</td>
<td><em>Trichoglossus moluccanus</em></td>
<td>3/5</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td>Musk lorikeet</td>
<td><em>Glossopsitta concinna</em></td>
<td>2/2</td>
<td>Healesville, Vic</td>
<td>Amery-Gale et al. (2017)</td>
</tr>
<tr>
<td>Orange-bellied parrot</td>
<td><em>Neophema chrysoaster</em></td>
<td>20/23</td>
<td>Melaleuca, Tas</td>
<td>Das et al. (2015)</td>
</tr>
</tbody>
</table>

WHA Fact sheet: Beak and feather disease virus in Australian birds | April 2020
Amery-Gale et al. (2017) investigated the prevalence of the infection in free-ranging non-psittacine birds by testing liver tissue by PCR. They found high numbers, including 5/23 tawny frogmouths (Podargus strigoides), 4/13 laughing kookaburras (Dacelo novaeguineae), 4/11 Australian magpies (Gymnorhina tibicen) and one each of sacred kingfisher (Todiramphus sanctus), southern boobook (Ninox boobook), powerful owl (Ninox strenua), barn owl (Tyto alba), Australian white ibis (Threskiornis moluccus), brown goshawk (Accipiter fasciatus) and Australian raven (Corvus coronoides). Clinical signs were not described in these birds.

PBFD is listed as a key threatening process under the Environment Protection and Biodiversity Conservation Act (1999) because of its potential effects on three endangered species: the orange-bellied parrot (Neophema chrysogaster), the Norfolk Island green parrot (Cyanoramphus novaehollandiae cookii), and the swift parrot (Lathamus discolor). A Threat Abatement Plan for Beak and Feather Disease affecting endangered psittacine species (www.environment.gov.au/resource/beak-and-feather-disease-affecting-endangered-psittacine-species; 2005), recommends targeted surveillance of PCD in psittacine populations.

### Statistics

Wildlife disease surveillance in Australia is coordinated by Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia’s States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within eWHIS is by application. See the WHA website for more information: [www.wildlifehealthaustralia.com.au/ProgramsProjects/eWHISWildlifeHealthInformationSystem.aspx#requests](http://www.wildlifehealthaustralia.com.au/ProgramsProjects/eWHISWildlifeHealthInformationSystem.aspx#requests).

There are over 700 cases in the National Wildlife Health Surveillance Database. There are cases from every state and territory and from over 30 bird species. Most cases are from native psittacines: rainbow lorikeets, sulphur-crested cockatoos, and scaly-breasted lorikeets (T. chlorolepidotus). Rare cases include diagnosis based on clinical signs, histopathology or PCR in a very small number of non-psittacine species. Data collected into eWHIS in recent years places a focus on new host species, new geographic areas and unusual presentations of the disease.

### Research

Research is required to determine the relative infectivity of the various circovirus genotypes for different host species, how the carrier state is maintained, the details of possible immunosuppression, ramifications of different viral strains for vaccination and the ecology of the disease in the wild.

Prevalence of the virus in psittacine species has been investigated in Victoria and New South Wales, but no published reports could be found for other areas of the country. Surveys in other states and ongoing studies in Vic and NSW are needed.

Although Amery-Gale et al. (2017) found viral DNA in the hepatic tissue of various non-psittacine birds, it is not known if this represents replicating virus, highlighting the need for more research into the ability of non-psittacine species to carry and disseminate the disease. Some species of *Trichoglossus* lorikeets appear to be inherently resistant to the infection and as such also require research as to their role in dissemination of the disease (Raidal et al. 2015).
Transmission between species that do not share habitat niches, such as nesting hollows, also requires further research. The hypothesis of insects as vectors requires further investigation (Amery-Gale et al. 2017).

More work needs to be done to assess the effectiveness of vaccination across a range of species and whether production could be commercially viable.

**Human health implications**

There are no known human health risk.

**Conclusions**

BFDV is a well-recognised disease of Australian psittacine birds. It occurs both in wild and captive situations. Questions around BFDV may complicate decision-making with captive breed-for-release and recovery programs for endangered Australian native parrots. As many common wild psittacine species show evidence of BFDV infection, there are community concerns around animal welfare and possible biodiversity impacts. Further research is required to address gaps in understanding of host susceptibility and impact on wild populations, as well as development of treatment and control options.

**Acknowledgements**

We are extremely grateful to Peter Holz who provided the initial draft and Chery Sangster who provided an updated version of this fact sheet and to those individuals, agencies and organisations that provided comment and external review. Production of these fact sheets would not be possible without their ongoing support.

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**References and other information**


To provide feedback on this fact sheet

We encourage those with laboratory confirmed cases of this condition in native Australian or feral animals to submit this information to the national system for consideration for inclusion in the national database. Please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

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