

Beak and feather disease virus in Australian birds

Fact Sheet

April 2020

Key points

- Beak and feather disease virus (BFDV) is the causative agent of psittacine beak and feather disease (PBFD), which occurs in both captive and wild birds.
- Disease may be acute or chronic and may vary in severity; most birds eventually die from infection.
- The virus is endemic in many wild parrot species in Australia and has the potential to impact several endangered parrot populations. It is listed as a key threatening process by the Australian government.
- The virus is believed to have originated in Australia many millennia ago; spread to other parts of the world occurred with modern movement of pet and aviary parrot species.
- More recently, the virus has been identified in various non-psittacine species.

Aetiology

Beak and feather disease virus (BFDV) is a non-enveloped DNA virus belonging to the family *Circoviridae*. Psittacine circoviruses are divided into two species and multiple viral strains. Beak and feather disease virus has at least 14 strains. Budgerigar circovirus (BCV), a newly defined species found so far only in budgerigars (*Melopsittacus undulates*), contains three strains ^[1]. It is likely that these numbers will change as further research is undertaken ^[2, 3].

One Health implications

Wildlife and the environment: BFDV is listed as a key threatening process and may impact endangered bird populations in Australia. Presence of the virus may complicate decision-making with captive breed-for-release and recovery programs. There are concerns around the welfare impact of the disease on affected wild (and captive) birds.

Domestic animals and humans: there are no known human or domestic animal health risks.

Natural hosts

All members of the psittacine superfamilies *Psittacoidea* (true parrots) and *Cacatuoidea* (cockatoos) are considered susceptible ^[4]. Various non-psittacine birds have tested positive for the virus, in

some cases with associated disease, including Gouldian finches (*Erythrura gouldiae*), rainbow bee-eaters (*Merops ornatus*) and a powerful owl (*Ninox strenua*)^[5-7].

BFDV is endemic in Australia's parrots. Table 1 lists published prevalence data for Australian parrots.

Table 1. Prevalence of BFDV infection in free-ranging Australian parrots, by species

Common name	Scientific name	No. positive/ no. tested	Location	Publication
Sulphur-crested cockatoo	<i>Cacatua galerita</i>	10-20% (estimate)	Victoria	McOrist et al. 1984 [8]
		95/135	Camden, NSW	Raidal et al. 1993 [9]
		15/17	Yeoval, NSW	Raidal et al. 1993 [9]
		12/17	Healesville, Vic	Amery-Gale et al. 2017 [10]
		12/13	Victoria	Sutherland et al. 2019 [11]
Little corella	<i>C. sanguinea</i>	4/6	Camden, NSW	Raidal et al. 1993 [9]
		1/1	Healesville, Vic	Amery-Gale et al. 2017 [10]
		3/3	Victoria	Sutherland et al. 2019 [11]
Long-billed corella	<i>C. tenuirostris</i>	10/19	Camden	Raidal et al. 1993 [9]
		0/1	Healesville, Vic	Amery-Gale et al. 2017 [10]
		17/30	Victoria	Sutherland et al. 2019 [11]
Galah	<i>Eolophus roseicapilla</i>	13/23	Camden	Raidal et al. 1993 [9]
		32/79	Yeoval, NSW	Raidal et al. 1993 [9]
		4/7	Healesville, Vic	Amery-Gale et al. 2017 [10]
Gang gang	<i>Callocephalon fimbriatum</i>	3/3	Healesville, Vic	Amery-Gale et al. 2017 [10]
Yellow-tailed black cockatoo	<i>Zanda funereal</i>	0/1	Healesville, Vic	Amery-Gale et al. 2017 [10]
Crimson rosella	<i>Platycercus elegans</i>	5/18	Healesville, Vic	Amery-Gale et al. 2017 [10]
		29/84	South-eastern Australia	Eastwood et al. 2015 [12]
Eastern rosella	<i>P. eximius</i>	6/11	Healesville, Vic	Amery-Gale et al. 2017 [10]
Australian king parrot	<i>Alisterus scapularis</i>	15/28	Healesville, Vic	Amery-Gale et al. 2017 [10]
Rainbow lorikeet	<i>Trichoglossus moluccanus</i>	3/5	Healesville, Vic	Amery-Gale et al. 2017 [10]
Musk lorikeet	<i>Glossopsitta concinna</i>	2/2	Healesville, Vic	Amery-Gale et al. 2017 [10]
Orange-bellied parrot	<i>Neophema chrysogaster</i>	20/23	Melaleuca, Tas	Das et al. 2015 [13]

Prevalence of infection in liver of free-ranging non-psittacine birds by PCR, showed high levels, 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 ^[10].

World distribution and occurrences in Australia

The disease is endemic in wild Australian and other South Pacific psittacines and occurs Australia-wide, with reports dating back to the 1880s ^[14, 15]. It has been introduced to free-ranging and captive psittacines throughout the world via the live bird trade ^[3, 16-20]. The virus may impact the survival of endangered in both Australia and South Africa ^[15, 21].

Epidemiology

The disease can present in acute or chronic forms, with varying severity and presentation of disease. Severity appears to be influenced primarily by the age and species of the host, with little variation resulting from the strain of virus ^[4]. More severe disease with peracute to acute death is seen in neonate and fledgling birds. In some species, such as African grey parrots (*Psittacus erithacus erithacus*), rapid death and marked immunosuppression is a feature ^[22]. The chronic form of the disease can vary from subtle feather colour changes, such as in the smaller *Neophema* species of parrots to more severe, progressive feather dystrophy and beak malformation typical in cockatoos ^[15]. Both clinical and subclinical infections are recognised in rainbow and scaly-breasted lorikeets (*Trichoglossus moluccanus* and *T. chlorolepidotus*) ^[4]. Chronically affected crimson rosellas (*Platycercus elegans*) have been shown to clear the infection following a viraemic period of several months ^[23], but this is considered a rare outcome.

Large amounts of virus are found in feather dust and faeces, resulting in opportunities for direct and indirect transmission ^[4, 24]. The organism is very stable, and extensive environmental contamination can promote indirect transmission e.g. when wild birds compete for nesting hollows ^[15]. The virus is shed in the crop which allows for transfer from adults to chicks during feeding ^[24]. Vertical transmission via the egg can occur ^[25]. Despite this, parental to offspring transmission may be less important than expected ^[26].

Methods of transmission to non-psittacine species is not known. Competition for nesting hollows may explain transmission for some, but not all, species. Predatory birds may become infected when preying or scavenging on infected psittacines ^[15]. Beak and feather disease virus has been identified in the gut of mites found on a BFDV-infected sulphur-crested cockatoo (*Cacatua galerita*), raising the possibility of insects acting as vectors ^[27].

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 ^[22, 28]. Feather changes are not a feature of the disease in these species ^[28].

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 ^[22].

Chronic BFD usually occurs in psittacine birds aged six to 12 months undergoing their first adult moult, but can also be seen in older individuals ^[4]. Abnormally developed feathers grow progressively worse during each successive moult. Changes include retention of feather sheaths, haemorrhage within the feather pulp, fractures of the feather, deformed curled feathers and constrictions at the base of the feathers ^[29, 30]. Birds may become emaciated to the point of death. In older birds one of the first signs is a loss of powder down and white birds such as sulphur-crested cockatoos will appear dirty. Beaks and feet can appear shiny due to the lack of powder. Lorikeets often only lose primary flight and tail feathers, and in some other species, feathers exhibit a colour change (green to yellow and blue to white) ^[30].

Beak changes may occur, particularly in cockatoos. These include elongation, fractures, palatine necrosis and oral ulceration ^[8, 29]. Claw abnormalities can develop.

Most affected birds eventually die as a result of impaired eating and/or secondary infections due to the immunosuppressive nature of the infection ^[4].

Diagnosis

In chronic disease, a diagnosis of PBFD can often be reliably made based on clinical signs of feather dystrophy and beak deformity ^[4].

Three main diagnostic assays are available for detecting evidence of PBFD infection (see Table 1). One or two blood feathers and a drop of blood on filter paper should be collected for testing. Tests 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:

1. **PCR** can be used to detect the presence of **virus** in affected feathers or blood.
2. 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.
3. 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 ^[31].

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. Haemagglutination 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. **Haemagglutination inhibition** 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 2 compares available tests. Biopsy of feathered skin are often not rewarding for diagnosis ^[4]. Highly sensitive techniques, such as PCR, may produce false positive results when applied to environmentally exposed samples (e.g. feathers and blood from toenail clippings) (A Peters, pers comm Mar 2020).

Table 2. Comparison of testing modalities for BFDV (based on Raidal et al. 2015 [4], Khalesi et al. 2005 [31], Sarker et al. 2014 [32], Chae et al. 2020 [33]).

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

Clinical pathology

Acutely affected juvenile birds, particularly African grey parrots, often present with severe leucopenia ^[4, 22, 28]. Chronically affected birds exhibit low serum protein, characterised by low prealbumin and gamma globulin concentrations ^[4, 34].

Pathology

In the **peracute to acute form**, few gross changes are noted, but weight loss, hepatomegaly with necrosis and splenomegaly may be seen ^[22, 28]. Secondary infections due to immunosuppression occur ^[28]. 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 ^[28, 35].

In the **chronic form** of the disease, gross pathology consists of feather changes, often with profound emaciation to 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 ^[8, 29]. Necrosis and atrophy of the bursa of Fabricius and thymus are frequently present ^[4]. 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 ^[4, 8, 29].

Immunohistochemistry and in-situ hybridisation are most reliable when performed on bursa of Fabricius, feather follicles, spleen, oesophagus and crop ^[4, 36]. 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 ^[30] and self or conspecific trauma i.e. feather picking. Rarely, endocrine disease such as hypothyroidism can mimic the bilaterally symmetrical loss of feathers ^[4].

Treatment

There are no treatment options for BFDV-infected birds. Most affected birds [including species such as cockatoos (*Cacatua* sp.)] eventually die as a result of impaired eating and/or secondary infections due to the immunosuppressive nature of the infection ^[4]. Euthanasia of clinically affected wild birds is often the most appropriate decision due to welfare concerns, the terminal nature of the disease, and the biosecurity risk these individuals pose to other birds.

Captive individuals of certain species, such as lorikeets (*Trichoglossus* sp.) and Eclectus parrots (*Eclectus* sp.), may be able make a clinical recovery if provided with appropriate supportive care, however they may still be able to infect other birds with the virus ^[37].

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 ^[4].

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 BFDV. They were circovirus PCR-positive for up to 41 days post-challenge ^[38]. This vaccination study does not appear to prevent viral replication and it is unclear whether shedding could still occur ^[4, 38].

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 ^[39].

Research

Further research is required to address gaps in understanding of prevalence, host susceptibility and impact on wild populations, as well as development of treatment and control options. 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.

There is a need for more research into the ability of non-psittacine species to carry and disseminate the disease ^[10]. Some species of *Trichoglossus* lorikeets appear to be inherently resistant to the infection and also require research as to their role in dissemination of the disease ^[4].

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 ^[10].

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

Surveillance and management

Psittacine beak and feather disease virus 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 novaezelandiae 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 BFDV in psittacine populations.

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>.

There are over 700 cases in the National Wildlife Health Surveillance Database from over 30 bird species. Most cases are from native psittacines: rainbow lorikeets, sulphur-crested cockatoos, and scaly-breasted lorikeets (*T. chlorolepidotus*). Data collected into eWHIS in recent years places a focus on new host species, new geographic areas and unusual presentations of the disease.

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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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