Beak and feather disease virus in Australian birds

Fact sheet

April 2020

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 [1]. 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*) [2].

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 [3], 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*) [4] and further strains in budgerigars in China [5].

Natural hosts

Seemingly all members of the psittacine superfamilies Psittacoidea (true parrots) and Cacatuoidea (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*) [6]. Various non-psittacine birds have tested positive for the virus, with associated disease in some cases including Gouldian finches (*Erythrura gouldiae*), rainbow bee-eaters (*Merops ornatus*) and a powerful owl (*Ninox strenua*) [7-9].

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 [5, 10-14]. Potential impact of the virus on survival of endangered species is recognised both in Australia and South Africa [15].

Occurrences in Australia

The disease occurs Australia wide with reports dating back to the 1880s [1, 16].

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 [6]. 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 [17]. The chronic form of the disease varies from subtle feather colour changes, such as in the smaller *Neophema* species of parrots to more severe, progressive feather dystrophy and beak malformation typical of the cockatoos [16]. Anecdotally subclinical infections are recognised in rainbow and scaly-breasted lorikeets (*Trichoglossus moluccanus* and *T. chlorolepidotus*) but in other cases these species demonstrate susceptibility [6]. Recent work indicates that chronically affected crimson rosellas (*Platycercus elegans*) can clear the infection following a viraemic period of several months [18].

Large amounts of virus are found in feather dust and faeces, resulting in opportunities for direct and indirect transmission [6, 19]. Extensive environmental contamination with this very stable organism particularly promotes indirect transmission through competition for nesting hollows, [16]. Shedding of the virus in crop epithelium facilitates transfer from adults to chicks during feeding [19] and vertical transmission through eggs can also occur [20]. 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 [21].

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 [16]. 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 [22].

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 [17, 23]. Feather changes are not a feature of the disease in these species [23].

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 [17].

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 [6]. 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 [24, 25]. 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) [25]. Beak changes may also occur, particularly in cockatoos. These include elongation, fractures, palatine necrosis and oral ulceration [24, 26]. 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 [6].

Diagnosis

In chronic disease, a diagnosis of PBFD can often be reliably made based on clinical signs of feather dystrophy and beak deformity [6].

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 [6]. 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 Raidal et al. 2015 [6], Khalesi et al. 2005 [27], Sarker et al. 2014 [28], Chae et al. 2020 [29].

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 leucopoenia [6, 17, 23].

Chronically affected birds exhibit low serum protein, characterised by low prealbumin and gamma globulin concentrations [6, 30].

Pathology

In peracute to acutely affected birds, few gross changes are noted, but weight loss, hepatomegaly with necrosis and splenomegaly may be seen [17, 23]. Lesions associated with secondary infections due to immunosuppression may be seen [23]. 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 [23, 31].

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 [24, 26]. Necrosis and atrophy of the bursa of Fabricius and thymus is also frequently present [6]. 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 [6, 24, 26].

Immunohistochemistry and in-situ hybridisation are most reliable when performed on bursa of Fabricius, feather follicles, spleen, oesophagus and crop [6, 32]. 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 [25] and self or conspecific trauma i.e. feather picking. Rarely, endocrine disease such as hypothyroidism can mimic the bilaterally symmetrical loss of feathers [6].

Laboratory diagnostic specimens and procedures

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

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 [27].

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 [6]. 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 [6].

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 [33]. This vaccination study does not appear to prevent viral replication and it is unclear whether shedding could still occur [6, 33].

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 [34].

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

Common name	Scientific name	No. positive/ no. tested	Location	Publication
Sulphur-crested cockatoo	*Cacatua galerita*	10-20% (estimate)	Victoria	McOrist et al. 1984 [26]
		95/135	Camden, NSW	Raidal et al. 1993 [35]
		15/17	Yeoval, NSW	Raidal et al. 1993 [35]
		12/17	Healesville, Vic	Amery-Gale et al. 2017 [36]
		12/13	Victoria	Sutherland et al. 2019 [37]
Little corella	*Cacatua sanguinea*	4/6	Camden, NSW	Raidal et al. 1993 [35]
		1/1	Healesville, Vic	Amery-Gale et al. 2017 [36]
		3/3	Victoria	Sutherland et al. 2019 [37]
Long-billed corella	*Cacatua tenuirostris*	10/19	Camden	Raidal et al. 1993 [35]
		0/1	Healesville, Vic	Amery-Gale et al. 2017 [36]
		17/30	Victoria	Sutherland et al. 2019 [37]
Galah	*Eolophus roseicapilla*	13/23	Camden	Raidal et al. 1993 [35]
		32/79	Yeoval, NSW	Raidal et al. 1993 [35]
		4/7	Healesville, Vic	Amery-Gale et al. 2017 [36]

Common name	Scientific name	No. positive/ no. tested	Location	Publication
Gang gang	*Callocephalon fimbriatum*	3/3	Healesville, Vic	Amery-Gale et al. 2017 [36]
Yellow-tailed black cockatoo	*Zanda funereal*	0/1	Healesville, Vic	Amery-Gale et al. 2017 [36]
Crimson rosella	*Platycercus elegans*	5/18	Healesville, Vic	Amery-Gale et al. 2017 [36]
		29/84	South-eastern Australia	Eastwood et al. 2015 [38]
Eastern rosella	*Platycercus eximius*	6/11	Healesville, Vic	Amery-Gale et al. 2017 [36]
Australian king parrot	*Alisterus scapularis*	15/28	Healesville, Vic	Amery-Gale et al. 2017 [36]
Rainbow lorikeet	*Trichoglossus moluccanus*	3/5	Healesville, Vic	Amery-Gale et al. 2017 [36]
Musk lorikeet	*Glossopsitta concinna*	2/2	Healesville, Vic	Amery-Gale et al. 2017 [36]
Orange-bellied parrot	*Neophema chysogaster*	20/23	Melaleuca, Tas	Das et al. 2015 [39]

Amery-Gale et al. 2017 [36] 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 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 PCD in psittacine populations.

Statistics

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. 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 [36] 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 [6].

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 [36].

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.

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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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References and other information

1. Ashby E (1907) Parrakeets Moulting. *Emu*, **6**(4): 193-194
2. Shearer PL, Bonne N et al. (2008) Beak and feather disease virus infection in cockatiels (*Nymphicus hollandicus*). *Avian Pathology*, **37**(1): 75-81
3. Varsani A, Regnard GL et al. (2011) Global genetic diversity and geographical and host-species distribution of beak and feather disease virus isolates. *Journal of General Virology*, **92**(Pt 4): 752-67
4. Peters A, Patterson EI et al. (2014) Evidence of psittacine beak and feather disease virus spillover into wild critically endangered orange-bellied parrots (*Neophema chrysogaster*) *Journal of Wildlife Diseases*, **50**(2): 288-296
5. Ma J, Tian Y et al. (2019) Identification and characterization of novel genotypes of psittacine beak and feather disease virus from budgerigar in China. *Transboundary and Emerging Diseases*, **66**(5): 1827-1833
6. Raidal S, Sarker S et al. (2015) Review of psittacine beak and feather disease and its effect on Australian endangered species. *Australian Veterinary Journal*, **93**(12): 466-470
7. Circella E, Legretto M et al. (2014) Psittacine Beak and Feather Disease–like Illness in Gouldian Finches (Chloebia gouldiae). *Avian Diseases*, **58**(3): 482-487
8. Sarker S, Moylan KG et al. (2015) Evidence of a deep viral host switch event with beak and feather disease virus infection in rainbow bee-eaters (Merops ornatus). *Scientific Reports*, **5**: 14511
9. Sarker S, Lloyd C et al. (2016) Forensic genetic evidence of beak and feather disease virus infection in a Powerful Owl, *Ninox strenua. Emu*, **116**(1): 71-74
10. Harkins GW, Martin DP et al. (2014) Towards inferring the global movement of beak and feather disease virus. *Virology*, **450-451**: 24-33
11. Fogell DJ, Martin RO et al. (2018) Trade and conservation implications of new beak and feather disease virus detection in native and introduced parrots. *Conservation Biology*, **32**(6): 1325-1335
12. Bassami MR, Ypelaar I et al. (2001) Genetic diversity of beak and feather disease virus detected in psittacine species in Australia. *Virology*, **279**(2): 392-400
13. Ha H, Anderson I et al. (2007) The prevalence of beak and feather disease virus infection in wild populations of parrots and cockatoos in New Zealand. *New Zealand Veterinary Journal*, **55**(5): 235-238
14. González-Hein G, Gil IA et al. (2019) Prevalence of Aves Polyomavirus 1 and Beak and Feather Disease Virus From Exotic Captive Psittacine Birds in Chile. *Journal of Avian Medicine and Surgery*, **33**(2): 141-149

15. Downs CT, Brown M et al. (2015) Review of documented beak and feather disease virus cases in wild Cape parrots in South Africa during the last 20 years. *Journal of Ornithology*, **156**(4): 867-875

16. Raidal SR and Peters A (2018) Psittacine beak and feather disease: ecology and implications for conservation. *Emu-Austral Ornithology*, **118**(1): 80-93

17. Doneley RJT (2003) Acute Beak and Feather Disease in juvenile African Grey parrots - an uncommon presentation of a common disease. *Australian Veterinary Journal* **81**(4): 206-207

18. Martens JM, Stokes HS et al. (2019) Persistence of beak and feather disease virus (BFDV) infection in wild crimson rosellas (*Platycercus elegans*). *Emu-Austral Ornithology*, **119**(4): 402-406

19. Ritchie B, Niagro F et al. (1991) Routes and prevalence of shedding of psittacine beak and feather disease virus. *American Journal of Veterinary Research*, **52**(11): 1804-1809

20. Rahaus M, Desloges N et al. (2008) Detection of beak and feather disease virus DNA in embryonated eggs of psittacine birds. *Veterinarni Medicina-Praha*, **53**(1): 53

21. Eastwood JR, Berg ML et al. (2019) Prevalence of BFDV in wild breeding *Platycercus elegans. Journal of Ornithology*, **160**(2): 557-565

22. Portas T, Jackson B et al. (2017) Beak and feather disease virus carriage by *Knemidocoptes pilae* in a sulphur-crested cockatoo (*Cacatua galerita*). *Australian Veterinary Journal*, **95**(12): 486-489

23. Schoemaker NJ, Dorrestein GM et al. (2000) Severe leukopenia and liver necrosis in young African Grey Parrots (*Psittacus erithacus erithacus*) infected with psittacine circovirus. *Avian Diseases*, **44**(2): 470-478

24. Pass DA and Perry RA (1984) The pathology of psittacine beak and feather disease. *Australian Veterinary Journal*, **61**(3): 69-74

25. Gill JH (2001) Avian skin diseases. *Veterinary Clinics of North America: Exotic Animal Practice*, **4**(2): 463-492

26. McOrist S, Black DG et al. (1984) Beak and feather dystrophy in wild sulphur-crested cockatoos (Cacatua galerita). *Journal of Wildlife Diseases*, **20**(2): 120-124

27. Khalesi B, Bonne N et al. (2005) A comparison of haemagglutination, haemagglutination inhibition and PCR for the detection of psittacine beak and feather disease virus infection and a comparison of isolates obtained from loriids. *Journal of General Virology* **86** (11): 3039-3046

28. Sarker S, Ghorashi S et al. (2014) Rapid genotyping of *beak and feather disease virus* using high-resolution DNA melt curve analysis. *Journal of Virological Methods*, **208**: 47-55

29. Chae H-G, Lim D-R et al. (2020) An advanced loop-mediated isothermal amplification assay for the rapid detection of beak and feather disease virus in psittacine birds. *Journal of Virological Methods*: 113819

30. Jacobson ER, Clubb S et al. (1986) Feather and beak dystrophy and necrosis in cockatoos: clinicopathologic evaluations. *Journal of the American Veterinary Medical Association*, **189**: 999-1005

31. Raidal SR and Cross GMD- (1995) Acute necrotizing hepatitis caused by experimental infection with psittacine beak and feather disease virus. *Journal of Avian Medicine and Surgery*, **9**(1): 36-40

32. Ramis A, Latimer K et al. (1994) Diagnosis of psittacine beak and feather disease (PBFD) viral infection, avian polyomavirus infection, adenovirus infection and herpesvirus infection in psittacine tissues using DNA in situ hybridization. *Avian Pathology*, **23**(4): 643-657

33. Bonne N, Shearer P et al. (2009) Assessment of recombinant beak and feather disease virus capsid protein as a vaccine for psittacine beak and feather disease. *Journal of General Virology* **90** (3): 640-647

34. Cross G (2006) The effectiveness of disinfectants used on viruses closely related to BFDV. In 'Hygiene protocols for the prevention and control of diseases (particularly beak and feather disease) in Australian birds.' (Ed Australian Government). (Australian Government,: Canberra)

35. Raidal SR, McElnea CL et al. (1993) Seroprevalence of psittacine beak and feather disease in wild psittacine birds in New South Wales. *Australian Veterinary Journal*, **70**(4): 137-9

36. Amery-Gale J, Marenda MS et al. (2017) A high prevalence of beak and feather disease virus in non-psittacine Australian birds. *Journal of Medical Microbiology*, **66**(7): 1005-1013

37. Sutherland M, Sarker S et al. (2019) Disease surveillance in wild Victorian cacatuids reveals co-infection with multiple agents and detection of novel avian viruses. *Veterinary Microbiology*, **235**: 257-264

38. Eastwood JR, Berg ML et al. (2015) Prevalence of beak and feather disease virus in wild *Platycercus elegans*: comparison of three tissue types using a probe-based real-time qPCR test. *Australian Journal of Zoology*, **63**(1): 1-8

39. Das S, Sarker S et al. (2015) Psittacine beak and feather disease virus in wild orange-bellied parrots, in Association of Avian Veterinarians Australasian annual conference: Sydney. 18-27

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