

Psittacine beak and feather disease (PBFD)

Fact sheet

Introductory statement

Psittacine beak and feather disease (PBFD) is endemic in Australia's wild parrot populations. It has the potential to impact on several endangered Australian parrot populations and is listed as a key threatening process by the Australian government (below).

Aetiology

Psittacine beak and feather disease is caused by 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. Beak and feather disease virus (BFDV) contains 14 strains, while budgerigar circovirus (BCV), which has only been found in budgerigars (*Melopsittacus undulatus*), contains three strains (Varsani et al 2001).

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).

Natural hosts

All psittacines are susceptible.

World distribution

The disease is enzootic in wild South Pacific psittacines but has been introduced to free ranging and captive psittacines throughout the world via the live bird trade (Macwhirter 2000).

Occurrences in Australia

The disease occurs Australia wide.

Epidemiology

Three forms of PBFD occur:

- Peracute disease occurs in neonates.
- Acute disease is usually seen in young or fledgling birds during their first feather formation and causes death in one to two weeks.
- Chronic PBFD usually occurs in birds aged six to 12 months undergoing their first adult moult but can also be seen in older individuals. Death generally occurs six months to two years after the onset of clinical signs due to the immunosuppressive nature of the infection. Incubation period can be as short as three weeks or as long as twelve months.

Virus is found in feather dust, faeces and crop epithelium facilitating transfer from adults to chicks during feeding. Vertical transmission can also occur.

Some birds, especially rainbow lorikeets (*Trichoglossus haematodus*), can be latent carriers shedding virus while appearing clinically normal (Gerlach 1994, Macwhirter 2000, Raidal 2008).

Clinical signs

Birds suffering the peracute form show signs of septicaemia, pneumonia, enteritis, rapid weight loss and death.

Acute disease is characterised by depression, diarrhoea, crop stasis, feather abnormalities and death.

Chronic PBFD results in the 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. In older birds one of the first symptoms is a loss of powder down. Beak changes may also occur, particularly in cockatoos. These include elongation, fractures, palatine necrosis and oral ulceration (Gerlach 1994, Macwhirter 2000).

Diagnosis

A diagnosis of PBFD is made by a combination of clinical signs and antigen/antibody testing.

Pathology

Epithelial cells within affected feather shafts may be necrotic and there is evidence of a predominantly heterophilic perivascular infiltrate within the feather pulp. Necrosis and atrophy of the Bursa of Fabricius is also frequently present. Large intranuclear and/or intracytoplasmic inclusion bodies occur most commonly in the bursa and affected feathers but can also be found in the beak, gastrointestinal tract, tongue, parathyroid, bone marrow, Kupffer cells, spleen and thyroid.

Differential diagnoses

The main differential diagnosis is infection with avian polyomavirus or self-trauma i.e. feather picking.

Laboratory diagnostic specimens

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

Laboratory procedures

There are three diagnostic assays available for detecting evidence of PBFD infection:

- 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, Raidal 2008).

Treatment

There is no treatment and birds usually succumb to secondary infections.

Prevention and control

All new birds should be tested using the HA and HI tests as a minimum:

- If both tests are negative then the bird has never been exposed to the virus.
- If the HA test is positive and the HI negative or low the bird has an active infection. If the bird has clinical signs it should be euthanased but if it appears clinically normal it should be retested as some birds will clear the infection. In this case the repeat test should show a negative HA result and a high HI result. If the bird is still HA positive the HI result will likely remain low and the bird may either develop clinical disease at some point in the future or be a latent carrier. In either case it should be euthanased.
- If the HA test is negative but the HI positive then the bird has been exposed to the virus at some time in the past but has cleared the infection.

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 it contacts the virus for a minimum of 10 minutes (Cross 2006).

Surveillance and management

While PBFD is endemic in Australia's parrots little work has been done to determine its prevalence in wild parrot populations. One study of wild sulphur-crested cockatoos (*Cacatua galerita*) estimated a prevalence of 10 to 20% (McOrist et al 1984). A second study found 95 out of 135 sulphur-crested cockatoos, 13 out of 23 galahs (*Eolophus roseicapilla*), 4 out of 6 short-billed corellas (*Cacatua sanguinea*) and 10 out of 19 long-billed corellas (*Cacatua tenuirostris*) at Camden, and 15 out of 17 sulphur-crested cockatoos and 32 out of 79 galahs at Yeoval to be seropositive for PCD (Raidal et al 1993).

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*). This has resulted in the publication in 2005 of a Threat Abatement Plan for Beak and Feather Disease affecting endangered psittacine species, which recommends targeted surveillance of PCD in psittacine populations. The plan is located at <http://www.environment.gov.au/resource/beak-and-feather-disease-affecting-endangered-psittacine-species>.

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 the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au.

Most cases reported in the National Wildlife Health Surveillance Database are from rainbow lorikeets sulphur-crested cockatoos, and scaly-breasted lorikeets (*Trichoglossus chlorolepidotus*) from the eastern states. Other cases include galahs, an eastern rosella (*Platycercus eximius*), Australian king parrot (*Alisterus scapularis*), and yellow-tailed black cockatoo (*Calyptorhynchus funereus*) from Victoria, a red-rumped parrot (*Psephotus haematonotus*) from NSW, a little corella and Australian king parrot from Queensland, swift parrots from Tasmania and little corellas from the Northern Territory.

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.

Research on a recombinant vaccine has shown promise. 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. PCR demonstrated reduced virus replication in vaccinated birds compared with non-vaccinated control birds (Bonne et al 2009). **More work needs to be done to assess the vaccine's effectiveness across a range of species and whether or not its production will be commercially viable.**

Human health implications

None.

Conclusions

While PBFD is reported to be endemic in Australia's parrots little work has been done to document its prevalence in different species or locations or what effect it may have on population numbers. This will hopefully be addressed when the Threat Abatement Plan is acted upon.

Acknowledgements

This fact sheet was written by Peter Holz.

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Updated: 10 June 2014.

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