Australian
Wildlife Health
Network

Psittacine Circovirus Disease (PCD, PBFD)
FACT SHEET

Introductory statement

Psittacine circovirus disease (PCD) 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 circovirus disease (PCD) is caused by a 14 to 16 nm non-enveloped icosahedral DNA virus belonging to the family Circoviridae.

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 PCD 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 PCD 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 PCD 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 PCD 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 PCD 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 PCD infection. The haemagglutination inhibition assay (HI) measures PCD 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 PCD 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).

PCD 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 production 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/biodiversity/threatened/publications/tap/pubs/beak-feather-tap.pdf.

Statistics

Limited information is available in the National Wildlife Health Surveillance Database (eWHIS – See http://www.wildlifehealth.org.au/AWHN/home.aspx). Cases reported in eWHIS include rainbow lorikeets (*Trichoglossus haematodus moluccanus*), sulphur-crested cockatoos, and galahs *(Cacatua roseicapilla*) from Victoria, a red-rumped parrot (*Psephotus haematonotus*), and rainbow lorikeets, from NSW, and swift parrots from Tasmania.

Research

Work has progressed for several years on research into the PCD immune response and the feasibility of producing a vaccine (Ritchie et al 1992). Recent work on a recombinant vaccine has shown promise (Bonne 2005).

Human health implications

None.

Conclusions

While PCD 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.

References and other information

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