

Hygiene Protocols for the Prevention  
and Control of Diseases  
(Particularly Beak and Feather Disease)  
in Australian Birds

Avian Polyomavirus Infection



**Australian Government**

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**Department of the Environment and Heritage**

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# Avian Polyomavirus Infection

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Avian polyomavirus or avipolyomavirus (APV) is a member of the Family *Papovaviridae*, which is subdivided into two genera, the papillomaviruses and the polyomaviruses. APV is classified under the polyomaviruses, and is unique among polyomaviruses because it can cause death. The infection has been called budgerigar fledgling disease, papovavirus infection, and APV infection. Most species of psittacine birds appear to be susceptible to infection, as well as *Estrilidae* and *Ploceidae*, appear to be susceptible to infection (Forshaw *et al.*, 1998; Pass, 1985; Johnston and Riddell, 1996). APVs cause pansystemic disease in finches and psittacine birds but have been reported as causes of skin disease only in psittacine birds.

APVs were first reported as a cause of skin disease and mortality in juvenile budgerigars (Bernier *et al.*, 1981; Bozeman *et al.*, 1981). APVs from different host species appear to be morphologically, antigenically and genetically similar. However, Phalen *et al.* (2001) reported genetic diversity of avian polyomaviruses.

Like BFDV, APV are probably capable of causing disease in all psittacine species. However nestling and juvenile birds are most susceptible. The majority of birds that die of APV infection are hand-raised nestlings (Phalen *et al.*, 2001). Most birds which recover from the acute phase of APV disease make a complete *clinical* recovery. Chronic progressive skin disease is not a feature of APV infection. However, persistent virus infection and excretion are common sequelae. Concurrent APV and circovirus infection can occur.

## ***Epidemiology***

The virus may infect birds by the following methods:

- Exposure to infected birds
- Introduction of infected nestlings from another aviary
- Introduction of infected nestlings into a pet shop
- Presence of PCD infected birds
- Exposure to APV-shedding cockatiels, budgerigars, lovebirds
- Nestling-nestling; parent-nestling; handfeeding-nestling
- Do not mix valuable birds with budgies, cockatiels or love birds as the disease is common in these species.
- Many PCD virus-infected birds shed APV in skin and feather dander. Many of these birds (lovebirds, budgerigars, cockatiels) go for long periods without showing signs of PCD, but shed both viruses.

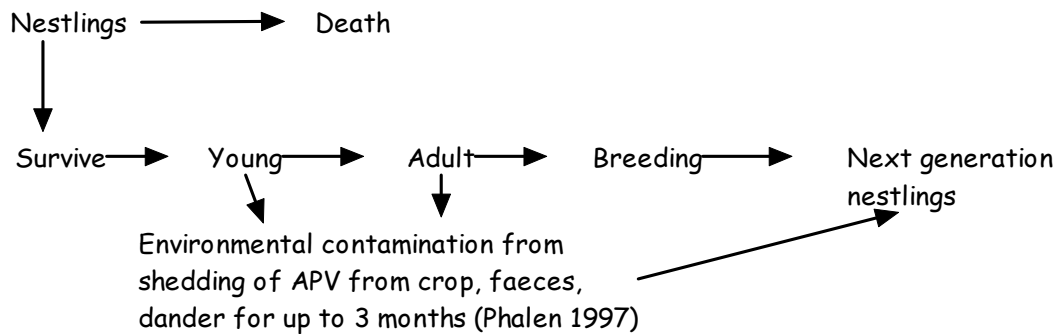
Horizontal transmission is the major method of infection in an epidemic but vertical transmission probably also occurs. Virus is excreted in feather dander and droppings. Infection persists in the kidneys of carrier birds and virus is excreted intermittently in the droppings, probably during times of stress. Polyomaviruses are thermostable, can withstand freeze-thawing and they remain infective in contaminated environments.

APV carriers may be seropositive or seronegative and their serological status can change over time. Up

to 100% of birds in a flock may be persistently APV-infected, but not all will be excreting virus at the time of sampling.

Parent-raised birds (not budgerigars or African lovebirds) do not seem to become diseased, but excrete the virus for up to 12 weeks. Hand-raised birds can have a high mortality.

In an Australian serosurvey of wild cockatoos, 64% of wild SCC had antibodies to APV. Thus it is possible that SCC and other Australian species are original host for APV, and that they have been shipped around the world (Raidal *et al.*, 1998).



**Figure 1:** Cycle of APV from generation to generation

### ***Clinical signs***

APV infection is primarily a disease of nestling birds. Affected nestlings may be ataxic or have head tremors - in some outbreaks the virus targets the cerebellum. There may be abdominal distension due to hepatomegaly and ascites; subcutaneous petechiae or ecchymosis or a generalised pallor. The mortality rate in this age group can be 100%. Death usually occurs within 48 hours of the development of clinical signs. Gross necropsy lesions may be absent. The crop is often distended with food. A severe drop in hatchability has also been reported.

Older budgerigar nestlings may fail to develop normal contour feathers and affected contour feathers may lack normal barbs. The rectrices and secondary remiges may fail to develop. There may be a lack of down feathers on the back and abdomen and a lack of contour feathers on the head and neck. At necropsy there may be cardiomegaly, hydropercardium and hepatomegaly or focal hepatic necrosis. Microscopically, inclusion bodies are found in the kidney tubules, heart, liver, spleen, thymus, bursa of Fabricius, skin and brain.

In non-budgerigar nestling psittacine birds, APV causes widespread haemorrhage, with or without enlarged liver and spleen. Phalen (2001) reported that affected nestling cockatoos may present with respiratory signs. Histologically there is necrosis of the liver and spleen and inclusion bodies are found only in the spleen, kidney and liver. Most cases have been reported in nestling macaws (*Ara* sp), eclectus parrots (*Eclectus* species), conures (*Aratinga* species), and Indian ring-necked parakeets (*Psittacula krameri krameri*). The disease is typically uncommon to rare in the nestlings of African grey parrots (*Psittacus erithacus*), cockatoos (*Cacatua* species), and Amazon parrots (*Amazona* sp) (Phalen *et al.*, 2001). Africa lovebirds and budgerigars are the species in which the disease predominantly occurs.

Most adult psittacine bird infections are asymptomatic and go unrecognised.

### ***APV in Finches and Canaries***

- Nestling deaths at 2-3 weeks old.
- Depressed, off food, delayed crop emptying, regurgitation, swollen abdomen.
- Birds that survive often have poor development and beak abnormalities.
- Recovered birds may become carriers.

### ***Diagnosis***

A presumptive diagnosis of APV infection can be made from the history, clinical and pathological features. However, histopathological, bacteriological and serological investigations should be used to rule out differential diagnoses. Tests which are sensitive and APV-specific are required for making a definitive diagnosis.

#### *Histopathology*

APV infections cause marked basophilic karyomegaly in many tissues, in particular the feather follicles, kidney and liver. Basophilic intranuclear inclusions can be found in persistently infected kidneys but they cannot be differentiated morphologically from other viral infections. A definitive diagnosis requires the use of electron microscopy.

#### *Virus isolation*

Psittacine polyomavirus can be cultured *in vitro* in budgerigar embryo fibroblast (BEF), chicken embryo fibroblast (CEF) or chicken embryo kidney (CEK) cell cultures. However, virus isolation is generally not available for routine diagnosis.

#### *Serology*

Antibodies to APV have been detected by immunodiffusion, virus neutralisation assay (VN) and indirect immunofluorescence. Serology is probably more sensitive than cloacal DNA-probe for detecting polyomavirus infection on a flock basis.

### ***Prevention and Control***

*Latency:* Must be taken into account when trying to control the disease.

#### *Quarantine*

Aviculturists with a disease-free situation, should be encouraged to maintain a closed flock with strict hygiene and quarantine procedures. This includes eliminating exposure to free-flying wild birds and regulating all food, utensils and humans with access to the birds. APV probably remain infectious under the fingernails of well-meaning visitors for long periods. New stock should be obtained from certified seronegative and APV-free aviary flocks. They must be held in quarantine and confirmed as APV-free preferably both by serology and DNA-probe before being incorporated into the breeding flock.

In a non-budgerigar non-African lovebird psittacine outbreak, the infection will spread rapidly in a nursery. If affected nestling birds survive, they may shed APV for up to 16 weeks. If a newly acquired cockatoo is positive, re-test in 12 weeks and 16 weeks. If negative at both these testings, the bird is safe to exit quarantine.

### *Control*

A vaccine is not available in Australia. In an endemic situation an effort should be made to eradicate horizontal transmission between birds and between batches of young birds. Accurate record-keeping and regular disease monitoring are most important. It may be desirable to identify APV carriers and isolate these birds in a separate facility. Incubators and brooders must be capable of being thoroughly cleaned and disinfected between clutches, using 2% Virkon S.

In USA the vaccine is used to immunise birds at 5 weeks and again 2-3 weeks later. Birds will be protected 4 weeks later. Thus protection is from 9-14 weeks of age, and vaccination cannot protect chicks that are less than 9 weeks of age from death. If a bird is shedding virus, vaccination of no use.

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