PSITTACINE BEAK AND FEATHER VIRAL DISEASE IN PARROTS IN THE ACT

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INTRODUCTION

Psittacine Beak and Feather Disease (PBFD) is caused by *Beak and Feather Disease Virus (BFDV)*, a circovirus, which is the smallest disease-causing virus family. The virus is 16 nm in diameter. It is resistant to most disinfectants, and is extremely hardy, surviving outside the host for many months in a harsh environment.

PBFD can affect parrots of all ages. The BFDV multiplies in the liver and is excreted in the feather dust, faeces and crop contents. Inhalation and ingestion of the virus can occur at any feeding, watering and roosting places and to the chicks via feeding. It is suggested that the mother can pass it to the egg. 92% of birds affected are under three years of age, but birds up to 20 years can become infected. The majority of birds die within six months of showing clinical signs. The virus accumulates in the feather follicles, and affects the growth of emerging feathers; and at the growth plate area of the beak, which then affects the beak integrity and shape.

Parrots are flock birds and breed in hollows. These two factors facilitate the spread of PBFD.

The word Psittacine is derived from *psittacinus* (Latin) meaning psittacus parrot, and from the Greek *psittakos*, meaning parrot. There are roughly 372 species in 86 genera that make up the order Psittaciformes which fit into three classifications: Cacatuidae (cockatoos), Stringopidae (New Zealand parrots) and Psittacidae (true parrots). Their characteristics are;

- Cacatuidae - distinguished by a mobile crest, such as Sulphur-Crested cockatoos, Major Mitchell Cockatoos (*Lophochroa leadbeateri*), and Gang Gangs (*Callocephalon fimbriatum)*. These birds lack the highly reflective bright colours.
- Stringopidae - include species such as the Kea and New Zealand Kaka, and the critically endangered Kakapo.
- Psittacidae - include all other parrots, such as rosellas and lorikeets. They are more highly coloured.

PBFD has only been recorded in parrots. Therefore the circovirus, in the wild, is limited to the parrot distribution areas of the tropics and the Southern Hemisphere[1], with most species found in Australia and South America (Figure 1).

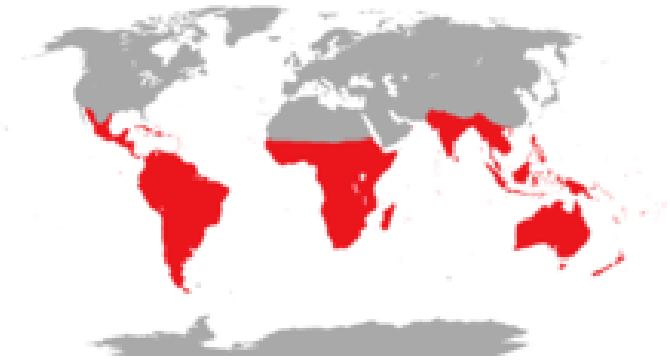

Figure 1. Range of parrots (all species) highlighted in red. (Cooke 2001)

Effects on parrot populations:

PBFD was first documented in wild Red-rumped Parrots (*Psephotus haematonotus*) in 1888 in the Adelaide Hills. Investigation into PBFD was prompted in 1987 when the

[1] Excluding Antarctica

endangered Orange-bellied Parrot population was decreasing. It was then identified as a virus. Birds were taken from the wild to breed for release, but most of the birds in captivity tested positive for PBFD.

Clinical Signs:

There are three types, or stages, of PBFD.

Peracute (sudden) - The peracute stage occurs in hatchlings. They suffer septicaemia, pneumonia, enteritis, weight loss and death even before feathers start emerging.

Acute - At about four weeks of age chicks show symptoms of depression followed by sudden changes in the developing feathers, crop stasis, diarrhoea, anaemia and death.

Chronic – Birds that survive the acute phase go on to show signs of symmetrical feather deformities after the next moult and become progressively worse with each subsequent moult.

The first clinical signs noticed in many parrots, in particular Sulphur-crested Cockatoos, are changes in new feather growth. On the rump the newly emerging, small contour feathers appear necrotic and misshapen. These feathers no longer have the ability to produce feather powder down and this results in the birds having dark and shiny beaks and feet, and dirty-looking feathers.

In species with coloured feathers there are characteristic abnormal colouring of some feathers.

Although there are common symptoms individual birds tend to show these symptoms in varying and differing degrees.

Characteristic clinical signs:

- **Feathers**: symmetrical feather abnormalities, retained feather sheaths, haemorrhage into the calamus, fractured shafts, and constrictions, stress lines and curled feathers; and
- **Beaks** can grow to extraordinary lengths and become necrotic. The beak is characterised by deformed shapes and are very brittle and "sponge-like", often breaking off.

Death usually occurs due to:

- **Secondary infections**, such as pneumonia, because of less feather insulation, or from a combination of candidiasis, aspergillosis, cryptosporidiosis, chlamydiosis and avian polyoma virus, an overburden of parasites; and
- **Starvation** due to the inability to eat with a deformed beak.

Necropsy will often show enlargement of the liver, smaller kidneys and atrophy of the thymus and bursa. There are accumulations of the virus within basophils (white blood cells) found around developing feather follicles.

Diagnosis: The three (of five) most commonly used tests in Australia are the haemagglutination assay (HA) which tests for virus particles in feather dander, the haemagglutination-inhibition (HI) antibody test, and the Polymerase chain reaction (PCR) which tests for viral DNA. A blood sample is required for HI and PCR tests.

This paper provides general factual information on PBFD and shows trends found in the ACT over a three-year period. It aims to provide relevant information for carers to equip them to make informed caring decisions to help prevent the spread of the disease.

Table 1. Clinical signs observed in this research

Clinical Sign – seen in (species)	Description
Feather dust absent – Cockatoos, Galahs	Swiping hand between feathers should result in you hand being covered with feather dust. PBFD reduces the amount of feather dust produced because the contour feathers are not normal
Shiny beak and feet – Cockatoos, Galahs	Instead of beak and feet being covered in feather dust their true colour is revealed
Abnormal feather growth – Cockatoos	Emerging feathers are small, twisted and very abnormal

Abnormal feather growth – Galahs and Rosellas	Feather cover looks ok but some feathers lack colour or have a different than normal colour
Grubby - Cockatoos	Feather dust cover keeps feathers looking nice and clean, lack of feather dust makes birds look very dirty
Crest loss – Cockatoo, Galahs	Crest feathers missing
Blood in feather shafts – Cockatoos	Developing feathers normally close off blood supply when mature. Feathers affected by BFDV do not close off or are fractured, and dried blood can be seen in the calamus.
Beak deformed – Cockatoos, Galahs	BPDV causes deformed beaks and unstable beak integrity
Tail feathers missing - all parrots	Missing tail feathers
Symmetrical wing feather loss – all parrots	After moult new feathers do not grow. Moult occurs symmetrically

My research attempts to show variation in common, visual clinical signs in Sulphur Crested cockatoos where disease is obvious by the time they are received into care and to define clinical signs in other parrots.

METHODS

From November 2006 to May 2009, psittacines brought to the RSPCA ACT Wildlife Clinic because they were sick or injured were tested for PBFD if they were suspected carrying it. However, because of time, financial and staffing restrictions only some of parrots were tested. Molecular Diagnostic Services uses the PCR test.

Initially Sulphur-crested Cockatoos were tested until it was realised similar trends in other parrots indicated possible PBFD. These clinical signs were different and less defined.

A form was designed with a column for each clinical sign that was recognised. MDS supplied kits, each consisting of a small tube with a strip of 'blotting" paper. The sample required was a drop of blood 'blotted' to cover the last 3mm of the paper. This was then sealed in the tube and posted to MDS in Queanbeyan. There were no other special provisions and the sample did not need to be kept fresh or processed quickly.

Blood samples were taken from the brachial wing vein and the bird was subsequently euthanased. They were euthanased, without the benefit of a test result, because even if they were to prove negative the birds were underweight and usually had other problems, for example; broken bones (hit by cars) or suffering other diseases. RSPCA ACT lacks quarantine facilities and if all tested parrots were housed in the same facility those that were negative at the time of testing would have been positive for PBFD by the time the results were returned, and other parrots in care could be infected.

A thorough examination of the birds followed and clinical signs noted on the form with the date and clinic number.

RESULTS

The following data demonstrates strong trends in the Sulphur-crested cockatoos and Galahs. Numbers of Eastern Rosellas and Crimson Rosellas and other parrots were insufficient to come to any viable conclusions.

Sulphur-crested Cockatoos

During the testing period 490 cockatoos were brought into the wildlife clinic, 187 (38%) of those had feather abnormalities and were undernourished and thin. Most others sustained injuries caused by vehicle strikes.

Of the 90 cockatoos studied, 10% were used as controls to establish normal parameters. The controls showed no clinical signs of PBFD. They had an average weight of 794g.

Figure 2 shows birds that tested positive to PBFD had an average weight of 580g and the negative birds had an average weight of 640g.

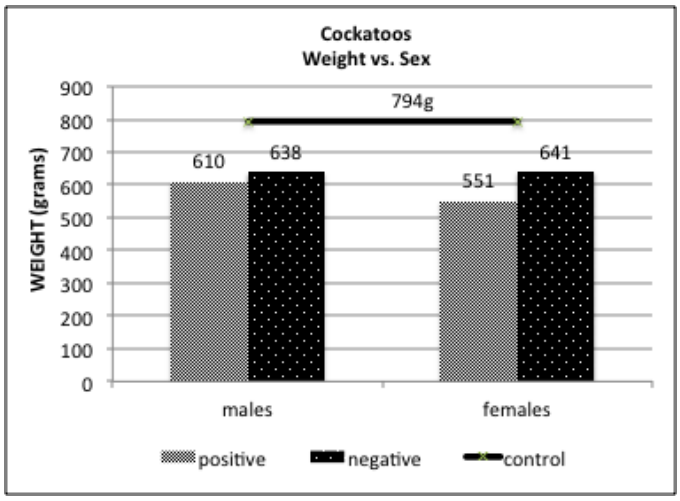

Figure 2. showing the weights of positive and negative cockatoos compared to the average weight of control cockatoos.

The clinical signs of 78 cockatoos were compiled and Figure 3 gives the percentage of birds that exhibited each symptom. Clearly the most common clinical signs were a lack of feather dust, shiny beak and feet and abnormal feather growth. Deformed beaks were only seen in 46.1% of birds. In positive tested birds females had significantly less symmetrical feather loss (Figs. 3 and 4).

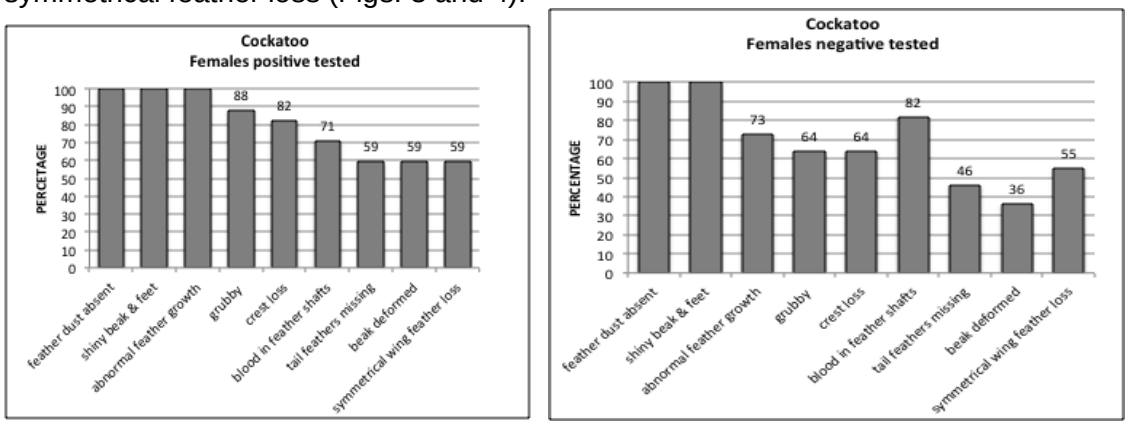

Figure 3. Percentages of clinical signs in female cockatoos. Positive (left), negative (right)

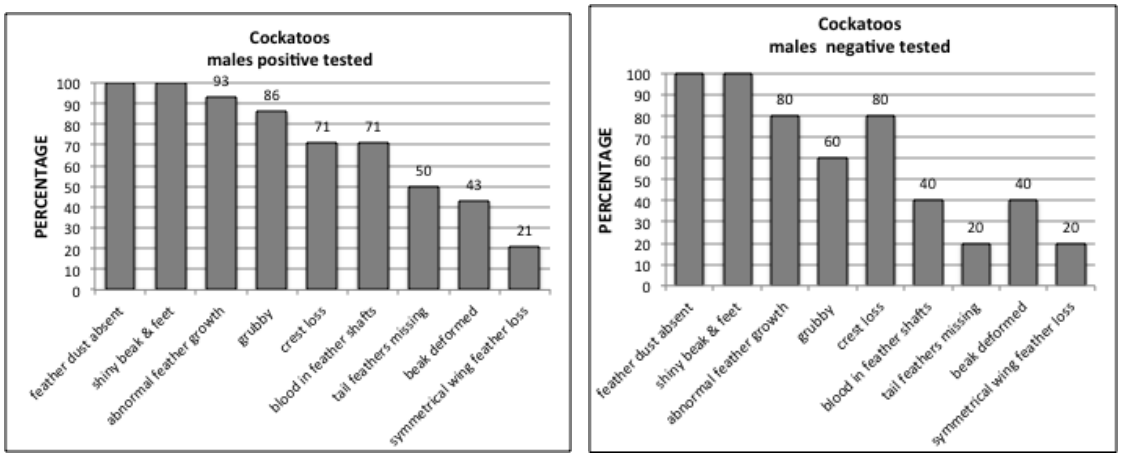

Figure 4. Percentages of clinical signs in male cockatoos. Positive (left), negative (right)

Cockatoos came from all suburban areas in the ACT.

Galahs

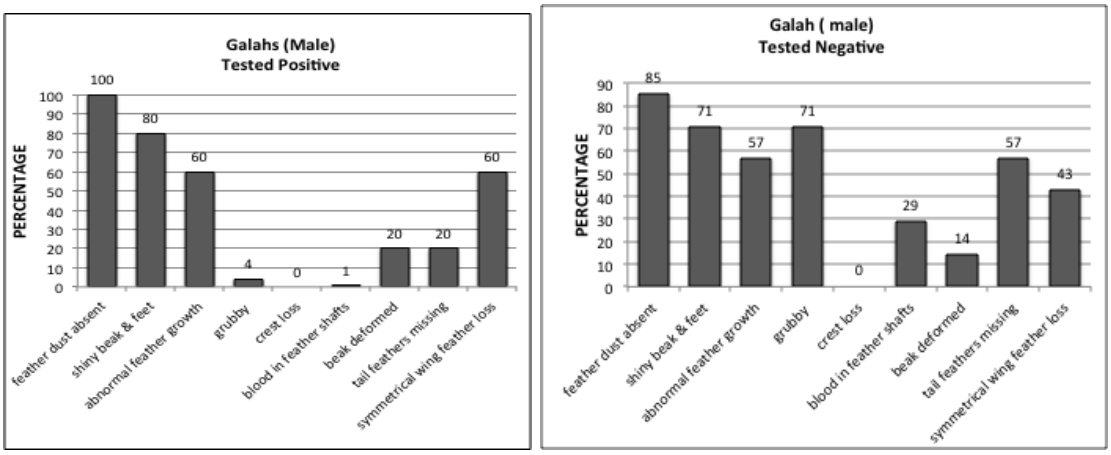

Figure 5. Percentages of clinical signs in male galahs. Positive (left), negative (right)

Positive galahs show different proportions of clinical signs than cockatoos although they still have a significant amount of feather dust loss (Figs. 5 and 6). Only females had lost some crest feathers. Not enough female Galahs tested negatively for meaningful interpretation.

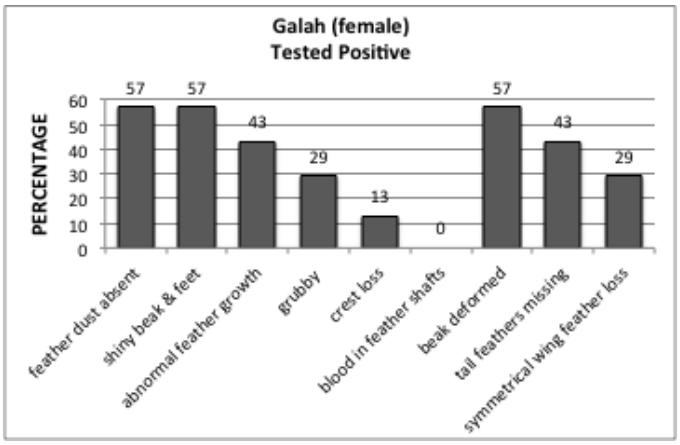

Figure 6. Percentages of clinical signs in female galahs

Crimson and Eastern Rosellas

There were not enough rosellas to give meaningful results except to say that their clinical signs were quite different to those of Sulphur-crested Cockatoos. Figure 7 (Eastern rosella) and Figure 8 (Crimson rosella) illustrates this. At least half the rosellas missing tail feathers were attacked by cats or dogs.

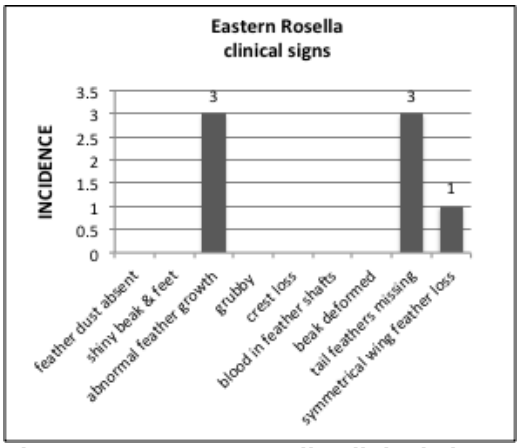

Figure 7. Eastern Rosella clinical signs

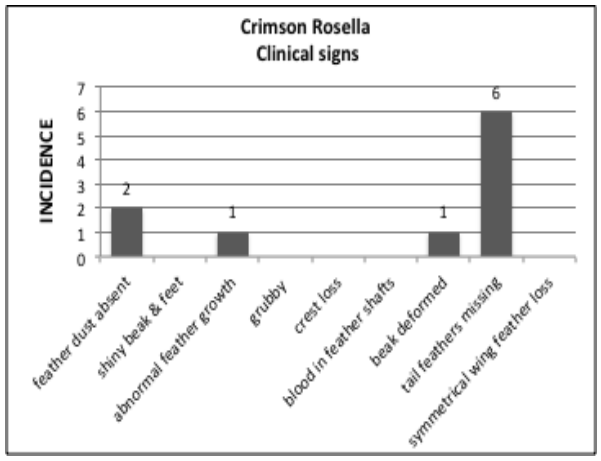

Figure 8. Crimson Rosella clinical signs

Other species

There was one Major Mitchell Cockatoo (not tested), three Corellas (one positive), two King Parrots (one positive), and one Gang Gang Cockatoo that arrived after the experiment had concluded but tested positive.

DISCUSSION

Cockatoos that were received into care were generally underweight. Figure 2 shows that the positive PBFD birds were 73% normal weight. The clinical signs shown in the highest proportions are the same as the visual signs in the wild. Positive male and female cockatoos showed the same trends. Negative tested cockatoos had the same signs of lack of feather dust and abnormal feather growth. This leads to the question why? The birds were chosen for testing because they exhibited those characteristic signs but still tested negative. More investigation needs to be carried out.

The same applies to Galahs (Figs. 7 and 8). Galahs have less feather dust than Sulphur-crested cockatoos and rosellas even less. The charts reflect this. But why do negative Galahs have loss of feather dust?

Whereas cockatoos show abnormally formed feathers, Galahs tend to have some of their feathers without grey colouring. Rosellas, on the other hand, show more feather discolouration, where their brighter colours are a different colour. Research into factors for feather colour development and the mechanism by which this is changed would be useful.

The. Sulphur-crested Cockatoos and Galahs, members of the cockatoo family, showed the most obvious clinical signs. However, Crimson and Eastern rosellas, being true parrots, showed different clinical signs. (Figs. 9 and 10).

Most parrot populations thrive despite the presence of PBFD. Large populations are not likely to be threatened but smaller populations could become extinct if the numbers are not self-sustaining. With a decline in numbers there is a loss of genetic diversity.

Most parrot species are widespread in Australia and their habitats overlap. This will facilitate spread of PBFD. Management of the disease is impossible in the wild.

We have seen the disastrous results of PBFD on the population of Orange-bellied parrots, despite the efforts to breed them in captivity. Populations of endangered Australian parrot species could potentially be affected by PBFD. Carnaby's Black cockatoo (WA), Kangaroo Island Glossy Black cockatoo (SA), Golden-shouldered parrot (Qld), Ground parrot (WA), Norfolk Island Green Parrot, Red-tailed Black cockatoo (Qld, NT, WA), and the Swift parrot (SE Aust.) are all critically endangered.

Looking at this list it is obvious that birds around the country are at risk. With altered habitats and diminished natural food supply there are extra stresses on birds and some populations have declined.
Wildlife carers have a responsibility to remove birds that are affected by PBFD. These birds would naturally die and stop spreading the disease and the population is sustained. If rehabilitated parrots no longer showing clinical signs of the disease are released, they are still latently infected. The circovirus remains active in the liver.
For any research to be undertaken it is essential that the object of the research is defined at the outset and the statistics to demonstrate it are built into the methods. The research undertaken here was ad hoc, sporadic and poor data collection from wildlife staff lead to insufficient quality data for accurate analysis. Future research should be well planned.

Acknowledgements
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MARG PEACHY: Marg Peachey began caring for wildlife in Canberra in 1992. In 1998 she was employed by RSPCA ACT as Education Officer and did the rounds of schools giving talks on domestic pets and wildlife. She belonged to The Wildlife Foundation which amalgamated with the RSPCA so injured, orphaned and sick wild animals started to be taken to the RSPCA. She was the point of contact for these animals. She gradually built up a reasonable wildlife section and after a year or so could not manage as a part-time wildlife officer, and was made full time.
In 2003 the bushfires burnt out the RSPCA. And out of the ashes they moved into an old house and set up a decent clinic and by 2008 were taking in 3500 animals a year. She left RSPCA in 2009, and was asked by our TAFE to write a course on Wildlife Rehabilitation and is now teaching it. She is now studying for a Post Graduate Certificate in Ornithology through Charles Sturt University.