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Psittacine Beak and Feather Disease (PBFD)

Description: *Psittacine Beak and Feather Disease* - The virus causing this disease is a member of the *Circoviridae.* The molecular structure of the genome of the virus is roughly a 2,000 base, circular, single stranded DNA. PBFD virus has a strong resemblance to Porcine Circovirus as well as to a number of plant viruses such as the Banana Bungy virus.

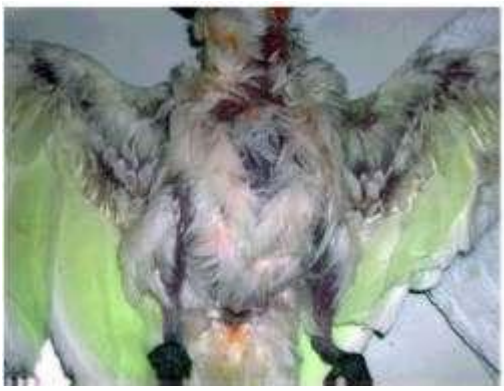
The disease is thought to be specific for psittacines and all psittacine species should be considered susceptible. Parrots known to be particularly affected by PBFD include, but are not limited to, Cockatoos, Macaws African Grey Parrots, Ringneck parakeets, Eclectus Parrots, Lovebirds.

Causes fatal infections, primarily in young birds. Older birds may overcome the disease with few lasting affects. Some believe that these surviving birds become carriers able to shed the disease at a later date. Others believe that a percentage of birds are able to eradicate the disease from their system leaving them with a natural immunity that can be passed on to their offspring.

The virus that causes PBFD can also affect the liver, brain, and immune system causing diminished resistance to infections. Consequently premature death usually occurs from these secondary bacterial, fungal, parasitic, or viral infections.

Transmission: Transmission of the virus from one individual to another is primarily through direct contact, inhalation or ingestion of aerosols, crop-feeding, infected fecal material, and feather dust. The virus can also be transmitted via contaminated surfaces such as bird carriers, feeding formula, utensils, food dishes, clothing, and nesting materials. The viral particles, if not destroyed can remain viable in the environment for months, long after the infected bird is gone.

Symptoms:



Symptoms include irreversible loss of feathers, shedding of developing feathers, development of abnormal feathers, new pinched feathers, and loss of powder down. Other possible symptoms include overgrown or abnormal beak, symmetrical lesions on the beak and occasionally nails. Immunosuppression, rapid weight loss, and depression are also possible in later stages of the disease.

Secondary viral, fungal, bacterial or parasitic infections often occurs as a result of diminished immunity caused by a PBFD viral infection. Additional symptoms not mentioned above including elevated white cell counts are generally due to secondary infections and may not be directly related to PBFD virus infections.

Prevention: Strict isolation of all diseased birds to halt the spread of the disease. DNA testing of all birds of susceptible species to rule out latent infection. DNA testing of aviary equipment and environment to test for possible contamination.

Treatment: No known treatment. Experimental vaccines are being developed.

Diagnosis: Skin biopsy, surgical biopsy of feather and shaft, or PCR testing of blood, swab, and feather samples.

PBFD should be considered in any bird suffering from abnormal feather loss or development. A biopsy of the abnormal feathers including the calimus (shaft) of the feather can be examined for signs of virus. However, since the PBFD virus does not affect all feathers simultaneously this method of evaluating a sample may have a high degree of error. Additionally, birds with PBFD can have normal feathers and the PCR test is the most effective method available for detecting the virus in birds before feather lesions develop.

Some birds infected with the virus, test positive, but never show clinical signs. Other birds which test positive may develop an immune response sufficient enough to fight off the infection and test negative after 30-90 days. Therefore, it is recommended to re-test all PBFD positive birds 60-90 days after the initial testing was completed. If the second sample remains positive, the bird should be considered permanently infected and can be expected to show clinical symptoms of the disease.

Sample: To test an individual bird a whole blood sample is recommended in conjunction with a cloacal swab or feathers (especially abnormal or suspicious-looking feathers) when possible. If the sample tests positive the bird should be placed in quarantine and re-tested after 4-6 weeks. If the bird tests negative the second time a third test after 4-6 weeks is recommended.

Post-mortem samples include liver, spleen, kidney, feather samples in a sterile container; postmortem swabs may also be submitted.

Environmental testing using swabs of aviaries, countertops, fans, air-filters, nest-boxes, etc. is extremely effective in determining the presence of PBFD DNA in the environment.

*It is recommenced to submit both a whole blood and cloacal swab sample for analysis when possible.

Handling: Prior to shipping samples should be stored at 4 C. (refrigerator). Samples must be shipped in a padded envelope or box. Samples may be sent by regular mail, but overnight is recommended.

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Psittacine Beak and Feather Disease (PBFD)

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Psittacine Beak and Feather Disease (PBFD) is a contagious, fatal viral disease that affects the beak, feathers, and immune system of birds belonging to the Psittacidae family. It was first recognized in 1975 by veterinarians in Australia, where the disease affects wild birds. Although birds showing signs of disease usually die, it is common for birds to be exposed to the virus, develop a mild infection, and recover.

What birds are at risk for PBFD?

PBFD has been diagnosed in over 40 species of psittacines, including South American parrots. Although all members of this family appear to be susceptible, PBFD is seen more often in cockatoos. Eclectus parrots, lovebirds, budgies, and African grey parrots are also affected. Younger birds are more commonly affected, especially with the ***acute*** form of the disease. Most birds affected are under 2 years of age.

What causes PBFD?

PBFD is caused by a ***DNA*** virus that affects the cells of the immune system and those that produce the beak and feathers. The virus is a circovirus, which is one of the smallest viruses known to cause disease. A similar virus affects doves and other birds.

How is the virus that causes PBFD transmitted?

PBFD is extremely contagious. Large amounts of the virus, which can become airborne, are found in the droppings, contents of the crop, and the feather dust of infected birds. The feather dust is easily dispersed and can contaminate food, water, cages, clothing, and other areas of the environment. The virus is thought to be transmitted by inhalation or ingestion of the virus. It has been suggested that the virus may be transmitted in utero from the female bird to the egg.

The incubation period (time between exposure to the virus and the development of signs) can be as short as 3-4 weeks, or up to several years, depending upon the amount of virus transmitted, the age of the bird, the stage of feather development, and the health of the bird's immune system.

What are the signs of PBFD?

There are both acute and chronic forms of the disease.

Peracute/Acute Form: The peracute and acute forms most commonly occur in very young birds, and may begin with signs unrelated to the beak or feathers. Affected birds are often depressed and regurgitate due to crop ***stasis***. They may develop a diarrhea-causing ***enteritis*** or pneumonia, and die without displaying any lesions of the feathers or beak. This is often called the peracute form of the disease. In the acute form, juveniles losing their down and developing feathers may have lesions on the feathers, including circular bands around the feathers which constrict its base. These feathers are often loose, break easily, may bleed, and are very painful.

Common Signs of Psittacine Beak & Feather Disease	
Acute Form	**Chronic Form**
Depression Regurgitation and diarrhea Loss of appetite and weight Abnormal feather development Death	Loss of feather dust and powder Abnormal feather development Abnormal growth and deformities of the beak Necrotic beak and oral lesions Secondary infections Death in months to years

Chronic Form: In the ***chronic*** form of PBFD, which is more common in older birds, the powder-down feathers are often the first feathers affected. The feathers are fragile and fracture easily, have constricting bands, may ***hemorrhage***, and may be discolored, deformed, or curled. As the feather ***follicles*** are damaged, the bird will soon be unable to replace feathers, and the primary, secondary, tail, and crest feathers are lost. Bare skin is exposed, and the normal feather dust is not found on the body or the beak, where it normally accumulates due to preening. Feather abnormalities, often termed "***dystrophic*** feathers," may not appear until the first molt after infection, which could be a period up to 6 months.

The beak may develop irregular sunken areas. Brown *necrotic* areas may be found inside the upper beak, and the beak may elongate, become deformed, and fracture. Secondary beak and oral infections often occur. In some birds, the nails can also be deformed or slough.

Mucus in the droppings, or a green tint to the droppings may occur. In some birds, the liver will be affected, and liver failure may be the cause of death.

Birds with the chronic form of the disease may live for months to years before dying of a *secondary infection*. This long period of illness in which the bird may be featherless, and gradually weakens can be very emotionally difficult for owners.

How is PBFD diagnosed?

The review of the medical history, presence of clinical signs, and observations during the physical exam support the diagnosis of PBFD. Other conditions such as nutritional deficiencies, infection with polyomavirus (causes budgerigar fledgling disease and other diseases of psittacines), hormonal abnormalities, and drug reactions can cause lesions on the feathers similar to PBFD. Histopathology (microscopic examinations of biopsies) can confirm the diagnosis. Affected cells will have abnormalities in their nuclei, called "basophilic intranuclear inclusion bodies." The diagnosis may also be confirmed by a PCR (polymerase chain reaction) test on whole blood or biopsy samples from the affected bird. The test detects the presence of the virus. This test may also be used on swabs of surfaces in the environment to detect contamination.

False positive and false negative test results can occur. For example, infected airborne cells could contaminate a sample and cause a false positive result. Healthy birds with a positive test result should be retested after 90 days. If they still have positive test results, they should be considered carriers of the virus. If the retest is negative, the bird may have eliminated the virus, and become immune.

False negative results may occur if too much anticoagulant is present in the sample, an extremely high number of viral particles are present and interfere with the test, or there are an insufficient number of infected white blood cells in the sample.

How is PBFD treated?

There is no specific treatment for PBFD. Supportive care including good nutrition, supplementary heat (incubator), beak trimming, and treatment of secondary infections can be offered. The disease, however, is progressive, and very few birds recover. Euthanasia may need to be considered for birds with severe and/or painful signs. Birds who die a natural death usually succumb to a secondary bacterial, fungal, or viral infection despite treatment, since their immune systems have been critically suppressed. Most birds die within 6 months to 2 years of developing the disease.

How is PBFD prevented and controlled?

Birds should be purchased from suppliers with disease-free birds. New birds coming into facilities should be quarantined and tested. Repeat testing in 3-4 weeks to allow for the incubation period is recommended. Infected birds should be isolated and removed from breeding programs. Juvenile birds should be housed separately from adults. Bird owners need to understand that if they handle other peoples' birds, it may be possible for them to bring the virus into their home and infect their birds. Good hygiene and sanitation should be used. The susceptibility of the virus to disinfectants is unknown. Disinfectants which are known to be effective against parvoviruses are probably the best choice.

In Australia, a *killed vaccine* has been developed which can protect unexposed birds; it can cause more severe disease in birds already showing signs of PBFD. Birds should be vaccinated as young as possible, as soon as 14 days of age. The vaccine should be boostered after one month, and breeding birds should be vaccinated one month prior to breeding.

References and Further Reading

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Raidal, SR. http://wwwvet.murdoch.edu.au/caf/pbfd.htm. Murdoch University. Perth, Western Australia.

Rupley, AE. Manual of Avian Practice. W.B. Saunders Co. Philadelphia, PA; 1997.

http://www.vetark.co.uk/pbfd.html

This dreadful disease is caused by a circovirus. It has a wide species range although it appears to be a natural virus infection of cockatoos in Australasia where it occurs in wild flocks. It has been known in wild cockatoos in Australia for many years and recently Ducorps cockatoos from the Solomon Islands have been round to be infected. Old world parrots show the infection most commonly. In the US eclectus and cockatoos led African greys. New World parrots such as Amazons and macaws showed less of the disease. Smaller species such as lovebirds, cockatiels and parakeets also showed the infection very commonly.-

African greys as an unnatural host seem particularly acutely affected, young birds may simply die or develop feather loss first, others may develop red feathering (seen in wild birds also unrelated to PBFD).

Feather colour changes are also reported in Vasa parrots. It causes typical French Moult signs in budgies, and similar signs in lovebirds and ringnecks. A few species have been reported to eliminate infection and recover, this seems commoner in lovebirds than any other species.-

Variable levels of feather loss are seen, in some birds it may develop slowly with only a few abnormal feathers each moult. Rapidly growing feathers are affected first, eg. powder downs in african greys and cockatoos, losing their natural dust they often develop an untidy greasy plumage and shiny beaks. It may be seen at the first formation of feathers to replace down. Sudden loss or deformities of feathers, often blood in the sheath. It may also develop in adult birds at subsequent moults. Perhaps a few affected feathers each time.-

The virus is found in feather dust, faeces and in crop fluids. It is believed to spread through the egg.

It has been reported that birds may simply die of either disease without showing signs. Liver or kidney swabs clipped off and dropped into carrier medium may be sent from probing if these diseases are suspected. PBFD tests PCR testing is very sensitive. Contamination by virus from other positive birds, whether dead or alive will make a sample positive. The test finds virus if it is there - from any source.

In just the same way that your sample may be contaminated from via environment (from an unsuspected carrier) it is not uncommon for chicks to show severe problems yet parents are negative. These chicks are often being infected from a contaminated environment.

We cannot control potential contamination at the time of collection so ensuring the sample is not contaminated is your responsibility. To avoid contamination of the sample with PBFD virus from the environment (originating from other birds) it is essential to thoroughly clean the birds nail area. We recommend Ark-Klens (from VETARK) for this. RELYING ON DISINFECTION IS NOT SUFFICIENT. CLEANSING IS VITAL. Dead virus will give as strong a positive reaction as live virus. Alternatively have your veterinary surgeon collect the sample direct from a vein by venipuncture. Birds become immuno-suppressed and may die from other diseases.

You can now use the standard DNA collection kits to take blood from parrots for PBFD testing. Alternatively a veterinary surgeon will collect the sample directly from a vein using a syringe and needle (venipuncture). Feather pulp from abnormal feathers is also a potential source of virus. This is squeezed straight into our collection tubes. Because birds with a serious degree of feather lesions may be immunosuppressed, and because this can hide the virus we recommend that such birds be sampled by collecting blood and feather pulp samples in the same tube. Do not simply send us dried feathers - they are a poor sample for reliable testing - so as a policy we don't test them.

Please use or at the very least liaise with your avian vet. Interpreting what the test means to you and your birds and what if any action is required in your situation needs veterinary input. We can assist your vet but we cannot get involved with cases directly.

What does a positive test result mean ?

A positive PBFD test result means that the the PCR test detected PBFD DNA in the sample. A positive result from a bird with feather abnormalities suggests strongly that the bird has an active infection.

A positive result from a bird with no feather problems may mean either that the bird is a carrier or that it has been recently exposed to the virus. In these cases we recommend re-testing in 90 days. We also recommend that the second sample is collected by venipuncture to ensure that contamination does not occur. The majority of birds which are merely exposed will mount an immune response and eliminate the infection.

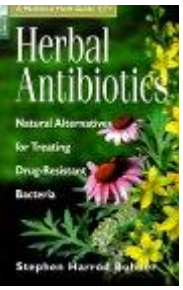
Those still positive at the 90 day test should be considered carriers. One day they are likely to show the disease, and be potentially infectious.

Various uses of the disease tests

- to test clinically suspect birds
- to examine material from post mortem examinations of dead birds
- to check collections for carriers and to look for in-contacts
- to test 'new birds' at pre- or post-purchase veterinary health checks eg. as pets or before entering breeding collections
- to test birds in the pet shop

http://www.avianweb.com/PBFD.html

Psittacine Beak & Feather Disease or PBFD is caused by a virus which infects and kills the cells of the feather and beak. The virus also impairs the immune system. Consequently many diseased birds succumb to bacterial and other infections.



Psittacine Beak and Feather Disease is a viral disease that has first been noticed in cockatoos, but has since been diagnosed in many species of birds, specifically in African Greys, budgies, cockatoos, Eclectus parrots, lovebirds, macaws, and Rosellas.

PBFD is one of the diseases that can be passed from bird to bird and the risk of spreading this, or other diseases, is a good reason to quarantine any new bird that comes into your household. PBFD is extremely contagious and there is no known cure and vaccines are only now being developed. Birds carrying this disease may not show any symptoms until stress brings it out, but they may infect other birds before they become symptomatic

Visual symptoms include feather abnormalities, beak abnormalities and missing feathers and occurs normally in young birds but can also be found in older birds. Some birds die from the disease before showing the above symptoms. Loss of appetite, diarrhea and regurgitation may be the only signs of illness. Often death is caused by a secondary infection due to the reduced immunity caused by PBFD.

Potential Treatment / Supportive Care

Very few birds survive PBFD although they may live a fairly long life with good care and very little stress. Supplementing with vitamins, minerals, and probiotics to boost the immune system will help, and treatment of secondary infections will be required regularly.

According to Dr. Ross Perry FACVSc (Avian health) notes that some parrots presenting with acute to subacute disease made a clinical recovery just by being put on a "balanced diet", usually based on organic well-formulated pellets or crumbles supplemented with a little of a lot of fresh organic greens, vegetables and fruit, and given a little tender loving care for 1-2 moults.

- **A Beak and Feather Disease Survivor- Sweetpea's Story** (a very encouraging and uplifting story about a lovebird that sufferered from this disease but recovered)

- Providing **supportive care** helps birds focus all their energy on getting well.

Identifying whether your bird has the Beak & Feather Disease (Psittacine Beak & Feather - PBFD)

Feather Plucker or PBFD? Young chicks are not likely to pluck; so this would not apply to chicks. However, if an adult bird develops bald spots, you might consider either possibility. You can distinguish between PBFD and normal feather plucking by looking at where the feathers are being lost. If they are missing from the head and crest - an area they cannot get to with their beak to pluck - then it is likely to be Psittacine Beak and Feather Disease.

A Bird Suffering from PBFD ...

- is likely to show the characteristic abnormal feather and beak growth
- might have feathers that look like stubbles and are obviously deformed
- is likely to have short 'clubbed' feathers
- may develop curly feathers
- may have feather shafts that often break, or you might see narrowing or pinching of the shafts (this condition worsens with each molt and your bird will usually become progressively balder due to inactivity of its feather follicles).
- may have a beak that is deformed, especially the upper beak, and often overgrown; the beak usually splits or breaks.

Your Birds:

- Young birds suffer from an acute form of PBFD that occurs during their first feather formation, after replacement of down feathers. The developing feathers often fracture, bleed or fall out.
- Some chicks may die following a short period of anorexia (loss of appetite), depression and diarrhea, with very little feather abnormality.

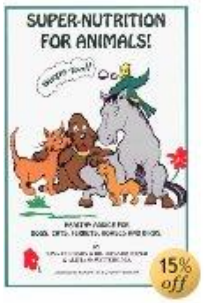
Symptoms / Disease Progression

PBFD should be considered in any psittacine bird that displays progressive feather loss or abnormal feathers. Most birds which succumb to PBFD are less than 2 years of age. However, all age groups should be considered susceptible to circovirus infection.

Young birds are affected by an acute form of PBFD, which occurs during their first feather formation, after replacement of down feathers. The developing feathers often fracture, bleed or fall out. Young birds may die following a short period of anorexia (loss of appetite), depression and diarrhea, with very little feather abnormality.

Older birds are thought to develop a chronic form in which dystrophic feathers stop growing shortly after emerging from the follicles. The feathers become increasingly abnormal with each successive molt. Contour feathers are usually affected early, while primary feathers are affected later in the disease. Contour feathers often are lost over most of the body. New feathers may have retained feather sheaths, blood within the shafts, are curled and deformed, or are short and clubbed. The beak may also be involved in the disease process. It may change from a dull black to a glossy appearance. It may grow abnormally long and develop splits and cracks which break and peel. Bacteria and fungi often invade the abnormal beak, causing further destruction and necrosis (death) of the tissues. The abnormal beaks often make it difficult for the bird to eat as it may be very painful.

Spontaneous recovery from acute PBFD can occur in many species. However, the majority of chronically affected birds do not recover from the disease.



Transmission:

PBFD is spread by inhalation or ingestion of virus particles. Feather dust has been found to contain a large amount of virus. The virus has also been found in crop secretions and in fecal material. The virus may also be ingested as a result of preening. The incubation period of variable among species and the age at which the bird is exposed. Again, neonates and young birds are most susceptible, while adult birds over two years of age are thought to be at less risk.

It is possible for a bird to undergo a transient subclinical infection. This means that the bird's immune system is able to eliminate the virus. This is why it is recommended that a normal appearing bird who tests positive be retested 90 days later. If the bird has eliminated the virus it will test negative. If it remains positive, it should be considered latently infected and should be expected to break out with clinical disease in the future.

This is considered a fatal disease, and there is no cure, or treatment known.

A pet bird with PBFD can live a long life, if it is in a stress-free environment. It would never have contact with other birds since it is capable of spreading the virus.

Diagnosis / Testing:

Whole, anticoagulated blood should be submitted from a bird without feather abnormalities, while both blood and several abnormal feathers should be submitted from a clinically abnormal bird. A test was developed by Dr. Brandon Ritchie, and is run by Avian Research Associates Laboratory.

Interpreting the Results of the Psittacine Beak and Feather DNA probe test.

- **A. If Bird Has Dystrophic, Necrotic Feathers and you Test Blood for PBFD Virus using DNA probes:***
    - 1. If **Positive:** Suggests Active Infection

      Management: : If bird is from a breeding aviary: Bird should be removed and all areas that could be contaminated with feather dust from the infected bird should be repeatedly cleaned. If companion bird: Bird should not be exposed to other birds outside of the household and you should be aware that the virus can be transported to other locations on your clothes or in your hair. Be courteous of other birds and do not expose them. It should be noted that, occasionally, some PBFD infected Psittaciformes of South American descent have spontaneously recovered from the disease.
    - 2. If **Negative:** A feather biopsy (including the feather follicle) should be submitted for histopathologic examination.
- **B. If Bird's Feathers are Normal and you Test Blood for PBFD Virus using DNA probes:***
    - 1. If **Positive:** Indicates that the bird has been exposed to PBFD virus and that the virus is present in the blood. The bird must be retested in 90 days. If the bird is negative when retested, it indicates that the virus was not detected in the blood cells. If the bird is still positive, it indicates that the bird is either clinically infected or that the bird is being repeatedly exposed to the virus. It should be noted that most birds that are

exposed to the PBFD virus develop a transient viremia followed by an appropriate immune response that results in the bird clearing the infection.

- 2. If **Negative:** Indicates that PBFD virus was not detected in the blood.

*Testing available from:
INFECTIOUS DISEASES LABORATORY DEPARTMENT OF MEDICAL MICROBIOLOGY COLLEGE OF VETERINARY MEDICINE UNIVERSITY OF GEORGIA
ATHENS, GA 30602-7386

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A Beak and Feather Disease Survivor- Sweetpea's Story (a very encouraging and uplifting story about a lovebird that sufferered from this disease but recovered)

Beak and Feather Disease Survivor- Sweetpea's Story

Story by Mary W.

Sweetpea came into our lives in December of 1995. My daughter, then 11 years old, wanted a pet of her own, one that she could "cuddle with", she said. We debated about the nature of such a pet, and decided that another bird, preferably a hand-fed tame baby would best fit into our avian (two cockatiels) and human family. So, with that in mind, we chose a three month old peachface lovebird who'd come right to us, and cuddled in my daughter's hand. We named this little green fluffy ball of energy Sweetpea, the name suited her, she would cuddle in our hands, on our shoulders, and under our shirts, or in a pocket, and she loved nothing better than a scratch. She joined us, along with our cockatiels, at meals, and accompanied us as we went about our daily activities at home. We were amazed at her exhuberance and her "go-for -the gusto" attitude towards everything she encountered. She was playful, curious, and fearless in her approach to everything she encountered. Her antics kept us laughing , we never knew what Sweetpea would do next!

About two months after we got Sweetpea, we noticed that the feathers on her back and wings seemed to be fading from the bright grass green they had been, to a brownish-green, muddy- looking color. In my ignorance I thought perhaps the color change indicated that Sweetpea was acquiring her mature coloring! Sweetpea ate a varied diet, consisting of seeds, pellets, healthy (for the most part) "people food." Her droppings were normal, and her activity level was what we had come to expect as normal. So we had no clue that this color change might indicate the presence of a serious disease in our beloved Sweetpea.

Following the color change in Sweetpea's feathers, and no doubt corresponding to her first molt, she began to lose feathers, beginning around her eyes and beak. This feather loss continued gradually over the next few months, from her face and head, extending down her chest, back, tops of her wings, and her legs.

When the feather loss first appeared, I had taken Sweetpea to our avian veterinarian. He examined her, performed several tests, and determined that she had a yeast infection around the beak and face. In addition to the feather loss, the skin around her beak and eyes seemed inflamed and itchy, poor Sweetpea continually scratched the area. The vet said that it was a little unusual to see a yeast infection in a six month old bird, these are seen more often in baby birds still being fed by parents or handfed. So it was possible the presence of a yeast infection under these conditions might indicate that Sweetpea had an underlying immune problem. He also performed a blood count, and found that her white blood cell count was extremely low (her WBC count was 900/mm3, normally counts in birds are 5000 to 10,000/mm3). He told me that this also suggested that Sweetpea might have a serious viral infection, and warranted further investigation, possibly for PBFD, if she continued her feather loss, and the infections did not improve.

The antifungal medication prescribed for Sweetpea had no effect , the inflammation around her face, and the feather loss continued. Thinking that the redness and swelling might be due to a bacterial infection, the veterinarian prescribed an antibiotic, which had no effect either. At this point, the veterinarian suggested that we have Sweetpea tested for PBFD. So, in March of 1996, blood was taken from Sweetpea and sent to Dr. Branson Ritchie's laboratory at the University of Georgia for testing. In this laboratory , the blood is tested for the presence of DNA from the virus which causes PBFD. This method is the most sensitive test for PBFD, and indicates only the presence of the PBFD virus in the blood, not the clinical status of the bird from whom the blood is taken. The results must be correlated with the clinical findings in the bird, but a positive test in a bird who has feather loss and other findings associated with this disease strongly suggests that the bird does have PBFD.

The PBFD results on Sweetpea came back positive. The veterinarian informed us that usually under such circumstances, euthanasia is recommended. He said that PBFD was generally fatal, and that we could expect Sweetpea's health to decline over a period ranging from a few months to a year, ending in her death, probably within a year or so. He told us that as PBFD progresses, the disease becomes painful and debilitating. The disease effects not only the feathers, but also the immune system, not unlike human AIDS. So the bird, besides losing feathers, falls prey to any number of infections because his immune system cannot function to fight the microorganisms which cause these infections.

We were, of course, heartbroken at this devastating news. But we declined the euthanasia at this time, since Sweetpea seemed well other than the feather loss, and some inflammation around her beak and eyes. She was still eating well, playing, enjoying life and appeared to be in no pain or discomfort, she still had her "go-for-the gusto" approach to life! We thought we would postpone the euthanasia until the time came when it was evident that Sweetpea was suffering. The veterinarian agreed with our decision, since Sweetpea didn't act sick, and he even expressed some surprise that she seemed so perky even with the physical evidence of PBFD.

We worried about the possibility of the PBFD being contracted by our cockatiels. The veterinarian told us that research had showed that PBFD could be contracted only by young birds, generally less than one year old. He said that this research had found that it was impossible for a bird greater than three years old to contract PBFD. Since the tiels were each about eight years old at this time, the risk of their getting PBFD from Sweetpea seemed virtually nonexistent. We decided to take Sweetpea home, and give her the best life we could, keep her warm, and loved, as long as we could, until it was evident that we had to end her suffering.

We spoiled that little bird rotten! We let her eat, or drink about anything she wanted, since we thought there was not much to lose. She drank milk, orange juice, ate ice cream, crackers, in addition to her own food. Even as the feather loss continued, she played, ate well, and exhibited her normal enthusiasm and curiosity for everything going on around her. During the three to four months after her diagnosis, she wanted to spend most of her time tucked inside someone's shirt, and we let her do this when we were home. She must have spent about 18 hours a day inside a shirt, she came out to eat, to satisfy her curiosity about

something, to hassle the cockatiels, and of course when we went out, or to bed. We covered her cage well for warmth at night, and when we went out. Fortunately we live in a warm climate so we didn't have to worry about trying to keep a featherless bird warm in cold temperatures. I think Sweetpea spent a great amount of the time under our shirts sleeping, and I now believe that she did not feel well much of that time.

There was no medication, or treatment for PBFD. Sweetpea had only the antifungal, and antibacterial medication, for treatment of her infections. I tried to make sure she had extra vitamins and protein, I sprinkled her food with Ornebac, rubbed Vitamin E oil on her face, I thought that might alleviate the redness and itchiness. I think she might have eaten some Vitamin E oil too, as I'd let her bite open the capsule containing the oil. I'd try anything anyone suggested which was reported to have anti-viral properties, or which might make Sweetpea feel better, as we waited for her decline, believing this was inevitable. We even debated the euthanasia issue, wondering if it would be kinder to put Sweetpea to sleep before she actually began to suffer from the PBFD.

About five to six months after her diagnosis, we noticed that Sweetpea looked as though she might be growing some feathers back around her beak and face, where she had first lost feathers. The feathers looked normal, and as this process continued , I took Sweetpea back to the veterinarian so he could verify that she WAS growing feathers back. He did verify that the feathers looked normal, and mentioned at this point that very rarely, a bird may survive PBFD. He said it might be possible that Sweetpea would be one of these rare survivors, but it was too soon to tell at this time.

Over the next few months, Sweetpea continued to grow feathers back on her head, chest, back, wings and legs. This time the feathers showed the adult coloring, ie, red on her forehead, peach around her face and chest, and the feather color on her back was again the bright grass green. Several times in the next year she lost some feathers again, and she still looked rather motley. But these feathers were always replaced by feathers which were normal in appearance. I took her back to the veterinarian a little over a year after her diagnosis, she was nearly fully feathered, and healthy by then. The blood test for PBFD was repeated at this time, and it came back still positive. The veterinarian said that a positive test at this time might indicate that either Sweetpea was in remission, but could relapse at any time with PBFD; that she could be an asymptomatic carrier of PBFD; or that she was fighting the PBFD virus and eventually her immune system would fight it off, and she would become negative, PBFD-free. He didn't know which of these situations would occur, since he had never seen a bird survive PBFD prior to his experience with Sweetpea.

Sweetpea continued to thrive in the year following her second PBFD test. She grew, gained weight, and became a beautiful, brilliantly colored bird, who of course, had never really lost her exuberance for life. We had the test for PBFD repeated a third time two years after the first, positive test. This time the test results came back negative. The veterinarian told us that this was a first for him, as he had never seen a bird who was symptomatic and sick with PBFD survive this disease.

Sweetpea is now a healthy, beautiful five year old lovebird. She still continues to delight us with her enthusiasm for life, her antics, and her wonderful personality. I realize how fortunate she is, and we are, to be among the handful of documented PBFD survivors. I never realized how rarely a PBFD infected bird survives this disease. I recently spoke to our veterinarian about Sweetpea, and he said at this time that he has not seen another PBFD survivor. I've spoken to other avian veterinarians as well, many of them say they have not seen birds who have survived this disease. I'm sure there must be other PBFD survivors out there, we just don't know about them.

Some of the people who know us, and Sweetpea speculate on the factors that contributed to her recovery from PBFD. Some folks say the biggest factor was the love and care we gave her while she was fighting the disease. The veterinarian suggests that maybe it was her own spirit, her own determination to keep going, and enjoy life regardless of how she might have felt. Personally, I'd say, all of the above, and a great immune system that eventually got the best of this PBFD virus. Maybe her recovery falls under the category of a miracle. I don't think we will ever know for sure what made Sweetpea recover from PBFD, but we all agree that she is a special, and amazing bird.

Story by Mary W.

CURRENT CONCEPTS ON PSITTACINE BEAK AND FEATHER DISEASE AND AVIAN POLYOMAVIRUS

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Introduction

Viral diseases of pet birds have some of the most severe emotional and economic impact upon aviculture of any disease group. They also have been historically the most difficult diseases to confirm on diagnosis and to manage in our avian population. Traditionally diagnostic methods have inherent problems in detection (sensitivity & specificity) and interpretation of results. They also often fail to accurately identify the actively infected individual, especially in the sub-clinical "carrier" state.

Over the last 15 years, advances in the field of molecular biology have allowed for the development of extremely sensitive and specific nucleic acid (DNA, RNA) detection methods. Diagnostic testing and improvements in test methodologies have allowed for a greater understanding of the epidemiology and management of avian disease.

DNA Based Diagnostics

The first applied use of viral specific DNA technology in avian disease diagnosis, was marked by the development of tests for the Psittacine Beak and Feather Disease (PBFD) and avian polyoma (APV) viruses (Psittacine Research Group, University of Geaorgia).(1) Research Associates Laboratory (RAL) has commercially offered these tests since 1992 and is both the oldest and largest molecular biology based laboratory serving the veterinary community.

RAL's DNA based diagnostics, use viral specific nucleic acid probes, to identify the unique DNA sequence making up the desired viral genome. These sequences are detected in DNA extracts from submitted blood and tissue swab samples. DNA amplification techniques coupled with internal sequence probes allow for diagnostic tests of extreme specificity and sensitivity.

The performance of RAL's diagnostic tests has been evaluated both in-house and by an independent biotechnology laboratory. Test sensitivity, which is how accurate the test is in reporting a positive infected bird, as positive, is 99.7% for PBFD and 98.2% for APV.(2) The accuracy of the tests in reporting a negative infected bird as negative was 100% for both tests.

Aviculturalists and veterinarians should be keenly aware of the intense capabilities of these diagnostic methods. The presence of contaminating virus in areas of high avian traffic (hospitals, stores, aviaries etc ...) should not be underestimated. Prudent sample collection and handling is necessary to prevent environmental contaminants from producing a positive test result. The levels of test specificity demonstrated above show that the incidence of "false-positive" test results is virtually non-existent in these tests.

Aviculturalists should also keep in mind that infection does not always equal disease. The majority of birds exposed to these viruses will remain clinically normal and mount an effective immune response which eliminates the virus. These birds are in essence "naturally vaccinated" and test positive only throughout the time active virus is present. Recent advances in RAL test technology allow for a quantitative (numerical) value to be assigned to the DNA test results. This valuation of test data helps to differentiate an active viral infection from non-progressive viral exposures. Results of this nature will prove useful in the management of test positive individuals in the near future.

PSITTACINE BEAK AND FEATHER DISEASE

The characteristics of PBFD have been well described.(1) The acute form of this disease is most commonly observed in young or fledgling birds during their first feather formation after replacement of the neonatal down. Chicks as young as 2 months of age have been described with classic PBFD feather lesions.(1) These infections may be characterized by necrosis, fracture, bending, hemorrhage, or premature shedding of developing feathers. Chicks that develop clinical lesions while the majority of feathers are still in the developmental stage exhibit the most severe feather pathology. The clinical progression of disease is less dramatic in young birds that develop clinical signs after body contour feathers are mature. In these birds, feather changes may be limited to the still developing primary flight and tail feathers.

In some peracute cases in young birds, PBFD may manifest itself as depression, anorexia, crop stasis, & diarrhea followed by death in 1 - 2 weeks. Since these birds are covered only with neonatal down, no feather abnormalities will be observed. This clinical picture appears to be particularly common in young cockatoos, African greys, and lovebirds.

A more chronic form of the disease is observed in older birds in which dystrophic feathers, that stop growing shortly after emerging from the follicle, appear during each successive molt. The powdery down feathers located over the flank region are typically the first to show signs. The disease then progresses to involve the contour feathers in most feather tracts, followed by dystrophic changes in the primary and secondary feathers of the wings, tail, and crest.

PBFD is generally considered fatal, with most infected birds surviving 6 months to 2 years after the onset of clinical signs. The PBFD virus is immunosuppressive and death usually occurs from complications due to secondary bacterial, viral, or fungal infections, or from terminal disease changes which necessitate euthanasia.

We have previously reported on the analysis of approximately 10,000 PBFD tests at this laboratory. (2) Approximately 5% of birds tested positive for PBFD. The majority of these birds were not exhibiting feather abnormalities or other outward signs of PBFD disease. Most of these birds were sub-clinically and transiently infected with the PBFD virus. With a mature, functioning immune system, most birds are capable of mounting an effective and protective immune response, which results in elimination of the PBFD virus. Retesting of these individuals 90 days later is recommended, at which time most will show a negative test result. These transiently infected birds represent a large portion of positively identified birds at our laboratory. Birds that continue to remain test positive should be considered latently infected and may break with clinical disease at a future date. All test positive individuals should be isolated from other birds until they test negative.

Old World psittaciformes show the highest incidence of positive tests. Eclectus species had an overall positive rate of 10% followed by 8.7% for cockatoos and 8% for African Grey parrots. New World psittaciformes had shown a much lower positive test incidence. The rate for macaw and amazon parrot species was approximately 4%. Of interest is the apparently high rate of positive tests in lovebird species, which exceeds 30%. This positive test rate most probably results from selectively using the test for diagnostic purposes (suspected clinical cases) as compared to testing as a screening tool to document a bird's health status (as is common with the larger pet bird species).

The current positive test rate for PBFD at this laboratory is approximately 3.5% - 4%. Essentially the rate has decreased only slightly over the past 5 years. Many avian practitioners however, report a dramatic

decrease in the incidence of observed clinical PBFD disease. This is probably due to the fact that by testing and isolating positive individuals, we have reduced the exposure of susceptible individuals (neonates), which are more likely to develop the clinical disease in response to PBFD viral infection.

Situations have arisen where multiple clutches of baby birds have tested positive and shown clinical disease, in light of negative blood tests on parent birds. Environmental contamination, as determined by testing environmental swabs, is the major source of infective virus in these situations. The duration of time that the PBFD virus remains viable in the environment has not been determined. It is generally accepted however that PBFD is viable for a prolonged period of time. Thorough cleaning of the nursery premises has eliminated the problem of pediatric infection in most of these cases. Contaminated nursery environments should be considered a major source of PBFD (and APV) viral transmission.

Observations of clinical PBFD in baby birds demonstrates that the time of viral exposure in relation to the maturity of their immune system is a determining factor in the progression of clinical disease. In one aviary situation, baby scarlet macaws were pulled from the aviary at 3-4 weeks of age. In the nursery, these birds developed transient feather abnormalities compatible with PBFD disease. Abnormalities were evident in new feather growth that occurred over a 2-3 week period of time. Blood and feather pulp tests during this time were positive for the PBFD virus. After several weeks, abnormal feather growth had ceased and all subsequent new feather growth was normal. Future blood tests on these birds were negative. Existing abnormal feather pulp at this time was still test positive. Eventually, no abnormal feathers could be identified and all tests were negative. Environmental and parent bird testing revealed that these babies were infected from exposure in the contaminated nursery envirorunent. They successfully mounted an effective immune response, which resulted in elimination of the virus.

Another recent case involved a 4-week-old African Grey parrot that was tested negative for PBFD immediately prior to sale to a store. The bird was sold from the store at 8 weeks of age at, which time it tested, positive. Evaluation of the pet store revealed a contaminated nursery environment with several other hand-feeding birds PBFD positive. The store requested euthanasia but the 8-week-old parrot was not showing any evidence of clinical PBFD disease. RAL's new quantitative viral testing indicated a transient rather than progressive viral infection. The baby was isolated and retested 4 weeks later. Quantitative testing revealed that while still positive levels of circulating virus were significantly lower. Repeat testing at 60 and 90 days later were negative and the bird remains negative and clinically normal to date.

DNA probe testing has had a tremendous impact on the incidence of observed clinical PBFD disease. It has proven to be a useful means by which to control and reduce this deadly disease. Observations derived from clinical testing and consulting with the avian community across the country summarize new points in our understanding of PBFD disease:

1. Most birds with adequate immune system function, when exposed and infected with the PBFD virus, mount an effective immune response, which results in elimination of the virus. These birds are in essence "naturally vaccinated."

2. New World Psittacines appear to be inherently more resistant to PBFD infection and disease when compared to Old World species.

3.The positive test rate incidence in birds has shown little change over the past 5 years.

4.The incidence of chronic clinical disease is declining in the U.S. avian population.

5.Most observed infections are now transient in nature.

6.Contaminated avian environments remain a major source of PBFD viral transmission.

AVIAN POLYOMAVIRUS

Avian polyomavirus (APV) was first characterized as a pathogen of pet birds in young budgerigars

(Melopsittacus undulatus) in the early 1980's. (3-5) Since this time, it has been determined that most species of psittacine birds, as well as some Estrilidae and Ploceidae, are susceptible to APV infection. (1,6 -11) APV is a serious, economically important disease affecting the pet bird industry. (7)

Although the APV virus that infects budgerigars and other psittacine species appears to be the same, the clinical disease, distribution of lesions, and epidemiology of infection differ. (1,7,8,14 -17) APV, also described as budgerigar fledgling disease, causes variable morbidity and mortality, and abnormal feather development (French Molt) in this species. The feather abnormalities caused by APV resemble those of PBFD. The difference is that normal feathers will return with the next molt if the initial abnormalities were due to APV. With PBFD, abnormalities become progressively worse with successive molts.

The true impact of APV disease in budgerigars has been subject to some misrepresentation. It has often been stated, albeit incorrectly, that 1) all budgies are infected with APV and that 2) once infected, budgies remain infected for life. Individuals and breeding colonies have been identified that have no history of APV disease. These birds have negative APV antibody titers and are negative on APV DNA testing. The obvious conclusion is that there is no evidence that these budgerigars are infected with the APV virus. APV negative budgerigars do exist. In regards to point #2, positive infected budgerigars that have converted to negative status and also produced negative offspring have been documented (Dr.D.N. Phalen, pers. communication). Once APV infected, all budgerigars are not infected for life.

In non-budgerigar psittacine birds, APV typically affects young birds, 2 weeks to 14 weeks of age. It generally causes death within 48 hours after the onset of acute depression, crop stasis, biliverdinuria, diarrhea, and hemorrhage. (1,5,7,8,17) While APV mortality in susceptible young birds ranges from 20% to 80%, (1,5,7) infection does not always result in death or disease. Some young birds infected with APV recover after a brief illness, still others do not show any evidence of clinical disease. Others may also become persistently infected with APV. These "carrier" birds have been considered a source of APV maintenance and spread within aviary populations. (18,19)

Polyomavirus diagnosis

Diagnosis of polomavirus infection in the live bird was previously based upon observation of the characteristic clinical disease. Other infectious agents however, may produce similar disease signs. Serological assays, which measure a bird's antibody levels, can help determine a history of APV exposure. While antibody assays may not accurately predict current infection, one assay does show excellent correlation with blood based DNA testing (Dr. D.N. Phalen, collab. research). (19-22) The molecular DNA based assay, developed by the Psittacine Research Group at the University of Georgia, has been offered by this laboratory since 1992. (2,4) The test uses virus-specific DNA probes to confirm the presence of APV nucleic acid in cloacal, fecal, tissue, and environmental swab samples. This test detects birds that are infected with and shedding polyomavirus in their droppings.(23) The fact that APV infected birds shed the virus intermittently however, makes this test unreliable in predicting the infected status of birds with negative swab test results.

While the identification of non-clinical, persistently infected "carrier" birds has been difficult, circulating APV has been demonstrated in blood and serum samples by DNA based diagnostics. (1,17) While this method was thought to have value as a screening tool, it did not appear reliable for individual bird testing. (1,11) New improved technologies for APV detection, were designed and researched at RAL. This new molecular based assay has been shown to consistently and reliably detect the presence of APV in blood samples from infected birds, independent of clinical disease or virus shedding. (23)

It is unfortunate that some veterinarians still erroneously recommend the swab test over the blood APV assay for screening birds infected with APV. They support this recommendation by stating that the swab test provides the aviculturalist with more pertinent clinical information because it detects birds that are actively shedding the virus. This is a disadvantage however, not a benefit. The swab test only detects birds that are shedding. The blood APV assay detects birds that are shedding the virus and those that are infected but not shedding. It has been documented in one APV study that the swab test only detected ~14% of the positive APV infected birds. This means that 86% of infected birds were diagnosed as negative when indeed they were infected with the virus. Would you feel comfortable adding one of these

birds to your aviary collection? While other attempts to develop a blood APV assay have been made, RAL researchers were the first to develop and validate the blood DNA, APV assay. This assay is currently the best test to perform when screening a bird for APV infection.

The practical applications of this new test technology, to the study of the epidemiology and management of APV disease, were previously reported. (23) Individual birds in an aviary with a confirmed APV epornitic, were tested for APV infection. The aviary population of 143 birds consisted of cockatiels (n=21), lovebirds (n=11), conures (n=88), Quaker parakeets (n=5), Senegal parrots (n=3), Amazon parrot (n=1), macaws (n=13), and cockatoo (n=1) species. All birds were initially tested for APV infection on blood and cloacal swab samples. Test positive birds were isolated in a physically separate facility and retested at monthly intervals. Negative retest individuals were isolated to minimize reinfection exposure and eventually returned to the aviary. Cleaning efforts were also conducted within the aviary to eliminate environmental contamination as a source of future APV infection.

Of the 143 birds in this aviary collection, 22 birds (15%) tested positive for APV. All positive test birds showed positive test results on whole blood samples whereas only three (14%) were positive for APV shedding on the cloacal swab assay. None of the birds tested showed a swab positive, blood negative test result and no adult birds were positive for APV viral shedding on the cloacal swab assay. The blood test was superior in detecting birds infected with the APV virus, when compared to the cloacal swab assay.

The original source of the virus was traced to two conures that were recently added to the aviary collection. Within two weeks of the onset of testing and isolation procedures, no new cases of APV disease were observed. Eventually all birds were returned back to aviary with the exception of the two conures. Over the last two years, this aviary has produced in excess of 300 babies and has not experienced any recurrence of APV disease. The blood APV assay has proven useful in the control and elimination of APV infection in this aviary.

However, aviculture has been misled about the benefits of this useful technology. It has been reported by some individuals, albeit incorrectly, that "fragmented DNA", the product of cellular viral processing, results in a positive diagnostic test. This infers that the blood APV test identifies many birds as positive for infection when active virus is not actually present. Analysis of extracted DNA, in actual diagnostic samples submitted to our laboratory, does not reveal evidence that DNA fragmentation is a significant problem. Isn't it odd that the criticism of "fragemented" DNA is directed only at blood APV testing when testing for PBFD, which uses the exact same sample and test format, is the "saving grace" for eliminating this deadly disease? These same molecular biology methods are considered "gold standard" in human medicine where fragmented DNA is not a confounding issue.

There exists a purposeful attempt to perplex both avian veterinarians and aviculturists in regards to this DNA technology. Lay individuals and veterinarians, with no background in the field of molecular biology, have been supported in their supposed factual interpretations of blood polyomavirus testing. These articles are wrought with factual error and distort the true scientific facts. Such misinformation serves only to confuse and confound the industry. Aviculture must demand a higher level of honesty and integrity from these "leaders."

Researchers at RAL were the first scientists to develop and prove the benefits of blood polyomavirus testing. Our interpretations have been dervied from scientific study and compare favorably with the findings of other prominent researches in the field. Additionally, RAL consults with aviculturalists and veterinarians across the country on a daily basis where these interpretations continue to prove valid.

Age related infection and disease

The age of a bird at the time of infection and more specifically, the competence of it's immune system, appear to affect the outcome of APV infection. (17,19) While some young birds exhibit mortality characteristically associated with APV infection, others appear to recover, while still others show no clinical illness. It has also been suggested that resistance to APV disease results from a modified host response to virus infection and not by an increased resistance to infection. (17) Young Blue and Gold macaws (Ara ararauna), experimentally infected with APV, developed high virus neutralization antibody

titers indicating infection, but failed to show clinical disease.(1) Viral induced cytopathic changes, were observed in experimentally infected Budgerigar nestlings, although the birds never showed signs of APV disease.(3) No birds over two months of age, which tested positive in the RAL aviary study, showed evidence of clinical disease. Additionally, most positive testing birds, including new aviary additions, became consistently blood negative in a short time. These observations suggest that most immune competent psittacine birds, when infected with APV, mount an effective response, sufficient to prevent disease and eliminate the virus.

Persistently infected "carrier" birds
It has been theorized that birds infected before they are inununocompetent, may become tolerant of APV and remain persistently infected. (1,20) A clinically ill and recovered Goldcap conure in the aviary study, continued to test APV positive until 10 months of age. It is interesting to note that this bird had both polyoma virus and high levels of APV antibodies in its blood. Antibody titers do not successfully eliminate the viral infection in these "carrier" birds. Many researchers believe that active cellular immunity (killer T lymphocytes) not antibody, are needed to eliminate the viral infection. We have also confirmed circulating APV in the blood of an adult Sun conure (Aratinga solstitialis) that was diagnosed as a "carrier" for a period of four years (Dr. D. Phalen pers.com). It appears that birds can remain asymptomatically infected with APV for extended periods of time. Intermittent viral shedding from these "carriers" can serve to maintain and spread APV throughout an avian population.

Polyomavirus disease in adult birds
While it has been reported that polyomaviral disease can occur in adult psittacines, the incidence of this is extremely rare. Contrary to what the avicultural community has been led to believe, almost all infections in adult birds are asymptomatic. (11,27) Combined infections of APV and PBFD have been shown to occur. (11,13,23) It appears that APV disease in adult birds requires immunosuppresion, such as that from PBFD infection, for clinical APV disease to occur. (11,16) We recently tested DNA samples from three adult Ecelectus parrots and two cockatoos which died from confirmed APV disease. All these birds also tested positive for concurrent PBFD infection. APV disease in finches has also been suggested to result from immune suppresion. (28) Of all 143 birds tested in the aviary study, no adult birds were observed to exhibit clinical APV disease. It has also been suggested that adult birds recently infected with APV, serve as an amplification source for virus infection within the aviary. However, none of the adult study birds which tested blood positive, were shown to be actively shedding APV virus on cloacal swab testing.

APV and AVIARY MANAGEMENT
The aviary we studied had a 14 month history of morbidity and mortality from confirmed APV disease. With the implementation of blood testing and strict preventive practices, the last clinically observed case occurred at the end of 1995. Through the 1996 and 1997 breeding seasons, the aviary has produced over 300 neonates, with no incidence of APV disease. Additionally, random testing of young birds has confirmed the negative APV status in this aviary. Blood APV testing has proven to be a useful tool for the management and elimination of APV in closed aviary populations. It is the best and most economical method to screen new birds and prevent the introduction of this virus into your aviary population. Absolutely, no bird should be added to your aviary unless it has tested negative on blood APV testing.

The following statements derived from blood DNA and serology APV tests along with clinical disease observations, summarize new points in our understanding of APV disease:

1. Most birds with adequate immune system function, when exposed and infected with the APV virus, mount an effective immune response, which results in elimination of the virus. These birds are in essence "naturally vaccinated".

2. Infection in adult birds is inapparent, as is infection in many juvenile birds.

3. APV disease most commonly occurs in New -World species. Disease is most often observed in lovebirds, budgies, ringneck parakeets, conures, macaws, caiques, and eclectus parrots.

4. Clinically diseased birds most often show signs within 2 weeks of virus infection. They are viremic at this time and have begun to develop circulating antibody.

5. Birds that are infected but do not die, still develop a viremia and also develop high antibody titers. Viremia persists in these birds even in the face of high circulating antibody. These birds often become persistently infected for extended periods of time and can serve to spread the virus in a population.

6. Blood APV testing, combined with effective management procedures, can reduce and eliminate APV disease in a closed aviary population.

DISCUSSION

Much controversy exists surrounding our interpretation of APV disease. There is a strong disagreement and difference of opinion among prominent researchers in regards to the epidemiology, pathogenesis, and management of APV disease. Much of this controversy however is "man-made" and due to a rampant distortion of the true scientific facts. The prudent aviculturist should use common sense and relay on the honest interpretation of scientific observation and data. Likewise, it is unfair that aviculturists should be demeaned into thinking they are inferior or sub-standard for not complying with a particular technology, especially when such disagreement exists. Remember many statements and opinions are made but they are often meaningless without the firm support of sound scientific data.

There is no perfect solution to controlling APV disease. It is unlikely that we will ever achieve 100% compliance with any control method and ever totally eliminate a particular viral disease threat. We can however achieve control and reduce the likelihood of these diseases in our avian populations. If you have not had a problem with PBFD or APV you can insure that you will not in the future by not bringing the virus into your collection. The DNA-based tests offered by Research Associates Laboratory provide the avian community with the best method currently available for insuring that a new bird is negative for these disease agents.

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Less common problems are dealt with in Part II.

Over the past few years fanciers have increasingly reported feather abnormalities in their exhibition type budgerigars. In particular, they have noticed a tendency for birds to lose their tail feathers which then fail to regrow ("tail-less wonders"). Reports of similar abnormalities in larger members of the parrot family increased at the same time.

In view of these reports, discussions took place between the Budgerigar Society Lancashire, Cheshire & North Wales BS and myself. It was decided that the two societies would fund work on feather diseases to be carried out at the University of Liverpool. Their financial support is acknowledged with thanks. The work was begun on 1 July 1993 and concluded on 30 June 1995.

We are grateful to members of the fancy for the supply of birds and feathers together with related information. Birds and other samples were received from all areas ranging from the Isle of Wight to Glasgow and from Cornwall to Norfolk, though the majority came from Lancashire, Merseyside, Cheshire and North Wales. The total number of birds and feather samples received was 198.

Results Published to Veterinary World

A technical version of this report will be produced and submitted to the veterinary press, making the results available to veterinary surgeons. They will then know which diseases commonly occur and will be able to advise clients with feather problems in their birds.

Results

The diseases and other abnormalities found in the survey are summarized in Table 1 (at the bottom of this page). Also in the table are figures from the other comparable work found in the literature. This was a survey of biopsies (small pieces of tissues taken from live animals) from a variety of psittacines with feather disease, which were examined microscopically. The work was carried out in America (Schmidt 1987). It will be noted from the table that the diseases found in the American survey were of a limited nature.

Only some of these were seen in my survey but at a different prevalence. The exception was psittacine beak and feather disease, where the incidence was equivalent in both surveys. In the Schmidt survey the causes of the diseases were not investigated, nor was there any attempt to correlate certain diseases with feather type. Caution is required when interpreting the figures in Table 1. The figures are correct for the birds we received, but some fanciers supplied large numbers of birds with the same condition and this has skewed the results. This was particularly true in the case of psittacine beak and feather disease, where two breeders supplied over half the affected budgerigars. With pulpitis, the same tended to occur, though to a lesser extent. Conversely, many fanciers did not submit birds with French moult as this disease was specifically excluded from the project. For these reasons the figures in the table give only a rough guide to the prevalence of these conditions in the overall budgerigar fancy.

It will be noted from Table 1 that there was an average of 1.48 conditions per bird. Most of these were cases of mite infestation and damage in birds with other feather disorders. In other cases two apparently unrelated feather conditions occurred in individual birds.

Terms

Before considering the results in detail it may be useful to define a few terms.

Viruses

These are a type of germ which lives and breeds inside the cells of animals. They are capable of living outside the animal but will not multiply in this situation. As far as birds are concerned no treatment for viral diseases is available.

Bacteria

These are much bigger than viruses and usually live between the cells of the infected animal and multiply both within the animal and outside. They can usually be killed by antibiotics.

Congenital Disease-

This is a disease which the bird has from the time it hatches, although occasionally the symptoms may not show for months or even years. Such diseases may or may not be inherited.

Inherited Disease

This is a disease which is passed from parent to offspring via the animal's genes. Depending on the mode of the inheritance the parents may, or may not show evidence of the disease.

Vertical Transmission of a Disease

This is transmission of a disease from parent to offspring by contact or via the egg. Such diseases are not inherited although they may appear so.

Horizontal Transmission of a Disease

This is transmission of a disease to other in-contact birds but excludes parent to offspring transmission.

Follicle
A small pocket-like depression of the skin from which the feather grows and by which it is attached to the bird before a moult.
Barb
The branches coming from the feather shaft and forming the bulk of the feathers.
Barbules
These branch off from the barbs and have small hooks on them (barbicels) which lock together the barbs and thus the feather.

The part of the report which follows details the findings in each condition or groups of conditions and gives an indication of the cause and treatment when this is known.

Psittacine Beak and Feather Disease

It came as a considerable surprise that this topped the list of diseases occurring in 21.2% of all the birds submitted, because in the first year no cases were seen at all. One possible explanation is that the disease has only recently been introduced into the exhibition budgerigar and only became widespread in the last year or so.

The disease is caused by a virus. Infected birds may be symptomless carriers although no such cases were found in the survey. This was to be expected as only sick birds were seen. Fanciers were unlikely to send in a symptomless bird for examination. There appeared to be two manifestations of the condition depending on the age of the bird. In budgerigars less than 6 months old the feathering was poor with extensive bald or semi-bald areas on the body and legs, loss of most or all of the large wing and tail feathers, and loss also of some of the smaller feathers in these sites. The remaining feathers tended to be of poor quality and sometimes misshapen. Young affected birds tended to be small for their age. In older birds the contour feathers were usually unaffected, but all, or some of the primaries, secondaries and large tail feathers were missing. Regardless of age of the bird the head and upper neck was spared. In no cases was the beak abnormal. Affected birds seemed lively and active and no bird died from the condition, although such birds were kept at the university, only a few months at most.The disease is transmitted to other birds on the premises both horizontally and vertically, although transmission tends to be relatively slow. There is no treatment for the disease, nor are there any preventative treatments in the UK. Measures such as cleanliness, the testing of new arrivals and the isolation of affected birds, will reduce the rate of the spread of the disease. An experimental vaccine has been shown to be effective in the USA. Should fanciers or societies wish to support further research into this disease I can provide a contact name in America.

Pulpitis

This is an inflammation of the pulp of the growing feathers, especially the large ones of the wing and tail. Such diseased feathers may break off or be shed prematurely. Once shed the feather may not regrow. A significant proportion of the birds submitted with this disease had been sent in as "tail-less wonders". Pulpitis is believed to be a major cause of this condition. The cause of pulpitis is usually a bacterial infection,predominately Staphylococci or Streptococci although about 15% of cases are probably of viral origin. These germs are thought to live on the bird's skin but only cause problems when they invade the growing feathers. There is a strong correlation between this condition and markedly buff, suggesting there may be something inherent in buff feathers which makes them susceptible to this condition. There is also some evidence of vertical transmission.

While in theory, prolonged antibiotic treatment might cure the condition when a bacteria is the cause, this has not been tried. Repeated bathing of the birds in Virkon S seems to cure about 40% of cases. A few appear to recover spontaneously. At the present time there appears no way of predicting which birds will respond to treatment and which will not.

Mites

Feather mites of a variety of species are very common on budgerigars, but are not usually seen by the fancier as they are mostly very small. They are visible only under microscope. They also tend to live deep in the feathers close to the skin. In the majority of birds they cause no problems at all. Occasionally mites can cause feather disease in a number of different ways. There were feather abnormalities in all the cases of mites listed in the table. Firstly, in heavy mite infestations these active parasites caused irritation. This in turn led the birds to scratch and bite their plumage, leading to tatty feathers, or feathers which had been bitten off at various positions along the shaft. As mites prefer to live on a bird's rump, the tail feathers are frequently the most affected. Secondly, some species of mite will eat small segments of the growing feathers, so when they are fully formed a small part of the feather is seen to be missing. These are most obvious on the large wing and tail feathers, although the contour feathers can be affected at times. The third way mites can affect the feathers is by invading the follicles, which results in the growth of a distorted feather. Only the follicles of the large feathers of the wing or tail seem big enough for the

parasite to enter, and usually only one or two feathers are affected. These are frequently very short or horn-like.

The diagnosis of this condition depends first on finding the mites. However, as these parasites are so common, one has to eliminate the causes of the feather disease before a diagnosis of the mites can be made. Treatment with any of the proprietary anti-mite drugs is effective.

Feather Cysts

It had not been anticipated that this condition would be such a common cause of feather abnormalities. The other unexpected feature of this condition was, that in the vast majority of the cases, the cyst or cysts had not been spotted by the fanciers. The birds had been sent to me because they had feathers which would not regrow.

Almost all the feather cysts occurred on the outer parts of the wing or the tail, although some were seen on the necks of birds. The cysts were of three types, the first and most common for 70% of the cases, was roughly spherical, up to 1.25cm (just over half an inch) in diameter. Cysts of this type were either very hard or slightly soft, depending on the thickness of the fibrous capsule. This capsule surrounded a core of yellow cheesy material, and the distorted remains of one or more feathers.

The second type was very similar to the first, but the surrounding skin was inflamed and thickened. As most of the lesions had not been noticed by the fanciers it was not possible to establish whether the damage to the skin came before or after the development of the cyst.

The third type was only seen on wings and accounted for fewer than 10% of cases seen. These were multiple and long and narrow in shape, lying side by side with one cyst corresponding to the follicle of one primary feather. They contained cheesy material as in the other cysts but in a proportion, a very short malformed feather was protruding from the tip of the cyst.

There was a very strong correlation between the first two types of cysts and marked buffness. Birds with this type of inherited plumage have a strong tendency to develop cysts. Unfortunately fancier's records were insufficiently detailed to show if cysts themselves are inherited, as is the case with the equivalent condition in canaries (lumps).

Research suggests buff feathering is linked to feather problems. There is no treatment for this condition other than surgical removal and it must be borne in mind that the birds will never regrow the feathers. It is probably not advisable to breed from birds with feather cysts or from their close relatives.

Stress Marks

Under this category we will look also at the absence of barbs and barbules, poorly formed barbs or barbules, and improperly shed sheaths. Stress marks are lines on the feathers where the barbules have not formed. Sometimes there is a line of weakness in the shaft at the same level, and the feather may break off at this point. If the barb and barbules are absent from a section of the feather, or if they are poorly formed, the feather does not hold together, giving the bird a tatty appearance. Improperly shed sheaths is a condition when the feather sheath is not shed in the usual way and instead persists over a greater part of the feather than is normal.

Occasionally, fully-formed feathers are totally enclosed in their sheath. In all three conditions the abnormality is most easily seen in the large wing and tail feathers.

In all these conditions, the changes seen in the feathers usually indicate something wrong in the bird's system in general. This needs to be attended to rather than concentrating on the feathers. With stress marks, the disturbance is general short lived, a matter of a few days to a week or thereabouts. The other conditions are associated with longer periods of illness, which can be quite mild, periods of stress or poor diet. When one or two birds in a flight are affected, this does not mean that the overall diet is unsatisfactory. Some budgerigars are very pernickety feeders and while different foods need to be supplied, it does not mean every bird will eat all of them. Provided the underlying problem is identified and corrected, the birds will almost certainly grow a set of normal feathers at the next moult.

Feather Dusters

This well known condition in which the feathers of the affected birds grow continually is noted very shortly after the birds begin to feather up. Such birds usually die at 6 to 8 weeks of age. the budgerigar that we had for a long time eventually died at 2 years. It grew contour feathers up to 24cm (9.5 in) long.

Some, and probably all, cases of this disease have a genetic inherited basis. There is a strong correlation between affected birds and buff feathering. A paper by Kevin Eatwell in *Budgerigar World* said that unaffected siblings of feather dusters are excessively buff and therefore may be retained for breeding. My view is this is not advisable.

The 8 conditions described above were responsible for two-thirds of the cases seen. The other 29 diseases accounted for the remaining third. These will form the second part of this report.

Table 1 - Main Feather Diseases found in a Survey of 198 affected budgerigars
n another survey of all types of psitticines are included for comparison purposes (Schmidt 1987)

Condition	ases	ıses	ıidt data%
Psittacine Beak and Feather Disease	42	21.2	24.4
Pulpitis	40	20.2	4.2
Mites	36	18.2	
Feather cysts	29	14.6	
Stress marks	16	8.1	
Barbs & Barbules missing or poorly framed	12	6.1	
Feather dusters	12	6.1	
Improperly shed sheaths	12	6.1	

http://www.funnyfarmexotics.com/IAS/pbfdflow.htm

PBFD Diagnostic Flowchart**

modified from the *Proceedings of the International Aviculturists Society, January 13 - 16, 1994*

Dedicated to the Birds of the World!

Interpreting the Results of the Psittacine Beak and Feather DNA probe test.

- **A. If Bird Has Dystrophic, Necrotic Feathers and you Test Blood for PBFD Virus using DNA probes:***
  - **1. If Positive: Suggests Active Infection**

    Management:

    If bird is from a breeding aviary: Bird should be removed and all areas that could be contaminated with feather dust from the infected bird should be repeatedly cleaned. If companion bird: Bird should not be exposed to other birds outside of the household and you should be aware that the virus can be transported to other locations on your clothes or in your hair. Be courteous of other birds and do not expose them. It should be noted that, occasionally, some PBFD infected Psittaciformes of South American descent have spontaneously recovered from the disease.

  - **2. If Negative: A feather biopsy (including the feather follicle) should be submitted for histopathologic examination.**

- **B. If Bird's Feathers are Normal and you Test Blood for PBFD Virus using DNA probes:***
  - **1. If Positive: Indicates that the bird has been exposed to PBFD virus and that the virus is present in the blood. The bird must be retested in 90 days. If the bird is negative when retested, it indicates that the virus was not detected in the blood cells. If the bird is still positive, it indicates that the bird is either clinically infected or that the bird is being repeatedly exposed to the virus. It should be noted that most birds that are exposed to the PBFD virus develop a transient viremia followed by an appropriate immune response that results in the bird clearing the infection.**

- **2. If Negative: Indicates that PBFD virus was not detected in the blood.**

***Testing available from:**

INFECTIOUS DISEASES LABORATORY
DEPARTMENT OF MEDICAL MICROBIOLOGY
COLLEGE OF VETERINARY MEDICINE
UNIVERSITY OF GEORGIA
ATHENS, GA 30602-7386
Phone: 706-542-5812 FAX: 706-542-5233

** Please feel free to duplicate and distribute.

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Small Animal Dept.
Athens, GA 30602

http://www.parrotsociety.org.au/articles/art_007.htm

PSITTACINE BEAK AND FEATHER DISEASE
(Psittacine Circovirus Disease)

by Dr Garry Cross
Senior Lecturer in Animal Health,
University of Sydney.

Psittacine beak and feather disease (PBFD) is a viral disease that can affect all psittacine birds (Parrots, Cockatoos and Lorikeets) and possibly, Doves. The virus attacks the cells of the immune system and those which produce feather and beak. Affected birds gradually lose their feathers and develop beak abnormalities. Because the virus attacks the immune system, affected birds succumb to infection by other diseases. The disease occurs in captive and wild birds and probably originated from Australia, for it is now found in psittacine birds in Europe and America.
Parrot Society member, Mr Garry Walsh recalls an incident when a sick Sulphur-crested Cockatoo infected by PBFD came to his property.
"This Sulphur-crested *was seen feeding in the 'chook yard' of our property. It was being harassed by several species of birds, including Magpies and Mickey (Noisy Miner) birds. The bird was in extremely poor condition, probably due to being unable to feed properly."*

Sulphur-crested Cockatoo, Cacatua galerita
Photograph by & courtesy of Garry Walsh, Westbrook Qld, Australia

Researchers at the University of Sydney's Department of Animal Health at Camden started their PBFD project in 1991. The aims were to develop tests to detect the virus and anti-bodies to the virus; determine the presence of the disease in wild psittacine birds; and develop a vaccine.
Drs Shane Raidal and Garry Cross have developed testing procedures which can determine whether or not a bird is immune to, or susceptible to infection by PBFD virus. All that is needed is a small sample of blood, a feather and some droppings. They have shown that birds with PBFD excrete the virus in large amounts in their feathers and droppings. Such birds are a source of infection to other birds.
They have demonstrated that the disease is present in wild psittacine birds in New South Wales and is widespread in wild populations. A vaccine has been developed which protects psittacine birds from infection by the virus. Two injections, four to six weeks apart, must be given. Birds vaccinated in 1991 are still resistant to challenge by the virus.
There is close liaison with Dr Peter Brown of the Department of Parks, Wildlife and Heritage of Tasmania in the Orange-bellied Parrot *(Neophema chrysogaster)* captive breeding programme. This programme is now producing birds for release into the wild. We have diagnosed the disease in one of these birds, and determined that it is present in the population. The vaccine is be in assessed in two of the birds. The Department of Animal Health is greatly interested in the survival of these birds.
Another 10 - 12 months work are required to complete tests on the vaccine (mainly on small psittacine birds) before it can be used on captive and wild psittacine birds. It has shown that the vaccine is very effective in Cockatoo nestlings and adults.

Make a Donation towards PBFD Research

If you would like to make a donation towards Psittacine Circovirus Disease (Psittacine Beak and Feather Disease Research) please forward payment via the Parrot Society of Australia Inc, P.O. Box 75 Salisbury Qld 4107, Australia. International donations should be forwarded as international money orders drafted in Australian or U.S. dollars. Recent Donations towards Psittacine Circovirus Disease Research (P.C.D.) as at 1/10/98 from members and the Parrot Society now total $2445.05 - many thanks to all those who have contributed towards this worthy cause - fund raising will continue.
Want more information on PBFD?
"PBFD has been confirmed in wild Galahs, Sulphur-crested Cockatoos, Corellas, Rainbow Lorikeets, Orange-bellied parrots, Rosellas, Ringneck parrots, Major Mitchell's Cockatoos, Gang-gang Cockatoos, King parrots, Swift parrots and many other species. There is evidence that PBFD occurs in wild Budgerigars, Red-rumped parrots, Yellow-tailed Black Cockatoos and Narethra Blue bonnets."

Sulphur-crested Cockatoo (Cacatua galerita) with PBFD
Showing feather loss

for all information contained on this site.

http://www.biobest.co.uk/diagnostics/pbfd.html

Psittacine beak and feather disease (PBFD)

Psittacine beak and feather disease (PBFD) is caused by a circovirus. It has a wide species range although it appears to be a natural virus infection of cockatoos in Australasia where it occurs in wild flocks. It has been known in wild cockatoos in Australia for many years and recently Ducorps cockatoos from the Solomon Islands have also been found to be infected. Old world parrots seem most prone to clinical disease although other psittacines can also be affected. A related virus can cause disease in pigeons and doves.

The virus
Circoviruses are small non enveloped icosahedral viruses which have circular single stranded DNA.

Incubation of the virus can be as short as three weeks, depending on the amount of virus the bird was infected by, the age and the health status of the bird.

Viral replication
Primary replication of the virus is thought to occur in the Bursa of Fabricius and the gastrointestinal tract lymphoid tissue. It is the destruction of the bursa which weakens the immune system and causes the bird to be more susceptible to secondary infections. Secondary viral replication then occurs in the liver and thymus.

Transmission
The PBFD virus is extremely infectious. It can be passed through a colony of birds in two ways:
Ingestion or inhalation from infected material such as faeces, feather dust, crop secretions and from infected surfaces or equipment.
Vertical transmission from hen to egg embryo.
Clinical Signs
The severity of the disease is dependant on the age, health, and breed of the bird. Young birds tend to get an acute form of the disease, or a peracute form if they are really unlucky. Older birds with weak immune systems are most likely to get a chronic infection, older birds with a healthy immune system may simply have a transient infection.

The symptoms vary depending on which form of disease the bird has. In acute cases the main action of the disease is to destroy the cells of the beak, feather and immune system. this causes abnormal feather loss and replacement feathers to be malformed, with lesions on the feather shaft. Skin cells grow more rapidly and the outermost layer of skin can become thickened. In peracute cases birds can suffer additional symptoms such as depression, anorexia, crop stasis and diarrhoea.

Symptoms presented by birds with a chronic infection can be observed through a succession of moults. Feathers stop growing shortly after emerging from the follicle. Birds with sub-clinical or transient infections may not present any symptoms.

Definition of Psittacine Beak & Feather Disease (PBFD)

(last upd/06/05/2007)

Psittacine Beak and Feather Disease (PBFD) is a deadly avian disease that may show no symptoms.

When a bird develops the illness, the disease is characterized by feather abnormalities, feather dystrophy and ultimately, death.

PBFD can affect birds of any age, but is more commonly seen in young birds from 0-3 years. Although many older birds can suddenly turn up positive for the virus, even though they had been clinically normal most of their lives. Especially disconcerting is the fact that this disease is EXTREMELY contagious. Viral particles are airborne. Dried feces & feather dust can adhere to clothing, nesting material, feeding formula, feeding utensils, nets, bird carriers, toys, & a myriad of other vehicles of possible transmission.

SYMPTOMS:

Obvious symptoms of PBFD are feather-dystrophy, possible bald patches on the head (this may exclude certain breeding-pairs, in which the male bird may pluck the female's head-feathers out.) If your bird has feathers missing elsewhere such as the chest, it's likely that the bird has plucked out it's own feathers, but PBFD should be ruled out before one can make such an assessment. Other signs are missing primary wing feathers and/or tail feathers, ragged looking or half-developed feathers, powdery feather-sheaths, or sheaths that don't dissapear within a few weeks. Infected birds will eventually lose most or all feathers and become extremely ill. Then the bird will die a very painful death, usually from secondary infection, or possibly failure of one or more internal organs.

DON'T ASSUME THAT YOUR BIRD IS HEALTHY BECAUSE IT'S FEATHERS LOOK PERFECT!

*Many birds (including young birds) that carry the disease will show NO SYMPTOMS. This means that even healthy looking birds should be tested because they still may be carrying the disease. They may initially test negative. There have been current discussions by breeders who have experienced this disease. They have explained that birds can test negative, possibly for several months. They explained that after certain previously diagnosed "negative" birds had been through some traumatic ordeal (such as shipping) the birds show a positive result. Stress is thought to "pop" the disease. This will cause the dormant virus to begin the process of shedding itself. They have assessed that stressful times are the best times to test your birds. For new acquisitions, right after shipping would be the ideal time to test them. On established birds, you may want to set up a temporary nestbox, which can cause stress, or take the bird for a bumpy ride in your car.

A negative test result does not prove that a bird is free of the virus because an incubation period of up to 4 weeks may be neccessary before the virus can be detected in the blood. Birds who test negative should be tested subsequently at 30 days after possible exposure, and if negative, retested again at 60 days.

Author; L. Wagner-Chambers

PBFD - A Few Myths and Facts

MYTH: - "PBFD is only limited to certain birds like cockatoos and parakeets." WRONG

FACT: - PBFD can infect *ANY* member of the psittacine species (any species of parrot and parakeet), including lories and lorikeets.

MYTH: - "PBFD is not really a problem any more. The disease was brought under control and eradicated years ago when a few cockatoos were found infected." WRONG

FACT: - PBFD has always been around and has always infected psittacine birds, however it was not until recently that people began to find out how they could test their birds for PBFD. In doing so, it was discovered that an *alarming* number of birds in the US were infected with this disease. The true urgency of this matter is just now being realized.

MYTH: - "But my bird *couldn't possibly* have PBFD because it has perfect feathers, and besides, it doesn't act sick." WRONG

FACT: - DON'T ASSUME THAT YOUR BIRD IS HEALTHY BECAUSE IT'S FEATHERS LOOK PERFECT.

Many birds (including young birds) that carry the disease will show NO SYMPTOMS. This means that even healthy looking birds should be tested because they still may be carrying the disease. They may initially test negative....(Please Click here for a more thorough description of the above statement. Look for the asterisk*.)

Testing Information

TESTING BY VETERINARIAN:

Having your bird tested by an *avian-veterinarian is the best method because blood is taken from a vein in the bird's neck, which makes it a cleaner sample than one taken from a toenail (which could be soiled with feces or food.) Be very careful that you choose an "avian" veterinarian, because among other risks, blood is extremely tricky to take from the neck, and only experienced avian vets should attempt this. (if something goes wrong during this procedure, the bird could be paralyzed or worse...)

Keep your bird's carrier/container well-covered in the veterinarian's waiting room, because there is a small risk of exposing your bird to other airborne avian diseases. And don't be afraid to express your concern that the examination-room be as clean as possible. (after all, it's your bird's life that could be at stake).

Prices for taking your bird to the vet; usually $35-$50 -office visit fee. Individual tests can run anywhere from $20-$60 dollars depending on the individual vet, and/or test requested. This can easily add up to over $100.

Don't force yourself to go to a vet that might not be experienced with birds, because you think it's the best thing for your bird. It may not be, and taking your bird to an unexperienced vet may be worse than taking him at all.

(Be sure the veterinarian you choose is an "avian-veterinarian" or one who regularly, successfully treats birds, and is well-versed in his/her knowledge of avian-diseases. Ask for references, and follow up on them.)

Many bird owners may not have the money to test their birds at the vet. Others may simply live too far away to drive to the vet's office. There is another option:

TESTING YOUR BIRDS AT HOME:

Another option that is perhaps a little more practical for most bird owners, is testing your birds at home. This method of testing requires that you hold your bird(s) gently, yet firmly

in a towel (much the same as you would do when clipping their wings or toenails. Be very careful not to squeeze bird's chest area too tightly, as this could restrict breathing. (If you're not comfortable holding your bird in this manner, you can ask for assistance from another person who is experienced in holding birds in this manner.)

Toenail clippers should be well-sterilized before this procedure, and you should use a seperate clean towel for each bird (unless you are testing a pair of birds). Toenail clippers should also be sterilized between each bird to prevent cross-contamination.

After this, the toenail is sterilized by rubbing alcohol, and the toenail is cut about 2/3 up from the tip, causing the "quick" to bleed. (you should have "quik-stop" or cornstarch handy in case a coagulent is needed to stop excessive bleeding.) -4 or 5 drops of blood are collected in a small vial, labeled and sent to an avian laboratory. Prices for these tests can cost anywhere from $18 to $25 dollars per test. Some (but not all) laboratories offer "bulk-rates", with the individual tests becoming cheaper according to how many tests you order at once.

We have provided a listing of laboratories on the next page for your convenience.

****8

Bird Marts - The Single Greatest Threat to Avian Health in the New Millennium!

Keeping birds happy and healthy is the number one concern of most bird owners. What toys will they like? What is his/her favorite foods and treats? Is the paint on its cage safe? What can we do to insure our birds live long, healthy lives? All these questions and more go through our minds on a daily basis. Birds cannot care for themselves, it is up to their human caretakers to do the best job possible. Cleaning cages daily, fresh foods and water, toys, play time and more all go into a 'normal day 'of caring for birds. The mistakes most well-intentioned birds owners make are most frequently from ignorance just not knowing - as opposed to stupidity.

Today a serious new health risk is threatening the lives of birds across the country. A very real health risk that has the potential for epidemic proportions. It goes by the simple name of 'Bird Marts'. Some people call them bird fairs, bird expos, bird shows and more. These innocuous appearing events are being put on by bird clubs, aviculturists and other people for profit and/or fund raising. What can possibly be so dangerous as a diverse groups of birds of varying ages brought together for one or two days? Everything! Anyone who believes these events are safe are not being truthful, or are simply ignorant of the facts.

People talk about quarantine - referring to the U.S.D.A. Bird Quarantine Stations that operated between 1971 and 1993 - how dangerous and awful it was. Thirty (30) days confined to cramped quarters sharing food, water, air, perches and more. Several sick birds could infect many more during the quarantine period resulting in high mortality rates. Many stations were less than honest. (Does anyone remember an honest importer?) And what happened when a station was having a disease outbreak with high mortality? They would have a sale! Drop the price and move them out. Take their problem and spread it around. Yes, it was terrible, but without it we wouldn't have the breeding stock so necessary for our operations today.

No matter how dangerous quarantine stations were, the still cannot match the dangers of today's 'bird marts'! These events have risen to become the single greatest threat to bird health we have ever encountered! How can a simple bird event be so life threatening? Is it nothing more than breeders coming together to sell their birds to the public? An unsuspecting public! At least in the quarantine stations, birds were exposed to other birds from the same areas of the world. The diseases present were also from the same area (indigenous) and many would have some natural immunity to it. In a Bird event' setting, birds are exposed to many other birds from all over the world (even though they may be domestically bred).

These birds, no matter how young, have the potential to bring their specific diseases and problems with them. In essence, any disease from anywhere in the world may conceivably be present and many are!

Years ago, concerned veterinarians and aviculturists began to notice a serious rise in sick and dead birds following many such events. Serious diseases such as pachecos, Polyoma, P.B.F.D., Chlamydia (Psittacosis), Wasting syndromes and more would suddenly appear from nowhere. We frequently discussed these diseases, their possible sources and patterns of outbreaks to recently held 'bird events'! It was obvious where the diseases were originating from, but could we prove it?

After many lengthy conversations with Dr.'s Dahlhausen and Radabaugh from Research Associates in Milford, Ohio, we came up with a plan to determine the incidence of diseases present at various events. Our goal was to determine what diseases were present, which events or seasons were safe and possible ways to insure the safety of birds. To determine which diseases were present, we would employ the use of their advanced technologies. DNA, PCR technology was selected due to its extremely sensitive and accurate parameters. This method, when performed properly, does not have false positives. If the test was positive, the organism was there. The bad news is a negative does not mean the organism wasn't there, it simply means we did not detect it!

Testing began in 1998. Diseases we tested for were limited to the 'Big Three' (Polyoma, P.B.F.D. and Chlamydia), as these were the only DNA probes available for environmental testing. Sample collection was simple. Using a sterile culturette swab (looks like a Q-tip with a long handle), an individual would simply rub the tip across a table top or floor in the 'bird event' area. Considering how small the culturette tip is, if diseases are detected, it reflects just how much viral or bacterial contaminants must be present. In order to prevent bias of events or people skills for collection, the procedures were the same for all. Swabs would only be taken from tables or areas where live birds were not being displayed. In other words, only vendors selling supplies, magazine subscriptions, clubs, raffle areas, non-bird pets and even people.

Eight different events were tested from around the country. Our goal was to be able to demonstrate these problems exist universally throughout the U.S., not just in Texas or Connecticut or Florida, etc. Following are the results of these events.

Approximate Date	PBFD	Polyoma	Chlamydia (psittacosis)
May 1998	Positive	Positive	Negative
May 1999	Positive	Positive	Positive
October 1999	Positive	Positive	Negative
October 1999	Positive	Negative	Negative
December 1999	Positive	Positive	Negative
March 2000	Negative	Positive	Negative
March 2000	Positive	Positive	Negative
March 2000	Positive	Positive	Negative

As you can see from the above chart, P.B.F.D. was detected at virtually every event but one. Polyoma was also detected at all events but one. Chlamydia, being a bacteria may be harder to detect or as the chart shows, may be less of a problem than the viruses present. If we had been swabbing tables with birds, the likelihood of detecting the presence of Chlamydia may be much greater.

Typical results from individual swabs collected at various 'bird marts' as follows:

Swab Collected From:	PBFD	Polyoma	Chlamydia
Local Bird Club without birds	Positive	Positive	Positive
Raffle Table without birds	Positive	Positive	Negative
Toy Exhibitor	Positive	Positive	Negative
Reptile Exhibitor	Positive	Positive	Negative
Interior Arena Walls	Positive	Positive	Negative
Random Person (human)	**Positive**	**Positive**	**Negative**

Although not accounted in either chart, several bird sales areas were also swabbed at various events:

Swab Collected From:	PBFD	Polyoma	Chlamydia
Event #1	**Positive**	**Positive**	**Negative**
Event #2	**Positive**	**Positive**	**Negative**

Additionally,a hand-feeding African Grey Parrot was presented for DNA, Blood Testing after purchase from a 'bird event' with the following results:

Species	PBFD	Polyoma	Chlamydia
African Grey	**Negative**	**Positive**	**Not tested for.**

Although all types of psittacines may contract or carry any of the 'Big Three' diseases, certain species appear to be hosts for them. P.B.F.D. appears to have found a home within lovebirds. Lovebirds can develop all major signs of the disease and survive. The relationship between Lovebirds and P.B.F.D. is still not fully understood. In colonies of lovebirds with P.B.F.D., infection rates of up to 100% are common.

Budgies (parakeets) have found a way to co-exist with Polyoma Virus even though infection rates are very high. Nestling mortality, feather abnormalities - crawlers- and acute juvenile deaths are all typical of this virus. Polyoma Virus perpetuates itself with each successive generation and may be extremely difficult, if not impossible, to eliminate from breeding colonies.

Cockatiels appear to be the group of parrots most able to survive with endemic Chlamydial infections. Only recently has a reliable test for Chlamydia been available. We can now accurately screen these birds for infection as opposed to utilizing non-specific tests such as protein electrophoresis, B-ELISA or nebulous clinical signs which mimic many other diseases.

Not only do these three groups of birds comprise the greatest reservoir of the three major diseases, they also comprise the greatest number of birds present at typical bird events. It is hardly a surprise that the 'Big Three' diseases continue to remain so prevalent today!

So why all the concern over 'bird events'? First and foremost, these deadly disease causing organisms are found throughout the entire 'bird event' area. It is in the air, on hair, clothing and shoes, table tops, toys, supplies, cages, foods, virtually everything in the area! People and birds may not have entered with any of these diseases, but they definitely leave with them! As Dr. David Phalen of Texas A & M stated, 'people come looking for bargains, real or perceived, but the bad news is the diseases are free'! People take them wherever they go, to their favorite pet store, their friends homes and home to their own birds. Their role as courier has gone totally unnoticed!

Many people are unaware that several of these organisms, especially P.B.F.D. and Polyoma Virus, are extremely hardy. It is believed these viruses can remain stable for upwards of one year and still be infective. The items purchased from events bring these viruses into homes waiting for an opportunity to cause illness. Furthermore, these organisms can be transported from 'bird mart' to 'bird mart'. The

diseases present could easily come from a previous show and can certainly be carried to future shows on displayed merchandise.

Reflecting back on quarantine, several hundred birds may be exposed to one disease and most often a disease from their region of the world. A disease they may have some natural immunity to. When we attend a 'bird mart', hundreds of birds are exposed to numerous diseases. Diseases many have never seen before, with no natural protection. To complicate matters, several diseases together may produce a fatal illness, when individually they may be harmless (de, P.B.F.D. and Polyoma exposure to mature eclectus parrots can result in death). Some exhibitors proudly display signs stating their birds are protected by a Polyoma vaccine. Most psittacines at these shows are far too young to be protected by a vaccine. Whether or not the vaccine offers any protection from Polyoma is still up for debate, but it is highly unlikely the vaccine would have any effect against P.B.F.D., Chlamydia or any other viruses and bacteria present.

We must take this a step further. After attending a bird event, hundreds or thousands of people now leave to expose their bird(s) and homes or worse yet, their friends birds and birds in their favorite pet store. A single 'bird mart' can expose thousands of birds overnight! Remember we are talking about one 'bird mart'. Now add to those numbers the amount of 'bird marts' taking place across the country. Thru these 'bird marts', we perpetuate these and other deadly diseases. Bird events help insure the future ability of these organisms to survive and infect subsequent generations. This problem can only get worse!

The evidence is here in black and white. The potential for generating and spreading fatal avian diseases is unsurpassed anywhere in the world as it is at 'bird mart' type events. We have not found a single 'bird event' free of these diseases! With the methods employed for detection, there is no room for doubt of the extreme seriousness of these events. It is unconscionable to believe that:

A) Anyone who cares for birds would hold any event where young birds are present for display or sale.

B) Anyone would attend any event to purchase any items for birds.

C) Any person would ever attend any event with such a high degree of fatal organisms waiting to be transmitted to healthy birds.

D) Anyone would support or recommend any such event.

It is time to wake up to the dangers of 'bird marts' and the damage they create. The facts are conclusive, many diseases are present. Once DNA probes have been perfected for diseases such as Herpes Virus, Wasting Syndrome, P.M.V., Avian T.B. and more, testing may reveal how many more of these organisms have been present and infecting birds. Help prevent present and future illnesses and deaths. DO NOT ATTEND THESE FUNCTIONS! Stop supporting all events of this nature until one can be safely organized. When people attend these events, they risk the lives of all birds - both present and future generations - theirs, yours and mine! In the end, the ultimate losers are always the birds. Why must they suffer with their lives?

Ernie Colaizzi

CIRCOVIRUS IN LORIKEETS

Point Defiance Zoo & Aquarium, Tacoma Washington.
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ABSTRACT

Free flight lory and lorikeet aviaries in zoos are growing in popularity. Through quarantine screening and treatment of affected lories and lorikeets (members of the sub-family Loriinae and collectively referred to as lories) it is becoming increasingly apparent that psittacine beak and feather disease (PBFD) is a viral disease in these birds that can prove challenging to detect and manage. The following paper will provide a review of the disease and a case report from a lory exhibit that documents acute and chronic cases, possible carrier states, recovery from PBFD, and a management strategy.

INTRODUCTION

Circovirus, the cause of PBFD, is the smallest known virus capable of causing disease in birds.*7 Fourteen to sixteen nm in diameter, this non-enveloped virus contains a single strand of circular deoxyribonucleic acid (DNA). Though previously known to only affect psittacines, circovirus with antigenic variations have been documented causing similar disease in doves,*5 pigeons,*11 and ostrich.*3 To date, lesions suggestive of PBFD have been described in at least 53 species of psittacines. Descriptions of feather changes in wild red-rumped parrots (Psephotus sp) in the Adelaide hills of Australia in 1888, are thought to be the first report of PBFD.*1 The first clinical cases were reported in the early 1970's in Australian cockatoos,*6 and with the booming bird trade it has spread around the world.

Clinical features of PBFD include one or more of the following:

1) abnormal feather loss (large numbers or easily removed),
2) presence of abnormal feathers (thickened or constricted feather shafts, shafts with fault lines or clotted blood) starting with powder down and contour feathers, and then the primary and secondary remiges, retrices and crest feathers,
3) abnormal feather color, or
4) beak abnormalities (beak elongation, palatine necrosis and ulceration).
The incubation period can be as short as 21 d or as long as many mo to yr depending upon the dose of virus, age of bird, stage of feather development at the time of infection, and status of the bird's immune system.*4
For example, birds infected in the process of developing feathers may demonstrate signs of PBFD sooner than birds infected after a moult who may not develop abnormal feathers for mo. Transmission of the virus is horizontal (ingestion or inhalation of feather dust, feces, and crop secretions) or vertical (egg transmission).

***8 Peracute, acute and chronic disease patterns will vary in their presentation. In peracute cases, birds may die before feather abnormalities develop. These cases are most commonly seen in neonates that show signs of septicemia accompanied by pneumonia, enteritis, rapid weight loss and death. In this disease pattern necrosis and atrophy of the bursa and thymus may be the only microscopic changes seen. The acute pattern of PBFD is characterized by clinical depression, crop stasis, diarrhea and sudden changes in developing feathers of young birds.*2,*8,*10 The most dramatic acute presentation is observed in chicks undergoing feather development. The chronic disease pattern presents with symmetric, progressive appearance of abnormally developed feathers during successive molts. Feather abnormalities described in the first paragraph, as well as short clubbed and deformed curled feathers can be seen. In this situation the birds may be bright, active and have good appetites. With symptomatic care chronically infected birds can live mo to yr, but often succumb to a secondary infection because of the virus' destruction of the bursa and subsequent immunosuppression. It should be noted that some birds may spontaneously recover,**

though, acutely affected birds tend to have a greater chance than chronically affected birds. Spontaneous recovery has been reported in budgies, lorikeets and lovebirds.*8

Practical methods to diagnose and manage PBFD have only recently been developed. The application of viral specific DNA probe technology to identify PBFD virus has been an important development in the management of this disease. Using the polymerase chain reaction (PCR) PBFD antigen can be identified in blood, affected feathers and environmental swabs. With special attention to standardization of testing, quantitative evaluation of the test can distinguish high versus low viral load. When evaluating the quantitative results in series, transient versus progressive viral infection can be distinguished.*2 The high specificity and sensitivity of this test make it the best available test for PBFD. There is no effective treatment for PBFD. Many different anecdotal reports discuss the use of a variety of immunostimulants with mixed results.*12 A vaccine for PBFD, though developed in Australia in the mid 1990's, is still in the research and development stages both in Australia and the United States.

CASE REPORT

In 1998, Point Defiance Zoo & Aquarium (PDZA) opened a summertime free-flight lory aviary. Over a 2 1/2 yr period this group grew from 24 to 49 birds representing 12 species of lorikeets and lories. During that time the collection experienced some cases of acute and chronic disease patterns of PBFD, and saw some infected birds recover, demonstrated by conversion from positive to negative status via PCR and return to normal feather condition.

PDZA began to acquire lories in February of 1998. These birds were obtained from a variety of breeders in a staggered fashion as quarantine would allow. Most of the birds were in the process of being weaned so that they could be acclimated to being around and eating from people. On the ninth day of quarantine for the first group of birds (n = 7), a red lory (Eos bornea bornea) from this group was found dead. This bird had arrived thin, in poor condition and was started on antibiotic and antifungal treatment. Histopathology revealed basophilic inclusions of the bursa suggesting PBFD as the cause of a primary immunosuppression with subsequent opportunistic parasitic infections leading to the death of the bird. Tissues submitted for in situ hybridization confirmed the diagnosis of PBFD within the bursa. The rest of the group were given quarantine physical exams, and had samples taken for, fecal ova and parasite exams, choanal + cloacal cultures, blood chlamydia PCR, PBFD PCR and, in some cases, a complete blood count and abbreviated serum chemistry testing depending upon the size of the bird. If dystrophic remiges, retrices, powder down or contour feathers were noted, feather follicle biopsies were also submitted for histopathologic evaluation. All six birds were negative on PBFD PCR, yet five of the six were positive for PBFD on feather follicle biopsy using DNA in situ hybridization. Euthanasia was declined and the birds were sent back to the breeder.

Concurrently, a total of 15 new birds arrived and were housed in a separate quarantine room from the first group. Quarantine exams were performed within the first wk of their arrival and all birds were found to be PBFD PCR negative. Despite these results, 9 d after the exams and bloodwork, two black-capped lories (Lorius lory lory) and one swainson lorikeet (Trichoglossus haematodus mulucanus) from this group shed an alarming number of remiges and retrices. DNA in situ hybridization of one of the black-capped lory feathers was positive for PBFD. Two weeks into the quarantine a red lory from this group died without symptoms. In situ hybridization of tissue samples were negative for PBFD and positive for polyoma virus. Contact was made with an out-of-state veterinarian who had documented the recovery of lories from PBFD by following PBFD PCR status and feather condition. Since PDZA was not anxious to euthanize these birds or expose incoming juveniles, this group of birds (n = 14) was sent to the out-of-state veterinarian. The birds were followed over a 19 mo period utilizing PBFD PCR. During that time, 12 of 14 birds became PBFD PCR positive. Ten of the 12 PBFD PCR positive birds then converted to negative by October of 1998. Despite their negative status, seven of the 14 lorikeets died succumbing to secondary bacterial infections that were the result of immunosuppression and the other seven remained negative for PBFD PCR and returned to normal feather condition.

In May 1998, before receiving more new birds, all quarantine and exam room surfaces and equipment were disinfected with sodium hypochlorite at a 1:30 dilution. This was followed by copious rinsing with water, as the circovirus is eliminated more by dilution than by destruction.*9 Because of the similarities in ultrastructure and DNA composition to chicken anemia virus, PBFD virus is thought to have the same environmental stability and ability to remain viable in the environment for two to three years.*13 As sometimes recommended in severe PBFD outbreaks, previously contaminated walls were painted. Following this disinfection effort, environmental swabs were submitted for DNA PCR and found negative for PBFD. Despite the negative tests, these rooms were not used and new isolation exam and quarantine rooms were established. A new PBFD testing protocol was developed which required that 1) incoming lorikeets be housed by arrival groups in quarantine, 2) handlers dress in booties, caps, gowns and gloves until the birds tested negative twice via PBFD PCR at least 60 d (though preferably 90 d) apart and that 3) only then would the birds be released into the exhibit for the summer.*8 In the fall the birds were taken off exhibit into off-site winter housing.

In March of 1999 the whole process was repeated. One black lory (Chalcopsitta atra) had been of particular interest the previous year because of its unusual coloring. As a juvenile the yr before it had a small number of red feathers, which wasn't considered unusual for a black lory chick. In the winter of 1998-99, though, there was a marked increase in the number of red feathers. Abnormal feather coloration can be a sign of PBFD. African grays infected with PBFD have had normally gray feathers grow in red *4,*8,*10 and princess parrots (Polytelis alexandrae) have had their green feathers become yellow.*4 In February of 1999, this black lory lost primary feathers from both wings and tail. Incoming feathers had a clubbed appearance. The black lory was tested and found to be positive for PBFD, even though it had tested negative the year before via PBFD PCR by two labs and feather follicle biopsy DNA in situ hybridization.

In addition to the black lory, its cagemate and two birds from the adjacent cage (1 duyvenbode lory (Chalocopsitta duivenbodei duivenbodei) and 2 forstens lorikeets (Trichoglossus haematodus forsteni)) tested positive via PBFD PCR. A wk later a blue streaked lory (Eos reticulata), another cage mate of the black lory, died after 2 d of appearing fluffed. On necropsy a multifactorial crop lesion was thought to be suggestive of immunosuppression. Later the crop, proventriculus, small intestine and pancreas were all positive for PBFD on in situ hybridization. All other birds in the collection were isolated and tested twice via PBFD PCR 60-90 d apart and found to be negative. The twice negative birds were released into the exhibit. Following strict isolation protocol, the PBFD positive birds were isolated off-site and cared for by a person who didn't work with the exhibit birds.

Repeat PBFD testing of the four positive birds 90 d later demonstrated that they were all still positive. Though the forstens lorikeet was in perfect feather at this time, the black lory, second forstens, and duyvenbode lory had obviously dropped remiges and retrices, so the birds were euthanized and necropsied. Blood and tissue samples from these birds were sent to the clinical laboratories that had been analyzing our samples so that they might characterize the virus and enhance the specificity of their own probes/tests. Because of some inconsistent PCR laboratory results it was thought that the PBFD virus infecting these lorikeets might be a genetic variant of the more commonly isolated PBFD virus, causing it to be missed by some PCR tests. This thought was supported by a publication from Australia which recently documented variation in the genome of the PBFD virus isolated from eight different species of affected psittacines, with the isolate from the rainbow lorikeet (Trichoglossus haematodus) having the greatest variation.*14

The winter quarters where the four PBFD positive birds had been housed prior to detection of PBFD was destroyed. A new foundation was poured, building constructed (disinfected and painted) and environmental swabs tested by PBFD PCR. At the end of the summer of 1999 when declared negative for PBFD via PCR the new winter quarters were again occupied by lorikeets now PBFD PCR negative for a third time.

Testing has begun again for the year 2000. Except for some tattered retrices, all of the birds are in good feather condition. It is desired to keep a closed breeding colony. One surprise is that one of the birds, which tested negative, five times by the PBFD PCR was shipped out this winter to another aviary. Within

28 d of arrival and placement in a room shared with other lorikeets, it died acutely without symptoms of PBFD. The primary necropsy lesions were hemorrhage of liver, kidney, mesenteries as well as, hepatic necrosis. Though suggestive of a viral infection, PBFD wasn't a prime suspect. Microscopically no viral inclusions were visible. Subsequent in situ DNA hybridization of tissues was negative for Pacheco's virus, adenovirus, herpes virus and polyoma, but positive for PBFD. Attempts are being made to decipher the genome of the offending PBFD virus and compare it to the one that has infected some of our birds.

DISCUSSION

PBFD is a highly contagious and debilitating disease. Until an effective vaccine is developed, diligent screening and management programs must be put in place. PBFD PCR is a valuable component of these programs, but it must be used with clinical observations and DNA specific tissue testing to provide the most comprehensive screening. The ability to detect carrier states is still in question. The possibility of genetic variations of the circovirus is becoming more apparent and may explain some of the inconsistencies of PBFD PCR testing of lories and lorikeets.

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