# Pathobiology and Testing Recommendations for Psittacine Circovirus 2 in Lories

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The family Circoviridae includes several host-specific single stranded deoxyribonucleic acid (DNA) viruses that infect numerous species of birds and mammals, including humans.[1–7] The first pathogenic circovirus to be described is the cause of psittacine beak and feather disease (PBFD).[2] The original PBFD virus, now called psittacine circovirus 1 (PsCV-1) causes transient, subclinical infections in most psittacine birds, but can cause progressive feather dystrophy in some individuals that fail to mount an appropriate immune response.[1] Old World psittacine birds that develop PsCV-1 associated feather abnormalities usually die.[1] A few New World psittacine birds with PsCV-1 associated feather abnormalities will recover.[1] A polymerase chain reaction (PCR)-based assay and probes used for in situ hybridization, both developed by the Emerging Diseases Research Group at the University of Georgia, College of Veterinary Medicine, have been the mainstay of PsCV-1 diagnosis and control for the past decade.[1]

Three years ago, a circovirus variant was demonstrated in a group of lories with dystrophic feathers.[7] The blood of these lories contained a circovirus nucleic acid sequence that was detected using generic circovirus primers but not detected using primers specific to PsCV-1.[7] This circovirus variant has been named psittacine circovirus 2 (PsCV-2). Sequence analysis has shown that the genome of PsCV-2 is differs approximately 10% from that of PsCV-1 (Ritchie, et al, unpublished data, May, 2002). A PCR-based assay has been developed by the Emerging Diseases Research Group to differentiate between hematogenous PsCV-1 and PsCV-2 nucleic acid.

Several observations from the originally described outbreak of PsCV-2 in lories are considered clinically important: 1) the histologic lesions in affected lories were generally less severe than lesions in birds with PsCV-1 associated disease, 2) some lories with dystrophic feathers reportedly recover and 3) the period of hematogenous nucleic acid detection appears to be more prolonged with PsCV-2 than is typical for PsCV-1. Based on these observations, a study was designed to determine the pathobiology, PCR-based screening recommendations and prognosis for both lories with subclinical PsCV-2 infection and for lories with PsCV-2 associated feather abnormalities.

The blood from 18 lories was screened for circovirus nucleic acid. Using strain-specific assays, 9 of these lories were nucleic acid positive for PsCV-2 and all 18 were negative for PsCV-1. The 9 positive lories were removed from the collection and sent to the Emerging Diseases Research Group for subsequent testing and evaluation. Based on studies conducted in these lories, it was determined that: 1) PsCV-2 nucleic acid can persist in the blood of lories with subclinical infection for at least 6 months; 2) some lories with dystrophic feathers can

develop an appropriate immune response and recover; 3) circovirus-associated inclusion bodies can remain sequestered in affected feathers of birds that have otherwise cleared a systemic infection; and 4) PsCV-2 nucleic acid can be detected in the immediate environment (including the surface of feathers) where birds with feather abnormalities are housed.

The fact that minor variations in the genome of a virus can result in significant changes in pathobiology is not uncommon. For example, the originally characterized porcine circovirus (designated porcine circovirus-1 [PCV-1]) was recovered incidentally from a porcine kidney cell line (PK-15) and was described as non-pathogenic.[3] More recently, a second porcine circovirus (PCV-2) has been implicated in the postweaning multisystemic wasting syndrome (PMWS) of pigs. The genomes of PCV-1 and PCV-2 are between 69% and 76% homologous, yet these variants are associated with disparate clinical outcomes.[4,5]

Parvoviruses provide another example of varied clinical disease from related viruses. Canine parvovirus (CPV) type-2 emerged in the mid to late 1970s as a variant of a closely related carnivore parvovirus, possibly feline panleukopenia virus (FPV). Between 1979 and 1985, 2 variants (type-2a and type-2b) of the originally described canine parvovirus were identified. While these variants primarily affect dogs, they can also infect and cause disease in cats. The variability in host susceptibility between CPV and FPV is controlled by 2 or 3 region differences in the sequence coding for the capsid protein of these viruses.[8,9]

Based on the differences in pathobiology associated with PsCV-1 and PsCV-2, PCR-based screening assays are recommended to differentiate between these 2 variants and feather/follicle biopsies, in conjunction with in situ hybridization using circovirus specific DNA probes, be used to confirm circovirus-associated disease. While most psittacine birds with subclinical PsCV-1 will clear viral DNA from their blood within 90 days of initial detection, PsCV-2 DNA may persist in the blood of subclinically infected lories for at least 6 months. To date, mixed PsCV-1 and PsCV-2 hematogenous DNA have been detected in some psittacine birds, but PsCV-2 hematogenous DNA alone has only been detected in lories. Variations in clinical outcome, if any, in birds that have been infected by both variants remain to be determined.

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