Psittacid herpesviruses and mucosal papillomas of birds in Australia

Fact sheet

Introductory statement

Psittacid herpesvirus-1 (PsHV-1) has not been reported in wild Australian parrots. Psittacid herpesviruses have been identified in captive green-winged macaws (Ara chloroptera) in Australia. The four genotypes of PsHV-1 are the etiologic agents of Pacheco’s disease (Tomaszewski et al. 2003). Pacheco’s disease is an acute rapidly fatal disease of parrots that has caused significant mortality events in captive parrot collections. Many species of Australian parrot are among those that are susceptible to PsHV-1 infection and disease (Phalen 2006). Subclinical infections result in birds that remain infected for life. These birds are then sources for future outbreaks (Tomaszewski et al. 2006). Parrots subclinically infected with PsHV-1 genotypes 1, 2 and 3 may develop mucosal papillomas (Styles et al. 2004; Styles 2005). Mucosal papillomas have been detected in green-winged macaws imported into Australia (Gallagher and Sullivan 1997; Roe 1997; Vogelnest et al. 2005) and they have been confirmed to contain PsHV-1 (Raidal et al. 1998; Vogelnest et al. 2005). Although there is no evidence of PsHV-1 in wild Australian parrots (Raidal et al. 1998), the establishment of PsHV-1 in Australia poses a significant risk to captive parrots and a potential risk to wild Australian parrots.

Aetiology

Family (Herpesviridae), subfamily (Alphaherpesvirinae) genus (Iltovirus).

Natural hosts

Psittacine birds (parrots) are both the natural host and the most susceptible to disease. Whether infection proceeds to a carrier state, the development of mucosal papillomas, or Pacheco’s disease depends on the genotype of the virus and the species of parrot that it infects (Styles et al. 2004; Phalen 2006). These viruses have also been isolated from a very limited number of passerine species, but its presence in
passerines is likely to be rare (Tomaszewski et al. 2004). A Pacheco’s-like disease has also been described in toucans but this virus has not been characterized (Tomaszewski et al. 2003).

Pacheco’s disease has been reported in several Australian bird species held captive in overseas collections. Species affected included: the sulphur-crested cockatoo (*Cacatua galerita*); gang-gang cockatoo (*Callocephalon fimbriatum*); galah (*Cacatua roseicapilla*); green rossella (*Platycercus caledonicus*), Eastern rosella (*P. eximius*), pale-headed rosella (*C. adscitus*), Port Lincoln parrot (*Barnardius zonarius*); yellow rossella; regent parrot (*Polytelis anthopeplus*); princess parrot (*P. alexandrae*); fig parrot (*Cyclopsitta* sp.); eclectus parrot (*Eclectus roratus*); cockatiel (*Nymphicus hollandicus*); and red-rumped parakeet (*Psephotus haematonotus*) (Gaskin et al. 1978; McCluggage 1985; Arnold 1990; Gravendyck et al. 1998; Tomaszewski et al. 2003). PsHV-1 has also been suspected as the cause of disease in a zebra finch (*Taeniopygia guttata*) (D. Phalen pers. comm. 2005).

There is no age or sex predilection.

**World distribution**

Although not proven, it is highly likely that this virus has co-evolved with neotropical (South American) parrots. Pacheco’s disease and subclinically infected birds have been documented in North America, Europe, the Middle East, Japan, New Zealand (in quarantined birds), and Australia (Vogelnest et al. 2005; Phalen 2006).

**Occurrences in Australia**

Psittacid herpesvirus-1 (PsHV-1) has not been reported in wild Australian parrots.

Internal papilloma disease was first reported in Australia (southern Queensland) in 1997 in two captive green-winged macaws (*Ara chloroptera*) imported from the United Kingdom (Gallagher and Sullivan 1997). PsHV-1 was first confirmed in Australia (Sydney, NSW) in 2004, also in two captive imported green-winged macaws. Both birds were part of the original consignment that had been imported from the United Kingdom in 1993. A third green-winged macaw, an offspring of this pair, was also positive and the papilloma contained PsHV-1 genotypes 2 and 3, the same genotypes identified in the parents (Vogelnest et al. 2005).

**Epidemiology**

Many factors will determine what impact the introduction of PsHV-1 will have in a collection of parrots. These include; the genotype of the virus, the species of parrots in the collection, the level of immunity already in the collection, the housing density of the parrots and if they are housed indoors or out-of-doors. For example, Amazon parrots are highly susceptible to all 4 genotypes of PsHV-1 and densely housed collections and indoor collections are more prone to outbreaks of Pacheco’s disease (Tomaszewski et al. 2003; Phalen 2006).

Experimental infections have shown the incubation period to range from 5 to 10 days. Without treatment, birds that develop Pacheco’s disease die (Godwin et al. 1982). However, virus replication is rapidly inhibited by acyclovir and birds in the early stage of infection can be saved with treatment. All treated birds and all subclinically infected birds become carriers (Phalen 2006).
Mucosal papillomas develop in some species of subclinically infected parrots, particularly macaws, Amazon parrots and, less frequently, conures in the months following infection with PsHV-1 genotypes 1, 2 or 3. Why one species of parrot develops papillomas and another infected with the same virus does not is not known (Styles et al. 2004).

Recently infected birds shed virus in oral secretions and droppings. High concentrations of virus can be found in the blood and all tissues. Most infections occur when contaminated material is ingested.

The potential range of carrier species of PsHV-1 is not known. The majority of birds shown to be subclinically infected are neotropical parrots, but subclinical infections have also been documented in African and captive Australian species overseas (Tomaszewski et al. 2006).

**Clinical signs**

Pacheco’s disease has few clinical signs. Most birds are found dead. When signs occur, birds stop eating and are less active. Yellow or green stained urates reflect the severe liver damage that these birds are experiencing (Phalen 2006).

**Mucosal papillomas:** Mucosal papillomas can occur in the cloaca, oral cavity, and less frequently in the crop, oesophagus, proventriculus and ventriculus. Signs include straining to defecate, frank blood in the droppings and prolapse of papillomatous lesions from the cloaca. Oral papillomas rarely cause signs. When disseminated papillomas are found in the oesophagus and crop or proventriculus, birds may experience a chronic wasting disease with or without regurgitation. Mucosal papillomas are typically raised, pink and have a cauliflower-like surface. They can be found locally or diffusely in the cloaca and may ulcerate and bleed. Lesions in the mouth are most commonly found along the margins of the choanae and at the base of the tongue. Lesions can wax and wane, disappear completely or become progressive (Styles et al. 2004; Phalen 2006).

**Bile duct and pancreatic duct carcinomas** containing PsHV-1 DNA are fairly common occurrences in parrots that have mucosal papillomas containing PsHV-1 genotype 3. Birds with bile duct carcinomas will often exhibit signs of chronic liver disease such as weight loss, an overgrown beak, and poor feather quality. When they do finally go into liver failure, signs are generally not specific. Pancreatic duct carcinomas are much less common and clinical signs are vague and not specific. Bile duct carcinomas develop in the months and years following the onset of mucosal papillomas (Styles 2005).

**Diagnosis**

Subclinically infected birds can be detected with a nested PCR or real time PCR assay for PsHV-1 of swabs obtained from the oral cavity and cloaca (Tomaszewski et al. 2006).

Gross observation of the papillomatous lesions is sufficient for diagnosis of mucosal papillomatosis. These lesions will also be positive for PsHV-1 DNA. Biopsies will reveal the characteristic papillary changes to the mucosa.

In the live bird diagnosis of bile duct carcinomas can only be made with a liver biopsy.
Clinical pathology

It is rare that a bird with Pacheco’s disease survives long enough to have blood collected. Experimentally infected birds develop a leukopenia and have marked elevations in their serum aspartate aminotransferase concentrations (Godwin et al. 1982). Birds with bile duct carcinomas have been observed to have marked elevations in their gamma glutamyl transverse serum concentrations (Phalen 2006).

Pathology

Pacheco’s disease

Gross lesions may or may not be present. When present, there is usually enlargement of the spleen and liver. The liver be diffusely pale yellow and appear as though there is a diffuse lipidosis. Because most birds die suddenly, they are typically well muscled and have significant body fat.

A moderate to marked hepatic necrosis is found in nearly all birds with Pacheco’s disease. Often the only hepatocytes that are spared are the ones surrounding the portal tracts. Intranuclear eosinophilic inclusion bodies may be common or rare. Splenic necrosis is also common and inclusion bodies are regularly found in splenocytes. Pancreatic necrosis and necrosis of the intestinal and crop mucosa occurs less frequently. Inclusion bodies are commonly seen in these lesions (Schmidt et al. 2003).

Figure 1: Cloacal papilloma: green-wing macaw (Photo Australian Registry of Wildlife Health)

Mucosal papillomas

- See Clinical signs for gross lesions.
- Bile duct carcinomas are multifocal and coalescing. They will often replace much of the liver before birds die. They do not metastasize. Pancreatic duct carcinomas are grey, nodular, and coalescing. There may be atrophy of the associated exocrine pancreas.
- Microscopically mucosal papillomas are made up of multiple fimbriae with a variably wide to narrow base. Each fimbria is composed of a fibrovascular core surrounded by a pseudostratified or stratified cuboidal to columnar epithelium. The lesions may be ulcerated. Lymphoplasmacytic infiltrations of the fibrovascular cores occur intermittently (Schmidt et al. 2003; Phalen 2006).
Differential diagnoses

Pacheco’s disease:
- Acute systemic bacterial infections (e.g. pasteurellosis from a cat bite)
- Other acute viral infections (e.g. avian polyomavirus in nestling birds)
- Hepatic lipodisosis.

Mucosal papillomas:
- Other neoplasia of the cloaca and oral cavity
- Bacterial cloacitis.

Bile duct carcinomas:
- Other liver tumours
- Cirrhosis.

Laboratory diagnostic specimens

PsHV-1 from birds with Pacheco’s disease
- For histopathology formalin-fixed tissues (liver, spleen, pancreas, intestine, crop)
- For PCR (0.1 ml) blood, liver, spleen (submit frozen). Swabs of liver or spleen (ship chilled overnight).
- For culture in chicken fibroblasts, liver or spleen (submit frozen). (Include collection, volume/ mass, processing and transport requirements).

Subclinically infected birds
- Swabs of the oral cavity and vent, shipped chilled overnight
- PsHV-1 DNA can also be found in blood in some birds, however, oral and cloacal swabs are better sources of PsHV-1 DNA.

Laboratory procedures

Pacheco’s disease is readily confirmed by histopathology of the appropriate tissues. PsHV-1 genotypes 1, 3 and 4 can be cultured in chicken embryo fibroblasts from spleen or liver. PsHV-1 genotype 2 is less likely to be isolated in this system (Tomaszewski et al. 2003).
PCR assays have been developed that can detect PsHV-1 in samples from birds that have died from Pacheco’s disease and in swabs from the cloaca and the oral cavity. Nested PCR or real time PCR will generally be necessary to detect samples from subclinically infected birds (Tomaszewski et al. 2006). PCR testing for PsHV-1 is provided by request at the University of Sydney (dphalen@camden.usyd.edu.au).

**Treatment**

Birds with Pacheco’s disease can be treated with acyclovir (80-100 mg/kg three times a day for 10 days). These birds will, however, not be cured of the virus infection and will become carriers of the virus. Carriers and those with overt mucosal papillomas are not impacted by treatment with acyclovir. Surgical resection of papillomas is a palliative treatment (Phalen 2006).

**Prevention and control**

Subclinically infected birds are readily detected by PCR, positive birds can then be held in isolation. There is strong evidence of parent to offspring transmission after hatch, but no evidence of vertical transmission through the egg. Therefore, positive birds should not be allowed to raise their own chicks. There are three principal serotypes of the virus and infection of one does not protect against infection with another. A polyvalent vaccine would be necessary to prevent infection with all serotypes and a vaccine for this virus in not available in Australia (Tomaszewski et al. 2006).

PsHV-1 is an enveloped virus and is readily inactivated by commonly used disinfectants. A similar virus, the infectious laryngotracheitis virus is inactivated by temperatures of 58 C for one hour, but may persist for many days in biological tissues (Guy and Garcia 2008).

**Surveillance and management**

Wildlife disease surveillance in Australia is coordinated by Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia’s States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au. One of the events involving PsHV-1 (2004) is recorded in the National Wildlife Health Surveillance Database (eWHIS 60 - www.wildlifehealthaustralia.com.au).

There are currently no formal surveillance programs for PsHV-1 in birds in Australia. Any cases of PsHV-1 in wildlife would fit within the general surveillance category of “Interesting or unusual cases” and should therefore be captured by WHA wildlife coordinators as part of Australia’s general wildlife surveillance system (www.wildlifehealthaustralia.com.au).

Bird breeders, particularly those that breed higher risk species, should be encouraged to test their birds. Positive birds should be isolated. Owners of positive birds should not be compelled to destroy them, if they can show that they are permanently identified and can keep them isolated from other captive birds and from wild birds.
Research

It is known that PsHV-1 has entered Australia in green-winged macaws. It is not known if it has entered Australia in any other parrot species and it is not known how far it has disseminated in avicultural collections. Surveys of the high-risk species e.g. macaws, Amazon parrots and conures, would be a prudent step to determining if there is ongoing reason to be concerned about this virus’ presence in Australia.

David Phalen, (02 9351 1798, dphalen@camden.usyd.edu.au) is interested in frozen samples and swabs from birds with mucosal papillomas. He is also interested in frozen tissues from birds with suspected herpesvirus infections.

Human health implications

There are no human health implications

Conclusions

Psittacid herpesvirus-1 (PsHV-1) has not been reported in wild Australian parrots. PsHV-1 has caused significant mortality events in captive-raised parrots in collections around the world. It is also the cause of the mucosal papillomas and in some birds, bile duct and pancreatic duct carcinomas. Birds with mucosal papillomas have been imported into Australia and genotypes 2 and 3 have been detected in three birds that were tested. Because most importation of birds occurred before testing for this virus was available and most infected birds will be subclinical, it is likely that this virus is present to a low degree in some avicultural collections in Australia.

References and other information


Acknowledgements

We are extremely grateful to the people who had input into this fact sheet, including David Phalen. Without their ongoing support production of these fact sheets would not be possible.

Reviewed: January 2017

To provide feedback on this fact sheet

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. Negative data are also valuable. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.
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