Herpesviruses and Australian wild birds

Fact sheet

Introductory statement

Avian herpesviruses are extremely widespread and cause a large variety of diseases, including commercially significant conditions such as infectious laryngotracheitis and Marek’s disease of poultry, as well as highly fatal conditions such as Pacheco’s disease of psittacines. They are characterized by establishing latent infections which lead to periodic excretion of virus during times of stress and debilitation. This fact sheet focuses on herpesviruses in wild Australian birds, exclusive of psittacines. For information on herpesviruses in psittacines consult the “Psittacid herpesviruses and mucosal papillomas of psittacine birds in Australia” fact sheet.

Aetiology

Herpesviruses are enveloped DNA viruses that range in size from 120 to 200 nm. They are divided into three subfamilies. Alphaherpesviruses have a moderately wide host range, rapid growth, lyse infected cells and have the capacity to establish latent infections primarily, but not exclusively, in nerve ganglia. Betaherpesviruses have a more restricted host range, a long replicative cycle, the capacity to cause infected cells to enlarge and the ability to form latent infections in secretory glands, lymphoreticular tissue, kidneys and other tissues. Gammaherpesviruses have a narrow host range, replicate in lymphoid cells, may cause the development of tumours in infected cells and form latent infections in lymphoid tissue. All avian herpesviruses are either alphaherpesviruses or have not yet been allocated to a subfamily.

Natural hosts

Herpesviruses have been found in all groups of birds. However, severe disease usually only occurs when viruses specific for particular species find their way into unfamiliar species or if infected birds are immunocompromised in some way.
World distribution

Worldwide.

Occurrences in Australia


Epidemiology

Herpesvirus transmission generally requires close contact and usually occurs in birds by inhalation of virus containing dust derived from feathers, nasal secretions, saliva, urine and faeces. Birds, such as pigeons, that feed their offspring with crop milk experience activation of latent infection in the oropharyngeal region during egg incubation and transmit virus via the crop milk to their newly hatched offspring. The affected raptors likely contracted the disease by feeding on infected pigeons (Phalen et al 2011, Kaleta and Docherty 2007).

Infections are usually acquired early in life, leading to latency generally in the trigeminal ganglia. Virus can be expressed, leading to morbidity and mortality, at any stage depending on endogenous and exogenous factors. Vertical transmission through the egg has not been reported and arthropod vectors are not required.

Herpesviruses are fragile and do not survive well outside the body. However, the duck plague virus can retain infectivity for up to 30 days at room temperature (Geering et al 1995). Herpesviruses are killed by all common disinfectants including bleach and F10 (benzalkonium chloride/polyhexamethylene biguanide hydrochloride). Sunlight, high temperatures and low humidity will also inactivate the virus (Kaleta and Docherty 2007).

Clinical signs

Feral pigeons can carry the virus asymptomatically but clinical signs have been described in racing pigeons. These included rhinitis, tracheitis, dyspnoea, depression, anorexia, diarrhoea, vomiting, neurological signs and ulcers on mucous membranes. Older birds generally developed milder infections and recovered in one to two weeks (Macwhirter 2000).

Affected raptors and waterbirds were found dead with no clinical signs or showed brief periods of lethargy and anorexia. Some of the domestic geese developed leg weakness and prostration prior to death. Affected Gouldian finches displayed dyspnoea, conjunctivitis, a head tilt, lethargy and sudden death (Phalen et al 2011, Wellehan et al 2003, Ketterer et al 1990, Reece et al 1987).
**Diagnosis**

While clinical signs and histopathological lesions are suggestive of herpesvirus infection definitive diagnosis requires virus isolation from infected tissues. Electron microscopy and PCR can also be used. In live birds the detection of antibodies in neutralisation tests will confirm previous exposure (Kaleta and Docherty 2007).

**Pathology**

Affected raptors had marked multifocal acute hepatic and splenic coagulative necrosis with eosinophilic intranuclear inclusion bodies in both organs. There were occasional areas of focal pancreatic necrosis with inclusion bodies visible only in the hobby. All birds also had segmental multifocal coagulative necrosis of the small intestine with scattered intranuclear inclusion bodies (Phalen et al 2011).

Affected waterbirds had similar hepatic lesions. Swans and Cape Barren geese also had foci of necrosis, haemorrhage and congestion throughout the gastro-intestinal tract, and haemorrhage and necrosis in the spleen. Domestic geese had focal haemorrhages on the liver surface and fibrinous enteritis (Ketterer et al 1990, Reece et al 1987).

In contrast to raptors and waterbirds Gouldian finches tended to have respiratory, rather than hepatic, lesions. Affected finches had a tracheitis with necrotic epithelial cells, heterophils, macrophages, fibrin and cellular debris all sloughing into the tracheal lumen. Surviving epithelial cells showed karyomegaly and contained basophilic intranuclear inclusion bodies. Bronchial epithelial cells were hyperplastic and contained similar inclusions, which could also be found in the conjunctiva and oesophagus. The liver only had scattered apoptotic hepatocytes (Wellehan et al 2003, Gelis 2003).

Both sets of herpesvirus lesions have been described in pigeons. Two reports in racing pigeons described diffuse hepatic necrosis with eosinophilic intranuclear inclusions (Surman et al 1975, Boyle and Binnington 1973). However, a third report described an outbreak in squabs where the predominant lesions were epithelial cell necrosis with intranuclear inclusion bodies in the mucosa of the oral cavity, oesophagus, salivary gland, nasal passages and trachea. The spleen also had focal necrosis with intranuclear inclusion bodies and lymphoid cell depletion, but hepatic lesions were seen in only a small number of birds (Callinan et al 1979).

**Differential diagnosis**

Differential diagnoses include any causes of sudden death such as trauma, mycotoxins, bacterial infections such as salmonellosis, fungal infections such as aspergillosis, other viral infections such as paramyxovirus and influenza, and viral infections that produce similar lesions, such as adenovirus (Kaleta and Docherty 2007).

**Laboratory diagnostic specimens**

A complete necropsy should be performed. Collect a range of tissues, including liver and any obvious lesions, and submit them in formalin for histopathology. Fresh or frozen tissues should also be submitted for viral culture and PCR. Pharyngeal swabs were found to be a reliable indicator of infection in feral rock pigeons when tested by PCR.
Laboratory procedures

Homogenates of affected organs are inoculated onto avian cell cultures or into embryonated chicken eggs. In the latter case white foci appear on the chorioallantoic membrane within one week. These can be confirmed as herpesvirus by PCR or electron microscopy (Kaleta and Docherty 2007).

Treatment

Treatment is difficult as infected birds are usually asymptomatic carriers or are found dead with no previous clinical signs. One study was able to reduce morbidity and mortality in experimentally infected Quaker parakeets (*Myiopsitta monachus*) by giving 80 mg/kg acyclovir orally three times per day for seven days (Norton et al 1991).

Prevention and control

Autogenous vaccines have been used to control mortality in captive raptors, but would be difficult to use in wild birds. As these vaccines contain attenuated virus it may be possible for vaccinated wild birds to infect other species, resulting in disease. For captive breeding and release programs birds should be screened prior to release if herpesvirus infection is identified as a possible problem (Kaleta and Docherty 2007).

Given the apparently high prevalence of herpesvirus infection in feral pigeons it would seem prudent to discontinue feeding these birds to raptors and maintain adequate feral pigeon control measures.

Surveillance and management

There is no targeted surveillance program for herpesviruses in Australian wild birds. However, cases may be logged in the national wildlife health information system as part of Australia’s general wildlife surveillance activities. Herpesviruses are considered as part of routine Import Risk Analysis processes for Australia.

Statistics

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.

No cases of avian herpesvirus infection could be found in the National Wildlife Health Surveillance Database (eWHIS). However, an unpublished survey of 28 feral rock pigeons, originating from three flocks in the Melbourne area and one flock in the Sydney area, found a prevalence of infection ranging from 30 to 100%. None of the birds had clinical signs.

Research

While rock pigeons appear to be an obvious reservoir for avian herpesviruses in Australia no research has been done on native pigeon species to assess the prevalence of infection in this group. Infection prevalence in wild raptors and other bird groups is also unknown.
Feral pigeons are still fed to raptors despite the risk of contracting herpesvirus infection. To decrease this risk, it has been suggested to remove the pigeon heads prior to feeding as virus is harboured in the trigeminal ganglia and pharynx. No research has been done to assess the validity of this theory (Kaleta and Docherty 2007).

Further vaccine research is necessary to adequately protect at risk species.

**Conclusions**

While avian herpesviruses are likely widespread through the Australian avifauna relatively few reports of disease have appeared in the literature. Given the available information they do not appear to pose a threat to established populations of free ranging bird species. However, as habitat continues to shrink and birds are brought into captivity for breeding programs the potential for viruses to be introduced to naive populations increases, raising the probability of future herpesvirus disease outbreaks. Ongoing surveillance and awareness of the risks will be necessary to prevent this occurring.

**References and other information**


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**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. 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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