

Avian paramyxoviruses and Australian wild birds

Fact sheet

Introductory statement

Avian paramyxoviruses (APMV), in particular APMV-1, have been associated with highly contagious and infectious viral diseases, affecting all avian species including poultry, cage and wild birds (Animal Health Australia 2013). Avirulent APMV-1 and other strains are considered widespread in Australian native birds, with strains believed to be present in wild waterfowl populations in all states. Australia was free of pigeon paramyxovirus-1 (PPMV-1, a strain of APMV-1) until 2011, when it was first detected in domestic pigeons. PPMV-1 has since spread to feral pigeons and may be a risk for some Australian raptors. Other than PPMV-1, APMVs have not been reported to cause disease in wild birds in Australia (Ladds 2009). This fact sheet focuses on what is known about APMVs in Australian wild birds. Clinical disease, caused by virulent (or highly pathogenic) strains of APMV-1, has not been identified in Australian wild birds. For more information on Newcastle disease in poultry in Australia see the Animal Health Australia website www.animalhealthaustralia.com.au.

Aetiology

Avian paramyxoviruses are members of the genus *Avulavirus* (formerly *Rubulavirus*), family *Paramyxoviridae*, order *Mononegavirales*. They are a single stranded, negative sense RNA viruses, approximately 150 nm in diameter with a lipid envelope (Leighton et al. 2007). As with most RNA viruses, they are more prone to mutations compared with double-stranded genomes. This is associated with continual molecular evolution with the potential for avirulent viruses to mutate into virulent forms.

There are a number of different genetic groups contained within nine distinct serotypes, APMV-1 to APMV-9, with 4 additional proposed serotypes, APMV-10 to APMV-13 (Dimitrov et al. 2016). The most well recognised serotype is APMV-1 (USGS 1999).

- Newcastle disease (ND) is a commonly used term for virulent APMV-1, when it is found in poultry. There are strict definitions of when an APMV-1 infection is termed ND (OIE 2012)¹.
- There are two classes of APMV-1. Class I are predominantly isolated from wild birds and occasional spill over into poultry has occurred. Class II viruses are responsible for most of the outbreaks seen in poultry worldwide and have also been found in wild birds (Aldous et al. 2003). PPMV-1 is the pigeon strain of APMV-1 and is classified as a Class II, genotype VI virus.
- APMV-2 primarily causes disease in turkeys and wild birds, however disease in chickens has also been documented (Peroulis and O'Riley 2004).
- APMV-3 is only recognised in turkeys.
- APMV-2 and APMV-3 can cause respiratory disease, decrease in egg production and a range of other signs (Stanislawek et al. 2001).
- APMV-5 has been reported to cause fatal disease in budgerigars (*Melopsittacus undulates*) overseas (Peroulis and O'Riley 2004).
- APMV-4, and APMV-6 to APMV-9 are all considered non-pathogenic.

Natural hosts

The natural hosts for APMV infections include chickens, turkeys, ducks, geese, pigeons, ostriches, quails, guinea pigs, crows and many other species of aviary and wild birds (Alexander 2000). Over 230 species from dozens of avian orders have been found to be susceptible (USGS 1999). Susceptibility and severity of disease varies between host species. Wild birds and waterfowl may harbour subclinical APMV infections and are the least likely to be clinically affected. Raptors are usually resistant to APMV. Passerine birds are reported to vary in their susceptibility; some species show no signs of disease but excrete APMVs, while others may develop severe disease. There may be variation in the severity of clinical signs even within different species of an avian genus.

In North America, virulent APMV-1 causes significant epidemic mortalities in juvenile wild double-crested cormorants (*Phalacrocorax auritus*), with deaths of 20,000 or more and up to 90% mortality. Die-offs are seasonal and occur in young of the year in breeding colonies during the spring and summer months, often associated with neurological signs. Although other birds, including pelicans and gulls, are reported to have died in association with cormorant deaths, in most cases APMV-1 has not been confirmed as the causal agent, and there are no other reports of large-scale disease in wild birds in North America (USGS 1999; Sleeman 2016). APMV-1 infection has not been reported in cormorants in Australia.

Reports of APMV-related disease in naturally or experimentally infected Australian species overseas include a range of psittacine species, osprey (*Pandion haliaetus*), emu (*Dromaius novaehollandiae*) and finches. Budgerigars are considered particularly susceptible to disease from APMV-1.

¹ Poultry is defined by the OIE as 'all domesticated birds used for the production of meat or eggs for consumption, for the production of other commercial products, for restocking supplies of game, or for breeding these categories of birds'.

World distribution

Avian paramyxoviruses are endemic to many countries worldwide with various strains found in different parts of Africa, Asia, Central and South America. There have been three pandemics of virulent APMV-1 since the virus was discovered in 1926. The first virulent strain originated from Southeast Asia and infected poultry and domestic birds. The second pandemic originated in the Middle East in the 1960s and was attributed to the movement and importation of caged psittacine species. The most recent pandemic arose in the Middle East during the 1970s and primarily involves pigeons and doves (USGS 1999).

Pigeon paramyxovirus also has a worldwide distribution. It first appeared in the Middle East in the late 1970s, then in Europe in 1981, Japan and North America in 1984 and South Africa in 1986 (Dorrestein 1992; Abolnik et al. 2008; Alexander 2011).

Occurrence in Australia

APMVs occur in Australia, with avirulent APMV-1 and other strains considered widespread in Australian native birds. Prevalence is reported to vary from 0.04% in non-aquatic birds to 7% in aquatic birds (Peroulis and O'Riley 2004; Hoque et al. 2012).

Studies have found evidence of exposure to APMV in 10-16% of grey teal (*Anas gracilis*) sampled in Victoria (Hore 1973) and in a range of *Charadriiformes*, *Passeriformes* and *Anseriformes* in Western Australia (Alexander et al. 1986).

In one study, 605 tracheal and cloacal samples were collected from wild ducks, quail, pigeon and other wild birds throughout Victoria during 2001-2002. The viral isolates were cultured and then characterised based on the haemagglutination assay (HA) and haemagglutination inhibition (HI) test. Twenty-five avirulent viruses were isolated based on a positive HA result, including 18 APMV-6 and two APMV-1 isolates. Most of the isolates were from wild ducks including teal (*Anas* sp.), Pacific black duck (*Anas superciliosa*), pink-eared duck (*Malacorhynchus membranaceus*), Australasian shoveler (*Anas rhynchotis*), Australian wood duck (*Chenonetta jubata*) and quail (species not specified). There was no evidence of APMV infection in 133 pigeons (species not specified) (Peroulis and O'Riley 2004).

A study in north Qld, collecting samples from 1461 birds and 1157 environmental samples, found evidence of largely avirulent APMV by molecular studies in plumed whistling ducks (*Dendrocygena eytoni*) (4.2%) and Pacific black ducks (0.9%). Low level evidence of infection was also found in Australian white ibis (*Threskiornis molucca*). Overall prevalence was 3.5% for live bird samples (n = 1461) and 0.4% for faecal samples (n = 1157). A range of other waterbird species were sampled, and none showed evidence of APMV infection (Hoque et al. 2012). Other unpublished studies have found low level evidence of APMV-1 in a range of waterbird species in Victoria, but not in Qld or NT (WEDPP reports, some available online). Other studies failed to find evidence of infection in Qld and NT (Diallo et al. 2007; WEDPP report 2006-2007).

In 2011, an outbreak of an exotic strain of APMV-1 (PPMV-1) occurred in Australia, first in domestic pigeons (*Columba livia domestica*). The first cases appeared in August 2011 at Shepparton, Vic, before spreading to domestic pigeons in the greater Melbourne area. Domestic pigeons in the Sydney region were affected in May 2012, with subsequent detections in domestic pigeons in Tasmania in June 2013, WA in November 2015 (DAFWA 2015), and SA in January 2016 (PIRSA 2016). Genetic analysis suggests that the virus arrived in Australia from the Middle East (Qld DAFF 2012).

Since 2011, PPMV-1 has been predominantly found in domestic pigeons (racing, show and fancy). Wild bird infections have occurred in feral rock pigeons (*Columba livia*). Two cases have been confirmed in a spotted turtle dove (*Streptopelia chinensis*) and a collared sparrowhawk (*Accipiter cirrocephalus*) in Vic [see AWHN newsletter volume 10 issue 2 for more information]. The sparrowhawk was a juvenile bird and may have been predisposed to disease due to a concurrent fungal infection (Australian Wildlife Health Network 2012). The ability of the PPMV-1 strain present in Australia to cause disease in native pigeons remains unknown. While PPMV-1 has caused disease in poultry in Europe and South Africa, the Australian variant appears to be non-pathogenic for poultry. Experimental inoculation of poultry demonstrated infectivity and spread to in-contact poultry, but no apparent disease. Poultry in contact with infected pigeons have also not shown signs of disease (Abolnik et al. 2008; Alexander 2011; Qld DAFF 2012).

An earlier study in WA found 14 isolates of APMV-1 from wild birds belonging to the orders *Charadriiformes*, *Passeriformes* and *Anseriformes* (Alexander et al. 1986). Other Australian studies have failed to find evidence of APMV-1 in wild birds (Garnett and Flanagan 1989; Diallo et al. 2007). Other than PPMV-1, APMVs have not been reported to cause disease in wild birds in Australia (Ladds 2009).

Epidemiology

In Australia, wild birds such as geese, ducks and other waterfowl are considered the main sources of avirulent APMV-1. This may be due to the fact the virus survives well in water, which may have allowed waterfowl to be exposed during their evolution (USGS 1999).

The incubation period for PPMV-1 in pigeons is one to four weeks. Mortality rates vary widely, from 10-100%. Birds that recover may continue to shed virus for several weeks. One study found infectious virus in rock pigeon faeces over six months after infection (Leighton et al. 2007).

Avian paramyxoviruses may be introduced into bird populations by various means including movements of domestic poultry, contaminated feed and equipment and other fomites, transit of infected poultry products and migration of birds from overseas. In wild birds, intestinal infection by faecal-oral route is the main route of infection. The virus can survive in the environment for several weeks and can spread readily due to its high resistance to adverse environmental conditions, its wide avian host range and its high infectivity (Alexander 2000). Wild bird carriers may shed the virus for up to six weeks and may potentially spread endemic APMV to susceptible poultry flocks (Animal Health Australia 2013).

The taxa most likely to carry virulent APMV are pigeons and psittacines (Garnett and Flanagan 1989) and the main source of risk of a virulent APMV outbreak in Australia is considered to be through illegal importation of these species (Animal Health Australia 2013).

Since its introduction into Australia in 2011, the detection of pigeon paramyxovirus in feral rock pigeons in NSW and Vic and domestic pigeons in Vic, NSW, Tas, SA and WA indicates that the virus continues to circulate in pigeon populations in Australia.

Clinical signs

General

Clinical signs caused by APMV-1 infection are determined by the host species, virulence and tropism of the virus, age, immune status, exposure, viral dose and environmental conditions (Animal Health Australia 2013).

Even with milder strains of APMV-1, disease may be exacerbated when co-infections or adverse environmental conditions are present (OIE 2012).

Pigeons

Clinical signs include depression, anorexia, excessive drinking, polyuria, watery to haemorrhagic diarrhoea followed by neurological signs (incoordination, paralysis of wings and legs and torticollis) and death, usually one to three days after the onset of clinical signs (Alexander 2000). Neurological signs were more obvious in the Victorian cases seen in 2011, while birds infected in NSW showed more non-specific signs.

Wild waterfowl

Wild birds usually do not demonstrate classic clinical signs of virulent paramyxovirus infection however birds in a weakened state (e.g. during annual migrations) may develop clinical signs and die. Wild waterfowl in Australia do not migrate.

In North America, infection of double-crested cormorants with virulent APMV-1 has been associated with by neurological signs and death in juvenile birds.

Diagnosis

Diagnosis is based on clinical signs in conjunction with histological lesions and laboratory testing. Virulent APMV-1 is a nationally notifiable disease and rapid diagnosis is important. Initial laboratory testing is conducted by quantitative RT-PCR. Definitive diagnosis is based on viral isolation and characterisation, including molecular pathotyping (Alexander 2000). Serological tests include the enzyme linked immunosorbent assay (ELISA) and HI, which is the most widely used test as it has the highest specificity (Animal Health Australia 2013).

Pathogenicity testing is important as strains of APMV-1 vary in their virulence. A positive diagnosis of virulent APMV is confirmed by molecular testing of samples from a bird showing clinical signs. A virulent virus is defined by the presence of multiple basic amino acids at the fusion protein cleavage site, along with a phenylalanine at residue 117. The intracerebral pathogenicity index (ICPI) in day-old chicks is occasionally used and is the OIE approved test for virulence (OIE 2012; Animal Health Australia 2013).

Embryonated chicken eggs are used for viral culture. Allantoic fluid from infected eggs can be tested for the presence of virus via haemagglutation and for viral RNA by PCR.

Clinical pathology

No information could be found on clinical pathology for APMVs in wild birds.

Pathology

Gross lesions seen in pigeons infected with PPMV-1 during the 2011 outbreak in Victoria included reddened legs, splenomegaly and multiple pale foci of necrosis in multiple tissues including the spleen, kidney and pancreas. Histologically, focally extensive lymphoplasmacytic interstitial nephritis is common. Pancreatic lesions include degranulation and necrosis of acinar cells. Locally extensive to diffuse pulmonary oedema occurs less commonly (Phalen 2012).

No typical gross lesions of APMV infection are recognised in wild birds. Typical signs of virulent APMV-1 infection in poultry include conjunctival and tracheal haemorrhages, splenic and hepatic enlargement, and haemorrhage of thymus and bursa of Fabricius.

Differential diagnoses

Differential diagnoses of APMV-1 include salmonella septicaemia, adenovirus and herpesvirus infections and intoxications (Dorrestein 1992; Phalen 2012). Based on clinical and post mortem examinations, APMV-1 infection is not readily distinguishable from avian influenza, infectious laryngotracheitis, infectious bronchitis, Marek's disease, other paramyxovirus infections and toxicoses.

Laboratory diagnostic specimens

Specimens should be collected from live, clinically diseased birds and recently dead birds. In live birds, serum, cloacal and oropharyngeal swabs and fresh faeces should be collected. In dead birds, samples from the respiratory tract, digestive tract and brain should be collected (Animal Health Australia 2013). Serum may be used for HI to detect APMV antibodies. Tissues which can be used for viral isolation and pathogenicity testing include trachea, lung, kidneys, intestine (and contents), caecal tonsils, spleen, brain, liver and heart (OIE 2012).

For PPMV-1 investigation, it is recommended to contact the diagnostic laboratory for submission requirements. It may be requested to submit the whole bird in a zip lock bag on ice along with a cloacal swab in viral transport media (VTM). If no VTM is available, the swab may be placed in sterile saline on ice. If possible, the swab should be delivered to the laboratory on the same day.

Treatment

There is no specific treatment for APMV-1 infection, although supportive care, if appropriate, may reduce the severity of the disease and increase the chances of survival.

Prevention and control

Control of APMV-1 in domestic birds is aimed at preventing the introduction and spread of the virus through good biosecurity measures, including vaccination. Biosecurity may include methods to prevent wild birds from coming in close contact with domestic birds and their food or water (Animal Health Australia 2013). Other mechanical vectors in the environment such as vertebrate pests should be controlled. Additional measures include isolation and monitoring of newly-arrived birds for four weeks, and cleaning and disinfecting travelling boxes and other equipment after use. For further information see Animal Health Australia (2013).

There is no registered vaccine for PPMV-1 currently available for use in pigeons in Australia. However, inactivated ND vaccine gives some protection. The vaccine does not prevent birds from becoming infected, however, the severity of clinical disease and viral shedding is reduced. Two doses have been given four weeks apart followed by an annual booster. While side-effects following vaccination are rare, a report documented the development of a sterile granuloma at the vaccination site (Cowan et al. 2014). Vaccination could be considered for use in at-risk birds in captive facilities such as zoos and private aviaries (NAIEVEG 2010).

Prevention of PPMV-1 in wild birds would be largely dependent on managing or preventing spill over from domestic pigeons, during an outbreak. Preventative measures such as vaccination are not feasible in wild birds, however, attempts could be made to ensure virus is not inadvertently transmitted from outbreak sites to other areas of concern. Infected birds should be kept isolated, if possible, from other birds for at least 60 days after the last death or the last new clinical case. All waste should be disposed of and all surfaces washed thoroughly with water and detergent. Disinfectants effective against APMVs include phenolics, glutaraldehyde, sodium hypochlorite, and potassium peroxymonosulfate (Virkon-S), but not quaternary ammonium compounds. Alcohol based hand sanitizers will kill virus on the hands (Patnayak et al. 2008).

Surveillance and management

PPMV-1 and ND are nationally notifiable animal diseases. Wildlife Health Australia recommends consideration of exclusion of APMVs in wild bird morality events and specific exclusion of PPMV-1 in wild bird mortality events involving pigeons and raptors.

More state specific information on PPMV-1 can be found at http://www.dpi.vic.gov.au/agriculture/pests-diseases-and-weeds/animal-diseases/pigeon-paramyxovirus (Vic),

http://www.dpi.nsw.gov.au/agriculture/livestock/poultry/health-disease/paramyxovirus-in-pigeons (NSW), http://www.dpipwe.tas.gov.au/internnsf/Attachments/CYAA-

9949N7/\$FILE/Avian%20Paramyxovirus%20Fact%20Sheet.pdf (Tasmania),

http://www.daff.qld.gov.au/__data/assets/pdf_file/0003/67251/Avian-paramyxovirus-vet-guidelines-July-12.pdf (Qld),

http://www.pir.sa.gov.au/biosecuritysa/animalhealth/poultry/health and disease management of poultry/pigeon_paramyxovirus (SA), and http://archive.agric.wa.gov.au/PC_94697.html?s=0 (WA).²

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.

There are currently several dozen reports of PPMV-1 in eWHIS to August 2016. Most reports relate to disease in feral rock pigeons occurring in and around Melbourne, Vic and Sydney, NSW. Other reports include a single juvenile collared sparrowhawk and one spotted turtle-dove that tested positive to PPMV-1. APMV has been reported as an incidental finding (not related to pathology) in four wild bird mortality events in eWHIS.

² ND outbreaks in Australia are managed under the Animal Health Australia's disease strategy: Animal Health Australia. *Disease Strategy: Newcastle Disease (Version 3.1)* (AUSVETPLAN).

Research

Studies have been undertaken on the molecular characterisation of APMV-1 isolates to assess and understand the genetic origins and relationship of outbreaks (Peroulis-Kourtis et al. 2002). A study based on detection of APMVs amongst wild bird populations collected epidemiological information against which to compare future isolates from outbreaks of disease to determine their origin (Peroulis and O'Riley 2004).

More research is required to determine the extent of Australian species susceptibility to infection and disease, in particular of the recently arrived PPMV-1 strain, and whether native pigeons could be involved. The prevalence of the disease in wild pigeon populations is also unknown at this stage. Further work may also be required to assess the effectiveness of vaccination in pigeons and the duration of immunity and any impact upon shedding.

Human health implications

Although APMV can act as a human pathogen, infection is rare and usually occurs only in people who have very close contact with infected birds, resulting mild, short-term conjunctivitis or influenza-like symptoms.

Transmission to humans is often by aerosol rather than direct contact and is generally limited primarily to laboratory workers and vaccinations teams exposed to large quantities of the virus. The ability of the virus to multiply and transmit in humans is of no epidemiological significance (USGS 1999). Human infection with PPMV-1 has not been reported.

Conclusions

The recent incursion of PPMV-1, a disease previously considered exotic, highlights the dangers of infectious disease to Australia and the need for vigilance and timely investigation and reporting of disease events. It also highlights the risk of spill over of disease between taxa and the need for good biosecurity procedures that separate captive and wild birds.

Surveillance and monitoring of wild birds for APMVs would help to determine the prevalence of APMVs in wild bird populations and provide further understanding of the epidemiology of these viruses. This information could be useful in assessing risk and the role of wild birds in the ecology of APMVs in Australia.

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To provide feedback on this fact sheet

We encourage those with laboratory confirmed cases of this condition in native Australian or feral animals to submit this information to the national system for consideration for inclusion in the national database. 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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