Introductory statement

In 1988, a novel morbillivirus, phocine distemper virus (PDV) was the cause of an epizootic in which approximately 20,000 harbor seals (Phoca vitulina) died in north European waters (Osterhaus et al. 1988). Since this event further epizootics caused by morbilliviruses have been confirmed in Northern Hemisphere pinnipeds (Grachev et al. 1989; Osterhaus et al. 1997; Kennedy et al. 2000) but as yet, not in Southern Hemisphere species. There is, however, serological evidence that morbilliviruses are present in Southern Hemisphere pinnipeds, including some Antarctic species that are occasional visitors to the Australian mainland (Bengtson and Boveng 1991). The effective conservation management of Australian seals depends on a sound knowledge of their potential population regulatory factors. It is, therefore, important to investigate if pathogens recognised to be of significance in pinnipeds elsewhere are present in Australian seals. In addition, an assessment of the risk and consequence of pathogen introduction is required. This will enable infectious disease threats to Australian seals to be incorporated into their larger management plan.

Aetiology

Morbilliviruses found to cause disease in seals belong to the genus Morbillivirus, family Paramyxoviridae. They have been either characterised as strains of canine distemper virus (CDV) or a seal-specific agent, phocine distemper virus (PDV).

Natural hosts

The origin of CDV in seals is believed to be from the natural hosts of this virus, mustelids and canids. CDV is capable of causing epizootics in both wild and domestic populations of these natural hosts. The origin of PDV is more obscure. It is a novel seal-specific virus most closely related to CDV rather than human or ruminant morbilliviruses.
**World distribution**

CDV has a world-wide distribution associated with the presence of wild or domestic canids and mustelids. CDV was found to be responsible for the mass mortality events in Baikal seals (*Phoca sibirica*) in 1987 and Caspian seals (*Phoca caspica*) in 2000 and the virus is considered to have been introduced into these populations through contact with either domestic dogs or wolves (Grachev et al. 1989; Kennedy et al. 2000). Introduction of CDV into Antarctic phocid seals is thought to have been via infected sled dogs imported from Arctic Canada and Greenland, where the disease is endemic (Bengtson and Boveng 1991). CDV is prevalent in many terrestrial wildlife species of South America (Whiteman et al. 2007), and recently, debilitated South American sea lions (*Otaria flavescens*) have been observed with clinical signs of distemper (paresis, seizures and dyspnoea) as well as histological evidence of distemper in brain tissues (Grau et al., 2009). PDV has been isolated from phocid seals from both northern and western European waters, the Mediterranean Sea and North American waters. In New Zealand waters there is serological evidence of exposure to a PDV-like virus in both New Zealand fur seals (*Arctocephalus forsteri*) and New Zealand sea lions (*Phocarctos hookeri*) (Duignan et al. 2000).

**Occurrences in Australia**

There have been no reports of disease caused by morbillivirus infections in seals in Australian waters. The prevalence of CDV in domestic dogs in Australia is low, but the disease occurs sporadically (Norris et al. 2006). The prevalence of CDV is unknown in Australian foxes, dingos and feral dogs, but is likely to be low. There is serological evidence of exposure to CDV in leopard and crabeater seals, both of which are occasional visitors to the Australian mainland (Bengtson and Boveng 1991). A survey of Australian fur seals in Bass Strait and New Zealand fur seals on Kangaroo Island found no evidence of exposure to morbilliviruses (Lynch et al. 2011). Anecdotal reports suggest the presence of exposure to CDV in captive seals in some states in Australia (Blyde pers. comm.).

**Epidemiology**

There appears to be variation in seal species susceptibility to disease resulting from morbillivirus infection. PDV epizootics in European waters in 1988 and 2002, resulted in harbor seals dying in large numbers with minimal mortality among sympatric grey seals (Heidejorgensen et al. 1992; Jensen et al. 2002). It is believed that some species may act as asymptomatic carriers and fluctuations in their foraging ranges may be the means whereby virus is introduced to immunologically-naive, susceptible populations (Dietz et al. 1989; Barrett et al. 1995). In susceptible phocid seal species the introduction of morbillivirus has been estimated to result in >95% of animals becoming infected and in the order of 40-60% mortality (Heide-Jorgensen and Harkonen 1992; Klepac et al. 2009). In addition to variation in species-susceptibility the aggregation of large numbers of animals facilitates spread of disease. The numbers of animals present at haul outs will vary with species, season and over-arching climatic events. There have been no morbillivirus-related mass mortality events observed in otariid seals (fur seals and sea lions) world-wide but it is unknown whether this reflects an innate resistance in this group, the lack of exposure of susceptible species, or suboptimal conditions to sustain an epidemic.
Clinical signs

Clinical signs of CDV and PDV infection in seals reflect pulmonary and neurological disease. Commonly dyspnoea, convulsions, hyperthermia and subcutaneous oedema are seen (Di Guardo et al. 2005). In the early stages of infection pregnant females may abort (Heide-Jorgensen and Harkonen 1992).

Diagnosis

Exposure to morbilliviruses can be shown by serum neutralization testing utilising canine distemper virus (CDV) as the antigen. Antibodies to specific morbilliviruses will cross react with all related-virus species with the highest titres being obtained against the homologous species (Liess et al. 1989). Immunohistochemical staining of tissues will demonstrate virus, particularly in respiratory epithelium (Kennedy et al. 1989). Histological lesions (described below) may strongly suggest morbillivirus infection. Diagnosis can also be made by virus isolation (Osterhaus et al. 1988) and specific molecular techniques (Mamaev et al. 1995).

Pathology

Pulmonary lesions predominate on gross post mortem. Severe broncho-interstitial pneumonia is evident as congested, oedematous lungs that fail to collapse. Inter-lobular, subpleural and mediastinal emphysema is often present (Di Guardo et al. 2005). Oedematous and enlarged mediastinal lymph nodes may be evident. Bilateral keratitis has been noted in some species (Kennedy et al. 1989). Histological lesions resemble those seen in CDV infection of terrestrial mammals. Pulmonary lesions are characterised by diffuse, non-suppurative broncho-interstitial pneumonia with necrosis of bronchial and bronchiolar walls and significant exudation into lumina. Of particular significance is the formation of large multinucleated syncitia within alveolar, bronchiolar and bronchial lumina. CNS lesions are characterised by perivascular, mononuclear infiltrates and neuronal degenerative and necrotic changes. Inflammatory lesions generally occur within the cerebral cortex. Intracytoplasmic inclusion bodies may be seen within the lung. CNS, renal pelvis, urinary bladder, biliary and pancreatic ducts.

Differential diagnoses

It is unknown whether the appearance of morbillivirus infection of Australian seals would result in a mass mortality event as has been observed in some northern hemisphere species. Regardless of this, affected animals would be expected to demonstrate respiratory and/or CNS abnormalities. Influenza virus can cause severe respiratory signs and death in seals (Geraci et al. 1982). CNS disease may result from ingestion of algal toxins (Scholin et al. 2000; Fire et al. 2008) or infection with protozoa, including Toxoplasma gondii (Dubey et al. 2003).

Laboratory diagnostic specimens

- Serum: Include collection of blood from post mortem specimens
- Effusions: Pleural and pericardial. Store frozen
- Fresh Tissues: Include lung, lymph nodes (mediastinal, mesenteric, axillary), heart, liver, spleen, kidney, cerebrum, bladder and GIT. Store frozen
- Formalised tissues: Include those listed for fresh tissues
Laboratory procedures

- Virus neutralisation assay
- Immunohistochemistry
- Histological examination
- PCR

Treatment

There is no specific treatment for seals suffering from morbillivirus infection.

Prevention and control

The status of Australian seals with regards to morbillivirus infection is largely unknown. No antibodies to CDV were found in serological surveys of Australian fur seals and New Zealand fur seals (Lynch et al. 2011) and there have been no observed morbillivirus-related mortality events. Until more definitive disease studies are conducted it is wise to assume that Australian seals are naïve to these pathogens. Therefore, management of these diseases should be focused on prevention of their introduction. Leopard seals and to a lesser extent crabeater seals are occasional visitors to the Australian mainland and could carry CDV or PDV to Australian fur seals. However, close contact between these individuals and local otariids would be unusual, so the risk of virus transfer is probably low. New Zealand fur seals have a broad range and are found in both New Zealand and southern Australia (Warneke and Shaughnessy 1985). The identification of a PDV-like virus in the New Zealand population and evidence that individuals from New Zealand can cross the Tasman Sea to south-eastern Australia (Shaughnessy 1999), suggests New Zealand fur seals could bring morbilliviruses into the range of other Australian seals. There is no way of controlling this potential route of introduction. Domestic and wild canids resident in Australia could possibly introduce CDV to seals. The contact between dogs and seals must be strongly discouraged. This is achieved by a combination of existing government legislation restricting movements of domestic animals and by public education.

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.

There are currently no formal programs surveying for morbilliviruses in Australian seals.

There is no AUSVETPLAN for seal morbilliviruses. However, any cases of PDV or CDV in seals 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).
**Statistics**

There are no cases of morbillivirus-associated disease in seals listed on the National Wildlife Health Surveillance Database.

**Research**

They key research question is whether Australian seals have been exposed to CDV or PDV. While some limited serological surveys have been conducted this research activity needs to be extended to seal populations throughout Australia.

Greater understanding of the epidemiology of CDV in Antarctic species would inform the risk that these species pose to Australian seals.

**Human health implications**

Morbillivirus infections in seals carry no human health implications.

**Conclusions**

Morbilliviruses have been the cause of mass mortality events in phocid seals in the Northern hemisphere caused by CDV and PDV. Australian seals are classed as otariids (fur seals and sea lions) and although there have been no morbillivirus-related mass mortality events observed in this group, the susceptibility of Australian seals to disease following morbillivirus exposure is unknown. However, epizootics caused by CDV in a wide range of terrestrial carnivores and marine mammals suggests that this virus in particular would be capable of inducing disease. It is therefore important that the status of Australian seals with regard to these viruses be established and that there is continued surveillance for morbillivirus-related disease.

**References and other information**


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We are extremely grateful to those who had input into this fact sheet. Without their ongoing support production of these fact sheets would not be possible.

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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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