Leptospirosis and Australian seals

Fact sheet

Introductory statement

Effective management of Australian seals depends on knowledge of their population regulatory factors including disease. Leptospirosis is recognised as a significant cause of morbidity and mortality in some northern hemisphere seal populations. It is therefore important to determine if leptospirosis is present in Australian seals and if so, define its epidemiological characteristics. In addition, wildlife populations may act as reservoirs of pathogens of significance to domestic livestock and public health. Leptospirosis is well recognised as a disease capable of spreading between mammalian species and its identification in Australian seals would be of interest to agencies responsible for managing livestock and public health.

Aetiology

Leptospirosis is caused by infection with motile, Gram-negative bacteria from the Genus *Leptospira*. Worldwide, there are over 300 serovars of *Leptospira* spp.

Natural hosts

Rodents are the maintenance hosts for most leptospira serovars but many types are adapted to other wild or domestic mammalian species. While each serovar has a host preference they often are capable of infecting other mammalian species and producing disease.

World distribution

Although there are no reports of leptospirosis from Antarctic mammals, the disease can be considered to have a worldwide distribution. In seals, leptospirosis has been reported from North American waters, and is of particular significance as a cause of mortality and morbidity in Californian sea lions (*Zalophus californianus*) (Lloyd-Smith et al. 2007). It has not been isolated in southern hemisphere pinnipeds but antibodies to several *Leptospira* serovars, *L. Hardjo*, *L. Canicola* and *L. Pomona*, have been found in New Zealand fur seal pups without evidence of disease (Mackereth et al. 2005).
Occurrences in Australia

*Leptospira* spp. serovars endemic to Australia, their host species and zoonotic potential have been reviewed (www.wildlifehealthaustralia.com.au). There have been no reports of leptospirosis in Australian seals. A serologic survey of Australian fur seals (*Arctocephalus pusillus doriferus*) in northern Bass Strait found no evidence of exposure to six *Leptospira* spp. serovars (Lynch et al. 2011): Serovars tested for were Australis, Copenhageni, Grippotyphosa, Hardjo, Pomona, and Tarassovi.

Epidemiology

*Leptospira* Pomona is the most common serovar isolated from northern hemisphere seal species (Smith et al. 1977; Dierauf et al. 1985; Colegrove et al. 2005). The epidemiological parameters of leptospirosis in marine mammals are not well established for a wide range of species. In terrestrial mammals, leptospirosis epidemiology has been reviewed by (Heath and Johnson 1994). Briefly, infection of a mammalian species with a *Leptospira* serovar for which it is the preferred host usually produces sub-clinical disease with prolonged bacterial shedding. Antibody prevalence in the host population will therefore be high. In contrast, disease in non-preferred hosts is usually of the form of sporadic infections or outbreaks. Infection is not maintained and seroprevalence in the population will be low.

Investigations of leptospirosis in California sea lions have been unable to definitively establish if they are a reservoir or accidental host. Antibody prevalence in the population is high across years and prolonged shedding has been demonstrated suggesting endemic infection (Colagross-Schouten et al. 2002; Lloyd-Smith et al. 2007). However, in contrast to other mammalian species in which leptospirosis is endemic, the California sea lion population suffers from periodic and significant outbreaks of clinical disease every 3-5 years (Gulland et al. 1996). It has been suggested that this may indicate either repeated exposure to a reservoir host or be a result of fluctuations in herd immunity factors (Lloyd-Smith et al. 2007) but more recently, outbreaks have been linked to seasonal movements of male sea lions between colonies (Zuerner et al. 2009).

Leptospirosis in seals is primarily seen in juvenile (>1 yr age) animals although adult animals can also exhibit clinical disease (Smith et al. 1977; Dierauf et al. 1985; Norman et al. 2008). In one study, risk of disease was associated with proximity to areas containing a high density of parks frequented by dogs suggesting they are a possible reservoir host (Norman et al. 2008). Routes of infection transmission are unknown but as renal pathology and shedding of bacteria in urine have been demonstrated (Dierauf et al. 1985) they are likely to be similar to terrestrial mammals. These routes have been reviewed (www.wildlifehealthaustralia.com.au) and may be direct through contamination of skin cuts and mucous membranes or indirect through environmental contamination.

Clinical signs

Seals with severe leptospirosis exhibit anorexia, extreme lethargy and thirst and abdominal pain (Zuerner et al. 2009). Seals are often emaciated and infection has been associated with abortion and neonatal mortality (Smith et al. 1977).
**Diagnosis**

Diagnosis of leptospirosis is made by demonstrating serological evidence of exposure and detection of the organism in urine or tissues. Serological diagnosis is achieved by the use of the microscopic agglutination test (MAT) which incorporates live cultures as the antigens. The MAT is used for leptospirosis investigations in many species and have been demonstrated to be highly sensitive in detecting infection in seals (Colagross-Schouten et al. 2002). *Leptospira* organisms in urine can be directly observed by use of dark field microscopy. Molecular techniques (PCR) are a very sensitive and specific diagnostic tool for antigen detection. Immunofluorescent staining of tissue sections can also be used to detect antigen.

**Clinical pathology**

Seals with leptospirosis exhibit clinical pathology consistent with renal failure. These are dilute urine, elevated serum urea, creatinine, sodium and phosphorus.

**Pathology**

Gross post mortem lesions observed in seals with leptospirosis include obviously swollen kidneys and pale renal cortices resulting in a loss of the differentiation between these areas and the renal medullae. Subcapsular renal haemorrhages may also be observed.

The primary histopathologic lesion is multifocal, severe, tubulointerstitial nephritis characterised by a lymphoplasmacytic infiltration. Renal tubular dilation and degeneration and glomerulonephritis may be present. Hepatic pathology is occasionally observed and in seals this has been characterised by multifocal hepatitis (Dierauf et al. 1985).

**Differential diagnoses**

E maciation and lethargy are clinical signs common to many chronic diseases of seals. These include severe parasitism and chronic bacterial infections. Progressive renal failure, presumably immune-related is not uncommon in aged seals.

**Laboratory diagnostic specimens**

Serum. 200ul per serovar test. Transport at 4°C or store frozen
Urine. Transport at 4°C
Kidney. Fresh and frozen
Tissues. Formalin fixed

**Laboratory procedures**

Serology: Microscopic agglutination test
Culture: Urine and renal homogenate on specialised media (e.g. Fletcher’s)
PCR: Renal homogenate
Histological examination: H&E, silver and immunofluorescent staining
**Treatment**

Therapy for individual seals with leptospirosis is aimed at maintaining fluid balance while administering antibiotic therapy. In the instance of an outbreak of leptospirosis in a free-ranging population widespread treatment would not be possible.

**Prevention and control**

Control of the introduction of leptospirosis to Australian seal populations from terrestrial animals is not possible. Leptospires can survive in water and moist soils and seals are known to occasionally travel up rivers and creeks thereby potentially exposing them to infection. Control of leptospirosis in some domestic animal species is assisted by the use of vaccination. This strategy would be extremely difficult to apply to a wild seal population and might only be considered if the disease was posing a threat to population viability.

Seals diagnosed with leptospirosis should be regarded as potentially infectious to humans (see below) and other mammals. Control of the public health risk centres around appropriate personal protection equipment for groups likely to be exposed to respiratory tract secretions from diseased animals.

Leptospires are sensitive to most antiseptics and washing of hands with chlorhexidine gluconate and chemical sterilisation of equipment should be practiced as a minimum standard after handling free-ranging marine mammals.

**Surveillance and management**

Wildlife disease surveillance in Australia is coordinated by the 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.

**Statistics**

There are no cases of leptospirosis in seals listed on the National Wildlife Health Surveillance Database [www.wildlifehealthaustralia.com.au](http://www.wildlifehealthaustralia.com.au). NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au.

**Research**

They key research question is whether Australian seals have leptospirosis circulating within their populations. While one limited serological survey has been conducted in Australian fur seals, this research activity needs to be extended to seal populations throughout Australia.

Investigation of unusual mortality events in seals and exclusion of leptospirosis would be worthwhile.
Human health implications

Leptospirosis is a significant disease of humans globally and sporadic cases occur in Australia (www.wildlifehealthaustralia.com.au). Seals with leptospirosis pose a disease risk to humans. At risk groups include staff working in facilities holding captive marine mammals particularly those that accept wild individuals for treatment and rehabilitation. Other groups potentially at risk are research scientists, wildlife officers and members of the public and wildlife carer groups who assist at marine mammal strandings. Appropriate personal protection (gloves, protective clothing and mask) should be employed when conducting post mortem examinations on pinnipeds.

Conclusions

Leptospirosis has a world-wide distribution and is the cause of significant morbidity and mortality in some northern hemisphere seal populations. Therefore, Australian seal species are potentially at risk from this pathogen. Knowledge whether Australian seals have been exposed to leptospires is incomplete. Leptospirosis is a potential zoonotic disease so appropriate protective measures should be taken when handling seals exhibiting signs of illness.

References and other information


**Acknowledgements**

We are extremely grateful to those people who had input into this fact sheet and would specifically like to thank Michael Lynch who wrote the initial draft.

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**To provide feedback on this fact sheet**

We are interested in hearing from anyone with information on this condition in wildlife 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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