Phillip Island (Millowl) Little Penguin (*Eudyptula minor*)

Disease Risk Analysis

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Source: Visit Victoria

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Executive summary

- The little penguin is listed as 'Least Concern' by the IUCN Red list 2020.
- Key threatening processes include habitat destruction, human disturbance, pollution, climate change, predation by introduced species, and competition with fisheries and food availability.
- The process of Disease Risk Analysis (DRA) is used to identify significant disease risks and to propose mitigation measures which may impact little penguins, other wildlife, domestic animals and/ or humans.
- It was agreed by stakeholders that a wildlife DRA for little penguins should be undertaken in order to assess disease risks and provide management recommendations.
- A formal DRA, following a globally recommended framework, was undertaken by the University of Melbourne with input from a wide range of stakeholders, which included a one-day stakeholder workshop on November 30 2023 at Werribee, Victoria
- The little penguin DRA process identified 80 hazards of concern (both infectious and non-infectious) and ranked each hazard in importance.
- This comprised five high risk hazards (HPAI, oiling, predation (marine and terrestrial) and wildfire); and twenty-one medium risk hazards (Saumarez reef virus, APMV1, avian chlamydia, avian cholera, babesiosis, infection with *Plasmodium* sp., toxoplasmosis, infection with *Contracaecum* sp., infection with *Mawsonotrema eudyptulae ,* ticks, algal toxins, persistent organic pollutants and other emerging contaminants, per- and polyfluorinated substances, environmental stressors including climate change, habitat loss (marine and terrestrial), marine and terrestrial heat stress, reduction in food supplies, starvation, and tourism).
- Oil, wildfire, and predation (marine and terrestrial) were the most significant non-infectious hazards identified, however, PINP already has contingency plans in place for these hazards.
- Six moderate-high risk human health hazards were identified (HPAI, West Nile virus (neither currently present in Australia), Kunjin virus, Murray Valley encephalitis, Ross River virus, and avian chlamydia.
- The ongoing availability of penguin prey species considering natural fluctuations with predictions in climate change and commercial fishing, requires further active management and consideration of applications for further potential marine protected area classification.
- Active and passive population health and disease monitoring is required to better quantify and manage infectious disease threats in the colony.
- A detailed risk assessment was undertaken on all high and medium priority hazards (if risks were not already managed through other, established processes).
- It was determined that preventative measures should be employed to reduce risks in all infectious and non-infectious hazards that underwent assessment.
- Based on the risk assessment, general and specific disease risk management recommendations were made. These include recommendations for increased monitoring, biosecurity practices, veterinary examination, diagnostic sampling and ongoing review and active management for identified hazards with a focus on HPAI as the most time sensitive hazard.
- It was concluded that increased passive and active disease monitoring at Phillip Island (Millowl) is warranted to assess current disease prevalence. These data should then be used to assess and further quantify disease risk; the general and specific disease risk management recommendations should be implemented.
- Disease risk management should include targeted disease screening and necropsy of all individuals that are found recently dead, especially (though not limited to) if there is no obvious cause of death.
- General biosecurity and health and disease screening recommendations were included.
- Recommendations were also made regarding identified knowledge gaps with specific recommendations for further health and environmental research.
- Refinement of ongoing management should be based on an annual review which should be formally documented and discussed with relevant staff.

1 Contents

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2 Introduction

2.1 The little penguin (Eudyptula minor)

The order Sphenisciformes (penguins) includes 18 species of flightless, piscivorous birds representing six genera (Bodley and Schmitt 2014). Of these species, 12 (66.7%) have decreasing population trends (International Union for the Conservation of Nature (IUCN 2020). The little penguin (*Eudyptula minor*), also referred to as fairy penguin, little blue penguin or blue penguin, is the smallest penguin species, standing between 30-40 cm tall and weighing between 1-1.2kg. While they spend most of their life at sea, breeding occurs on land. There has been considerable discussion about the taxonomy of little penguins over time (e.g. Kinsky and Falla 1976), and Grosser et al. (2015) went as far as suggesting that they are in fact two different species: *Eudyptulae minor* (originated in New Zealand) and *E. noveahollandiae* (originating in Australia but having expanded to southern New Zealand). For the purposes of this DRA, we will be adopting the convention by Birdlife and IUCN to refer to *E. minor* as a single species, distributed around southern Australia and New Zealand. In Australia, it is distributed from Carnac Island off Western Australia (WA), along the southern coastline to South Solitary Island New South Wales (NSW), and further south to Tasmania. Little penguins, Kororā, are also native to New Zealand and the Chatham Islands in the Pacific Ocean (Mattern and Wilson 2018). The LP population at Phillip Island (from here on also referred to by its traditional name Millowl), which is approximately 120km from central Melbourne off the coast of Victoria, is thought to be Australia's largest colony of little penguins, with recent estimates of up to 37,000 breeding penguins on the Summerland Peninsula (D. Sutherland, unpublished data); however, several of the species' populations are data deficient. The Bunurong/Boon Wurrung Peoples are the traditional owners of Summerland Peninsula.

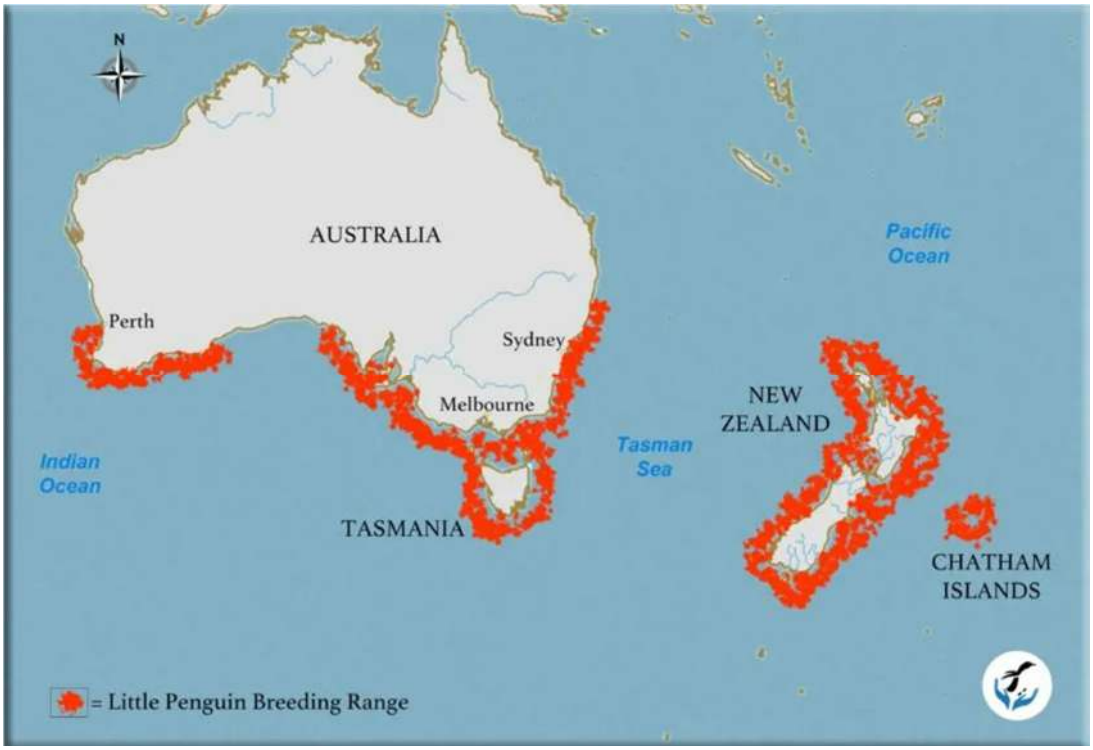


Figure 1. Distribution of little penguins (map from Penguins International 2023)

Conservation status and threats

Populations of LP's are considered stable, and 'Least Concern', both at the national and global level (IUCN 2020) (Figure 2), as their range and population size is below the threshold for Vulnerable criteria (which is

<20,000 km^2 and <10,000 mature individuals showing continuing decline) (IUCN 2020). They are listed as 'marine', under the *Environment Protection and Biodiversity Conservation Act 1999* (*EPBC Act 1999*) (Sutton 2022). Nonetheless, penguin numbers at Phillip Island (Millowl) have varied substantially over the last 100 years, ranging from an estimate of 200,000 adults in 1917 (Nicholls 1918) to a much-reduced estimate of approximately 12,000 in a study from the 1970s (Harris and Bode 1981), at which time the annual mortality rate was estimated at 14.2%, with an average life expectancy of 6.5 years (Reilly and Cullen 1979). Since then, little penguin numbers at Phillip Island (Millowl) have recovered significantly, and wild little penguins as old as 25 have been observed at the site (penguins.org.au).

Figure 2. The global conservation listing of little penguins is of 'Least Concern' (IUCN 2020).

Globally, there are estimated to be approximately 500,000 breeding adults (IUCN 2020). However, the overall stable trend masks localised population fluctuations, with population trends for 60% of the sites not being known (IUCN 2020). Furthermore, not all colonies have active conservation measures, with some of these colonies experiencing significant decreases in population size (Dann 1996; Stevenson and Woehler 2007). In Australia, a decrease in population size has been reported in NSW and South Australia (SA) while Tasmania is data deficient. The population at Penguin Island (WA) is estimated to have decreased from 2400 (in 2007) to an estimated 300 in 2019 (Cannell et al. 2011; Cannell 2018; Cannell 2020). Several Victorian populations are currently thriving, numbers on Phillip Island (Millowl) are estimated at up to 37,000 (D. Sutherland, unpublished data; Sutherland and Dann 2012) and at St Kilda 1,400 (CES 2021), although other populations are decreasing (e.g. Gabo and Middle Islands). The conservation status of little penguins In New Zealand is less well understood, because there is no consistent monitoring in many regions. Nonetheless, trends appear to be variable depending on location, with the species apparently decreasing along the South Island's Banks Peninsula, stable or increasing around Oamaru (Mattern and Wilson 2018, IUCN 2020).

Little penguins as seabirds are susceptible to many threats; key threatening processes are often cumulative and occur both on land and at sea (where little penguins spend approximately 80% of their time). These processes include habitat (including marine) degradation and destruction, human disturbance, pollution, climate change-related processes such as changing ocean temperature, predation by introduced species, and direct competition with fisheries affecting availability of prey species (Trathan et al. 2015; IUCN 2020; Gimeno et al. 2024). Increasing ocean temperature, frequency of marine heat waves and strong winds have been linked to negative effects on adult foraging and chick survival (Cullen et al. 2009, Cannell et al. 2012, IUCN 2020). Marine heat waves are in fact thought to have their most severe effects on range-restricted species such as the little penguin (Woehler and Hobday 2023). Although disease is a potential risk for all penguins, retrospective review has found disease events typically occur subsequent to key threatening processes (Dann et al. 2000). Threatening processes may impact on wildlife populations directly or indirectly, on their own or in combination; for example, immune suppression may occur as a result of nutritional stress, subsequently leading to increased morbidity and mortality (e.g. Ezenwa 2004). Penguin populations are already subject to the spread of introduced pathogens and toxicities (Kane et al. 2010;), and over time, the potential for disease to become a key threatening process may be of increased likelihood.

As population sizes reduce, disease is of potentially greater risk, as individuals may become compromised by loss of genetic diversity (Lyles and Dobson 1993), which can result in a reduced ability to respond to new pathogens. Subsequently, future survival and adaptability of species depends heavily on retaining or improving genetic variation and connectivity (Kardos et al. 2021). High genetic diversity is essential for providing species with the opportunity to adapt to environmental change (e.g. climate change or emerging

diseases) and avoid the negative impacts of inbreeding. Small, fragmented populations are more susceptible to inbreeding, which can result in reduced overall fitness due to the expression of deleterious recessive alleles, potentially leading to reduced survival and reproductive effort (Leedale et al. 2020). Subsequently, any significant population decline, including those resulting from disease outbreaks, may have important flow-on effects. The genetic variation and connectivity of LPs from 22 sites in WA, SA and NSW was studied by Vardeh et al. (2023). Using microsatellites, they found no evidence of critically low variation. However, they detected that penguins from the west and east appeared to represent two main, genetically distinct, groups. Nonetheless, the presence of high and complex dispersal patterns across their Australian range, including genetic similarity between locations that were not adjacent, suggests interdependence of LP populations and the presence of valuable genetic variation.

Management of little penguins

In Australia and New Zealand, LPs are fully protected and occur in many national and state parks which are regulated. While many of the management actions focus on the time penguins spend on land, it is important to recognise that management of the species at sea is of at least equal importance. In WA, little penguins inhabit Penguin, Breaksea and Cheyne Islands and Shoalwater Island Marine Park; the latter is managed through a Marine Park Management Plan (Birdlife International 2023). Management of this colony has addressed a number of issues: boardwalks have been erected to reduce disturbance of sensitive breeding habitat; speed limits have been imposed on vessels travelling in local waters; and the island is closed to visitors during the peak of the breeding season from mid-June to mid-September (Sutton 2022). The Manly population was listed as endangered by the NSW Scientific Committee in 1997, and this placed restrictions on landholder activities and development in the area. Other conservation management actions at the site include rat control through baiting, weed eradication, minimising access to areas through habitat management, community education and participation, and predator exclusion, particularly dogs, cats and foxes (NWPS 2000). The population has also been augmented through translocations of fledglings (Carlile et al. 2015). Another conservation action is the provision of artificial nestboxes (including ongoing optimisation of their design), which occurs at many sites throughout the range; this has been shown to potentially increase breeding success (Houston 1999; Sutherland et al. 2014). In South Australia, priorities for management include a focus on introduced predators, interactions with long-nosed fur seals (*Arctocephalus forsteri*), and the protection of penguins at tourism sites and the colonies in the south-east of the state (Dann 2016). Several LP colonies experience formalised ecotourism, with the colony at Phillip Island (Millowl) experiencing the highest visitor numbers (719,617 visitors in 2018-19, which was, presumably temporarily, reduced as a result of Covid19 to 218,960 in 2021-22) (Phillip Island Nature Park 2019 and 2022), followed by the Penguin Island WA colony, which reports up to 130,000 visitors annually (Sutton 2022). Several volunteer and research groups are also actively involved in monitoring and protecting sites in Australia and New Zealand (Birdlife 2023).

Management of the Phillip Island (Millowl) penguin population has concentrated on the elimination or mitigation of threats posed by introduced predators (now a fox-free habitat), habitat destruction and human disturbance (the Summerland Peninsula housing estate was part of a large buy-back scheme funded by the State Government of Victoria and was subsequently demolished and returned to penguin habitat), and this has resulted in stabilisation and an overall increase in population numbers (Sutherland and Dann 2014). The Nature Park operates a rehabilitation centre at the Penguin Parade to primarily treat penguins and seabirds from Phillip Island (Millowl) and other parts of Victoria. Veterinary support is provided by local veterinarians and pathology support by the University of Melbourne. Recently, a targeted disease risk analysis was undertaken by Scheelings (2022) to prioritise highlighted current and potential future impacts of infectious disease. This analysis was an important first step towards undertaking a holistic disease risk analysis which aims to identify, prioritise and recommend management plans for both infectious and non-infectious hazards to the Phillip Island (Millowl) and broader Australasian LP population.

2.2 Disease history of little penguins

General impacts of disease

The biology and ecology of LPs in certain populations, has been extensively studied. The LP population at Phillip Island (Millowl) has been studied since 1968, with extensive data available (Ramirez et al. 2021; Collins et al. 1999; Dann 1991; Reilly and Cullen 1968). Likewise, the presence of artificial nest boxes at the colony at Penguin Island WA has facilitated extensive research efforts at that site (Sutton 2022). However, the health and disease status of LPs has not been prioritised to the same extent. Disease occurs naturally in all populations and hosts can have long evolutionary associations with certain parasites without causing disease (Hudson et al. 2002). However, diseases, either directly or in synergy with other factors, can also represent significant threats to wild animal populations, responsible for population declines (Preece et al. 2017). All aspects of population dynamics may be affected by the impacts of disease, however, it is particularly increased mortality and/or decreased reproduction reduction which is most likely associated with species decline or extinction (Tompkins et al. 2015). Examples of species extinctions shown to be associated with disease include chytridiomycosis, which is thought to have directly led to the decline or extinction of over 500 amphibian species (including the extinction of seven Australian frog species) (Scheele et al. 2019) and white nose syndrome, which directly led to 90% declines of overall population numbers of three North American bat species (Cheng et al. 2021).

Population crashes

Little penguins are known to experience seasonally increased mortality rates during natural periods of high energy demand, such as the breeding and moulting season (Norman 1992, Rensburg 2010). However, additional large-scale mortality events appear to periodically affect little penguin populations throughout the Australasian region, leading to population crashes (also referred to as 'wrecks') (Norman 1992, Rensburg 2010). Affected penguins typically show signs of starvation, evidenced by low fat stores and empty gastrointestinal tracts during necropsy (Rensburg 2010). In sea birds, such die-offs are often the sequelae of food shortages and extreme weather events (Dann et al. 2000; Frederiksen et al. 2008, Rensburg 2010), which may also exacerbate the impacts of parasites and disease (Obendorf and McColl 1980). Immunocompromised penguins and those at either end of the age spectrum seem particularly susceptible (Norman 1992). Many of an increased number of LP mortalities in the late 1970s were thought to be due to parasitic infections exacerbating starvation (Obendorf and McColl 1980).

Previous disease investigations in little penguins

Norman's (2006) PhD thesis provided an excellent review of gastric parasitism in the LP. Necropsy examinations were undertaken between 1992-1995 to identify internal parasites and investigate relationships between pathogenesis and mortality given prior mortality events on the Victorian coast. The identity of the ascaridoid nematode *Contracaecum eudyptulae* associated with gastric ulcers was confirmed at a prevalence of 68%, while *Tetrabothrius lutzi* and *Tetrabothrius eudyptidis* with a combined prevalence of 48% formed the core internal parasites. Fifteen other species of nematodes, trematodes and protozoa were reported at lower prevalence (Norman 2006). Rensburg (2010) completed a Masters thesis on parasitism, disease and breeding ecology of LPs on Tiritiri Matangi Island, New Zealand which provided an excellent review of penguin disease in the Oceania region. Melanie Wells recently submitted a PhD thesis which completed a health surveillance of little penguins in Tasmania (Iutruwita); she completed active surveillance for pollutants (PFAS), blood parasites, ectoparasites and haematological reference ranges (Wells, M. pers comm). The results from this work are being prepared for publication. Scheelings (2022) conducted a qualitative disease risk analysis for the likelihood of incursion and spread of avian malaria, avian cholera, avian poxvirus, and avian influenza in the Phillip Island (Millowl) population of LPs. The risk assessment considered the likely impacts of disease on this population following incursion from both short- and long-term perspectives. However, a limitation of this analysis was the limited amount of data available for many

of the highlighted diseases in Australian populations of sea birds, including penguins. Despite the paucity of information, it was concluded that the diseases of greatest concern, and the most likely to cause serious disease outbreak in LPs at Phillip Island (Millowl) at that time were avian cholera and avian malaria. Avian cholera, caused by the bacteria *Pasteurella multocida*, may spill over into wild bird populations directly from domestic poultry or via unaffected carrier birds, is highly contagious and can cause mass mortalities in affected populations; such events have previously been reported from macaroni penguins (*Eudyptes chrysolophus*) (Cooper et al. 2009). On the other hand, avian malaria is an infection with blood-borne parasites (e.g. *Plasmodium* spp.); infection with haemoparasites is likely present but does not appear to be highly prevalent in Victorian LPs, however, mortality events may be associated with newly emerging species or concurrent immune suppression (Scheelings 2022).

The threat of avian influenza

More recently, a new threat has emerged. In June 2022, an outbreak of Eurasian H5 High pathogenicity avian Influenza (HPAI) was detected in the northern hemisphere, affecting a wide variety of species including gannets, geese, terns, gulls, skuas, ducks, eagles, puffins, and penguins resulting in high levels of mortality (National Science Foundation 2023). Subsequently, the virus spread to the southern hemisphere, with outbreaks detected in sea birds and marine mammals in Chile and Peru in mid to late 2023 (National Science Foundation 2023). A significant concern for Australasian sea birds, including LPs, is that HPAI was confirmed in brown skua populations on Bird Island, South Georgia in October 2023– the first known cases in the Antarctic region, followed by infections in penguins (gentoo and king penguins), elephant and fur seals in early 2024 (British Antarctic Survey 2024). A key risk is the possibility of migratory bird species transmitting HPAI to other bird, but also mammal, species in the Oceania and Antarctic region, especially between September and November, which coincides with the arrival of breeding seabirds including penguins (Dewar et al. 2022, PAHO 2023). In addition, elephant seals, sub-Antarctic fur seals and occasionally crabeater seals commonly frequent the region (R. McIntosh, pers comm.) and could potentially bring with them HPAI from the Antarctic and sub-Antarctic regions. This emerging disease threat was a significant driver to assess and recommend disease risk management strategies for the LP population at Phillip Island (Millowl) and more broadly contextualise the threat within the little penguin populations in the Australasian region. In anticipation of this threat, there is a recent, existing local response plan for HPAI at Phillip Island Nature Parks, which also overlaps a Ramsar wetland; the plan clearly outlines relevant precautions and actions for the site (Renwick 2022).

Infectious diseases

Penguins are susceptible to an array of viral, fungal, bacterial and parasitic infections; Rensburg (2010) provided an excellent review of known organisms relevant to little penguins. Exposure to several virus families, including birna-, flavi-, orthomyxo- and paramyxoviruses has been documented in penguins (Major et al. 2009). Similarly, a range of bacteria, including *Borrelia* sp., *Campylobacter* sp., *Chlamydia* sp., *Pasteurella* sp. and *Salmonella* sp., as well as the fungus *Aspergillus* sp. have been found in penguins species (Clarke and Kerry 1993; Barbosa and Palacios 2009). Aspergillus infections are usually found in captive animals, as a result of secondary infection due stress and/or concurrent disease (Stoskopf and Beall 1980; Reece et al. 1992). Nonetheless, some cases have been found in wild LPs, causing or contributing to their death (Obendorf and McColl 1980; Morgan et al. 1981), and free-living LPs in New Zealand appear to have a relatively high level of exposure to the organism based on seroprevalence (Graczyk and Cockrem 1995). Recently, a cluster of cases of toxoplasmosis, with concurrent *Haemoproteus* sp. was reported in 12 LPs from Penguin Island in Western Australia (Campbell et al. 2022); in addition, acute death from toxoplasmosis was reported from a captive LP in Tasmania (Iutruwita) in the 1990s (Mason et al. 1991).

Knowledge gaps

Most disease investigations conducted to date have comprised retrospective review of mortality events or necropsy records rather than active monitoring in healthy populations. Ongoing, strategic and systematic

surveillance of LPs and, ideally, sympatric species, is therefore required to identify endemic disease and detect exotic diseases should they occur (Clarke and Kerry 1993). It is also important to realise that identification of parasite species associated with wild animals is not complete. Therefore, it is possible wild penguins may have undetected or unknown parasites which could lead to future disease outbreaks.

Diagnostic challenges

The diagnosis of disease in wild penguins is often difficult, as while we can detect antibody response, we do not always know what is 'normal' (commensal). For example, serological tests provide evidence of exposure (infection) to the parasite rather than disease, and we often need to conduct multiple testing methodologies in conjunction with clinical examination to detect and infer disease. Furthermore, many of the commercial tests used are designed for poultry and not penguins. For example, serology developed to detect exposure to specific pathogens based on binding to chicken antibodies (immunoglobulin), will only be applicable to penguins if it also binds to their antibodies. In addition, the diagnostic performance of tests, relating to their sensitivity (ability to correctly detect positives) and specificity (ability to correctly detect negatives), can vary substantially between species, and this is usually not validate for penguin species (Parsons et al. 2016). However, it is worth noting this is a common challenge in wildlife population health studies, and other studies on the antigenic properties of penguin immunoglobulins suggest that the assumptions on similarity with chicken immunoglobulins are valid (Bizelli et al. 2015). Nonetheless, this is a methodological limitation of diagnostics in these species (Karesh et al. 1999) and should be acknowledged.

In this report the little penguin is recognised as a 'host' and a complete 'biological package' naturally containing multiple parasites (viral, bacteria, fungal, ecto- and endoparasites) which may become pathological (disease causing) if the appropriate host and environmental conditions prevail (Corn and Nettles 2001). Hosts coexist with their unique suite of parasites; they continually adapt to each other as part of an "arms race", which impacts and potentially enhances their fitness and each other's survival (Keusch and Migasena 1982). Almost all wildlife hosts are subject to complex parasitic co-infections (polyparasitism), which are generally poorly understood, and, in addition, other factors affecting immune response, such as non-infectious threats (e.g. pollutants, human disturbance, starvation), can be additive and reduce disease resistance. Current and ongoing surveillance is needed to effectively assess associated risks.

2.3 Disease Risk Analysis and definitions

The process of disease risk analysis (DRA) aims to identify significant disease risks and subsequently progresses to proposing appropriate risk mitigation strategies. The process is structured and evidence-based, addressing the impacts of both infectious and non-infectious diseases on ecosystems and the living organisms within it (especially, though not exclusively, wildlife, domesticated animals and humans).

Conservation translocations are a common reason for conducting a DRA (for example Jakob-Hoff et al. 2014*b,* 2016), but it can be applied to any context that examines the role of disease in populations, for example in native mammal declines (Pacioni et al. 2015; Reiss et al. 2015). This DRA will define and discuss disease and parasites and current practical methodology to undertake DRA in the Australian context. The risk analysis framework is starts with a problem description, progressing then to an as complete as possible hazard identification process, followed by risk assessment, risk management suggestions, implementation and finally review and risk communication (Jakob-Hoff et al. 2014*a*) (Figure 3).

In the past, the terminology 'risk assessment' and 'risk analysis' were used interchangeably. However, the term 'risk assessment' is a sub-component of DRA and the use of this term (and the process alone) should be discouraged in favour of a complete risk analysis.

The below definitions are adopted from Jakob-Hoff et al. (2014a):

Disease, constitutes 'Any impairment of the normal structural or physiological state of a living organism resulting from its physiological response to a hazard'.

A **hazard** is defined 'as a biological, chemical or physical agent, or a condition of an animal or animal product with the potential to cause an adverse health effect'. Hazards include parasites (as defined below) and non-infectious agents such as toxins and trauma.

Parasites are 'agents that live on or within a host and that survive at the expense of the host regardless of whether disease follows or not. This includes both microparasites (viruses, bacteria, fungi, protozoa) and macroparasites (helminths, parasitic arthropods)'

Pathogens are 'described as any disease-causing parasite'.

Morbidity is illness due to a specific disease or health condition.

Figure 3. The Disease Risk Analysis framework Jakob-Hoff (2014*a*)

'Ideally, DRA involves a multi-disciplinary team undertaking risk analysis to identify hazards that may enter a specified animal population, the likelihood of such introductions occurring, their consequences, and the measures that may be applied to mitigate either likelihood of introduction or the magnitude of consequences' (Jakob-Hoff et al. 2014*a*).

The methodologies for disease risk assessment and mitigation have been published by many authors (Davidson and Nettles 1992; Leighton 2002; Armstrong et al. 2003; Murray et al. 2004; Travis et al. 2006; Miller 2007; Sainsbury et al. 2012; Sainsbury and Vaughan-Higgins 2012, Jakob-Hoff et al. 2014*a*, Hartley and Sainsbury 2017, Vaughan-Higgins et al. 2021).

However, the gold standard for wildlife disease risk analysis remains the IUCN/OIE manual (Jakob-Hoff et al. 2014*a*) and the IUCN/OIE Guidelines (WHO/OIE 2014) for Wildlife Disease Risk Analysis. The latter was compiled by the IUCN Species Survival Commission's (SSC) Wildlife Health Specialist Group (WHSG), the Conservation Breeding Specialist Group (CBSG), the Reintroduction Specialist Group (RSG) and the Invasive Species Specialist Group (ISSG).

3 Methodology

This DRA primarily focuses on the logical, stepwise manner of the IUCN Guidelines for Wildlife Disease Risk Analysis (Jakob-Hoff et al. 2014*a*). This section subsequently quotes, adapts and cites the text of that resource, which outlines the process concisely and succinctly.

3.1 Problem description

Questions: 'What is the specific question for this DRA?' and 'What kind of risk analysis is needed?'

Method: Outlines the background and context of the problem.

- The DRA Question is formulated in consideration of the problem the DRA is being undertaken to address. This should include a synopsis of relevant information needed to describe the problem and provides context to justify the DRA.

Identify the goal, scope and focus of the DRA.

- Goal - to identify and assess the likelihood of the hazard(s) being introduced and spreading or becoming established, together with the likelihood of and the likely magnitude of the potential consequences for wild animal, domestic animal or human health as a result of the activity and to recommend risk mitigation measures if appropriate.
- Scope - will consider where the boundaries of the DRA lie (e.g. the species, populations and geographic areas of interest, time frame). It could also define the limitations under which the DRA is conducted such as availability of sources of information (including expertise and stakeholder input).
- Focus - The focus narrows the scope to determine the specific purpose of the DRA e.g. '*The focus of this DRA is to identify, assess and evaluate mitigation options for the potential health impacts associated with the acquisition and captive management of species X*.

State assumptions and limitations and specify acceptable level of risk. Zero risk is seldom, if ever, attainable, some degree of risk is unavoidable therefore trade-offs are often needed between what level of risk is acceptable and what is not. For example, disease risks posed by relocation of wild animals into a conservation reserve may be acceptable to those ecologists concerned with maintenance of a genetically diverse population of endangered animals, but maybe considered unacceptable to neighbouring farmers concerned with the health of their livestock.

Therefore, the risk to different populations must also be considered in the context of the DRA process:

- *Who or what is at risk?*
- *What are the consequences to individuals and the population?*
- *What is the acceptable level of risk within the biological system (on Phillip Island (Millowl))?*

The acceptable level of risk will determine what hazards are retained for determination of suitable risk mitigation procedures:

- Any hazard exceeding the acceptable level of risk for specified population will be retained for determining the most suitable risk mitigation measures.
- The proposed acceptable level of risk for each population of interest, is based on the 'first do no harm' principle.

In some cases, a site visit will also be required to accurately assess the risks and provide practical and logistically sound disease risk management.

3.2 Risk communication

(applies at every DRA step)

Questions: 'Who are the key stakeholders?', 'Who has an interest?', 'Who has knowledge or expertise to contribute?' and 'Who can influence the implementation of recommendations arising from the DRA?'

Method:
Involves continuous communication between stakeholders and engagement with relevant experts and stakeholders to maximise the quality of analysis and the probability that the recommendations arising from the DRA will be implemented.

3.3 Hazard identification

Questions: 'What can cause disease in the population of concern?', 'How can this happen?' and 'What are the potential consequences?'

Method:

- Identify all possible hazards of concern and categorise into '**infectious'** and **'non- infectious'**.
- **Infectious:** Disease due to the presence of an infectious agent that is capable of being transmitted to another host, e.g. avian influenza, this includes 'zoonotic' disease.
- **Toxic**: Disease caused by a toxin or poison, e.g. avian botulism and lead poisoning.
- **Nutritional:** Disease caused by nutritional imbalance or deficiency, e.g. starvation and metabolic bone disease.
- **Traumatic**: Disease caused by physical injury, e.g. following a collision, and electrocution.
- **Immunological:** Disease caused by disruption or abnormal function of the immune system, e.g. allergy.
- **Developmental:** Disease that interrupts normal development in growing animals. A developmental disease may affect a specific part of the body or affect multiple systems.
- **Congenital/ genetic**: Disease that is inherited genetically or caused by loss in heterozygosity, e.g. infertility due to the consequences of in-breeding.

- **Neoplastic:** Disease caused by abnormal new growth of tissue, a tumour, e.g. cancer.

- Establish criteria for ranking the importance of each hazard within the bounds of the goal.
- Consider potential direct and indirect consequences of hazards to help decide which should be subjected to full risk assessment e.g. consequences to/for: health (animal and human); welfare; environmental and ecological; social and psychological; national security (e.g. notifiable diseases).
- Exclude hazards with zero or negligible probability of release or exposure.
- Consider construction of graphical models e.g. a scenario tree for high priority hazards of concern, to facilitate identification of the various biological pathways leading to exposure of the susceptible animals or people to the hazard, as well as potential 'outbreak' scenarios.

3.4 Workshop

A stakeholder workshop was held at Werribee, Victoria 30 November 2023, both in person and online. The participants represented the major institutions involved in the DRA. The professions of the attendees included research scientists active and retired, field biologists, field ecologists, geneticists, independent consultants, researchers, and zoo and wildlife veterinarians. The agenda and minutes of the workshop are available in Appendix 2 and 2.1.

The aims of the workshop included:

- Present the draft hazard list and receive feedback on the perceived and actual significance of these hazards.
- Facilitate communication amongst stakeholders.
- Seek advice and opinions for inclusion and exclusion of hazards in the DRA.
- Formulate a list of higher priority hazards requiring full risk assessment.
- Discuss practical disease risk management principles and the need for targeted disease screening to inform the DRA.

3.5 Risk assessment

Questions: 'What is the likelihood and consequences of a specified hazard occurring within an identified pathway or event?'

Method:
For each hazard of concern construct a table to display:

- **Entry assessment** – an estimate of the likelihood of the hazard being introduced to LPs into the area.
- **Exposure assessment** – estimates the likelihood of susceptible animals being exposed to the hazard, becoming affected (parasite hazards) and disseminating (infectious) hazards.
- **Consequence assessment** – estimates the likely magnitude of potential biological, environmental and economic consequences associated with the entry, establishment or spread of the hazard and the likelihood of their occurrence. Includes consequences for the individuals moved, population of same and other species and for the wider ecosystem.
- **Risk estimation** – summarises the entry, exposure and consequence assessments to provide an overall measure of risk.

3.6 Risk management

Questions: 'What can be done to minimise the likelihood of a hazardous event?' and 'What can be done to minimise the consequences once a hazardous event has happened?'

Method:

- Identify and evaluate management options that can be implemented to minimise identified risks e.g. screening for parasite hazards, minimising stress (low stocking densities), hygiene (cleaning and disinfection), prophylactic or other medications, environmental treatments, vector control, quarantine, isolation.

Option evaluation – expert consideration of options for feasibility and effectiveness. Ideally, options should be feasible and highly effective.

3.7 Implementation, monitoring and review

Questions: 'How will the selected risk management options be implemented?' and once implemented, 'Are the risk management actions having the desired effect?' and if not, 'How can they be improved?'

Method:

- Formulate the action and contingency plan and establish a process and timeline for the monitoring, evaluation and review of risk management actions.
- Detail plans of actions to be taken, why, when and by who, and the associated resources (time, money, people, equipment).
- Monitor risk management measures to ensure that they are achieving the intended results.
- Develop processes to evaluate the effectiveness and practicality of risk management options - ideally annually.
- The review may result in a clearer understanding of the problem and enable refinement of the DRA.

3.8 Problem description, risk communication and DRA framework

The DRA outline and framework was tabulated to include the **problem description, risk assessment, risk management, and risk communication strategies** (Table 1). The text within all the tables in this section includes quotation and adaptation of the original sources cited.

Table 1. Outline and framework of the little penguin DRA including the problem description, hazard identification and prioritisation, risk assessment, risk management and risk communication (text adapted for little penguins from IUCN Guidelines for Wildlife Disease Risk Analysis by Jakob-Hoff et al

Problem description	
• The little penguin is listed as 'Least Concern' by the IUCN Red list 2020. • Key threatening processes include habitat destruction, human disturbance, pollution, climate change, predation by introduced species, and direct competition with fisheries. • Little penguins exhibit communal behaviours such as a large repertoire of social calls and 'rafting' together at sea, which suggest they are adapted to living in large colonies. • Health and disease issues have not been reported to same extent as biology and ecology of the species. • Aim of DRA – to identify key health hazards to the Phillip Island (Millowl) little penguin population and provide practical disease risk management recommendations to key stakeholders and to identify knowledge gaps which require further research effort.	
DRA questions	What are the risks to health and of disease from identified health hazards, that constitute a threat to free-living little penguin populations? How can these risks be minimised?
DRA goal	Develop a disease risk management strategy for little penguins based on structured, evidence-based analysis of current information to promote health of the population.
Scope and focus	SCOPE: known infectious and non-infectious diseases of captive and free-living little penguins and Sphenisciformes known to have a broad host range. To conduct a qualitative analysis of relevant literature (and other available information) on the susceptibility of LPs to infectious or non-infectious disease currently present, or that could be introduced to Phillip Island (Millowl). This analysis will also include an analysis of the susceptibility of humans and domestic animals (livestock and pets) to infectious and non-infectious disease currently present, or that could be introduced to Phillip Island (Millowl). Prioritisation of these identified hazards will occur with stakeholder consultation.

Assumptions	LPs are susceptible to health hazards reported in Sphenisciformes and are susceptible to pathogens demonstrated to have a broad host range in avian species. Available data combined with the analytical and decision-making processes will enable reasonable decisions to be made to minimise health risks.
Limitations	Limitations of baseline data, limitations of existing knowledge of disease and health in Sphenisciformes. Limited data and information available in peer-reviewed publications and open access sources. Reasonable understanding of the range and epidemiology of potential pathogens of LPs
Hazard identification and prioritisation	
• Review literature and other available data to identify hazards (infectious and non-infectious). • Collate information against key prioritisation questions. • Categorise hazards, in terms of likelihood and consequence of exposure (low, moderate or high). • Exclude hazards with low probability of release or exposure. Only high and moderate risk hazards will have a risk assessment undertaken. Present and review findings at November 30 workshop with stakeholder input.	
Risk assessment	
• All moderate and high priority hazards (not excluded) will have detailed information summarised on key areas such as host range, impact, transmission, and consequence. These findings will be collated and tabulated into the following sub-headings. • **Entry assessment** – an estimate of the likelihood of Phillip Island (Millowl) LPs introducing the hazard into an area. • **Exposure assessment** – estimates the likelihood of susceptible animals being exposed to the hazard, becoming infected (parasite hazards) and disseminating the hazard at the release site. • **Consequence assessment** – estimates the likely magnitude of potential biological, environmental and economic consequences associated with the entry, establishment or spread of the hazard and the likelihood of their occurrence. Includes consequences for the individuals moved, population of same and other species and for the wider ecosystem at the destination. • **Risk estimation** – summarises the entry, exposure and consequence assessments to provide an overall measure of risk.	
Risk management	

• **Identify and evaluate** the most practical and effective management options to minimise each risk e.g. disease screening, animal or environmental treatments, quarantine and biosecurity practices. • **Option evaluation** – expert consideration of options for feasibility and effectiveness. Ideally, options should be feasible and highly effective.
Risk communication
• Communicate the rigour of DRA process and key results with stakeholders, actively e.g. through stakeholder meetings, presenting findings at any agreed forums or passively, for example Phillip Island Nature Parks and the broader community. • Full detailed report also to be made available to interested parties and available online.

3.9 Acceptable risk

In discussing populations of interest and acceptable risk we considered the following questions:

- How does Phillip Island Nature Park impact people and how does mortality and morbidity in wild birds affect people?
- What situation would trigger a management response?
- Which scenarios of bird mortalities, population monitoring and diseases would result in significant welfare concerns?
- What values are attached to different components of Phillip Island (Millowl) by different people and organisations?

In particular, we focused on

- Impacts on bird abundance and biodiversity, including effects on reproduction
- Impacts on wider community
- Impacts on economy, given the Penguin Parade is significant from an ecotourism perspective
- Animal welfare in response to wildlife mortality events, especially zoonotic events
- Recreational use of Phillip Island Nature Park
- Reputational damage to organisations responsible for LP management

Population groups of interest were grouped as shown in Table 2.

Table 2. Population groups of interest for the LP DRA

Species Group 2	Wildlife resident at the site (including introduced or feral species) and migrating wildlife
Species Group 3	People and domestic animals (pets)
Species Group 4	Livestock

The following livestock are present on Phillip Island (Millowl): dairy and beef cattle, sheep, goats, pigs, alpacas and chickens. Livestock are also present at nearby Churchill Island. There is a free-range chicken farm (Bimbadeen) at Ventnor, approximately 5km from the Penguin Parade. The closest large scale poultry facility is located at Grantville, approximately 40km from Phillip Island (Millowl).

We then discussed how we could qualify acceptable risk to the highlighted populations which generated much discussion (Table 3).

Table 3. Terminology and classification system for acceptable risk to populations of concern

Terminology and classification system for acceptable risk to LPs and wildlife	
Negligible	No detectable effects on the individual, no detectable population consequences, no socio-economic consequences.
Low	Individual morbidity or mortality, but no detectable population consequences, minimal socio-economic consequences in the short or long term. Low impact on welfare
Moderate	Temporary detectable population decline without risk of extinction from this disease, low socio-economic consequences. No long-term population impact on the expected population growth and/or population levels of birds, short term welfare impacts.
High	High risk of local extirpation, due to significant population decline at unsustainable levels, moderate to major socio-economic consequences. Changed status listing of bird species, impacts multiple bird species, flow on impacts to ecosystem processes. Has a high impact on welfare – many animals affected and prolonged suffering.
Proposed risk classification for humans and domestic animals (pets)	
Low	No effects
Moderate	Self-limiting morbidity, not requiring treatment
High	Individual morbidity, requiring treatment or not self-limiting
Proposed risk classification for livestock	
Low	Minimal effects
Moderate	Short-term treatment, minimal welfare impacts and economic significance
High	Individual morbidity, significant treatment, welfare & economic impacts

It was generally discussed that there was a higher tolerance for livestock disease compared to other population groups such as humans and domestic animals (pets) amongst the stakeholder group, and the former tended to be driven more by economic impact.

The following levels of acceptable risk were assigned to the following identified populations at risk.

1) Little penguins at Phillip Island (Millowl): low to moderate level of disease risk to this population
2) Extant wildlife populations: low to moderate level of disease risk to this population
3) Human and domestic animals (pets): low level of disease risk to these populations
4) Livestock - low to moderate level of disease risk to this population

3.10 Process for hazard identification and prioritisation

Published literature and unpublished reports describing diseases affecting penguin species (Sphenisciformes) were reviewed and used to create a list of disease hazards that may be significant. A review of the national electronic wildlife health information system (eWHIS) (Wildlife Health Australia www.wildlifehealthaustralia.com.au) was undertaken for Sphenisciformes, within all Australian states from Jan 1974 to Jan 2024, with data drawn from both captive and free-ranging birds (WHA 2018*a*). This review generated 1160 LP cases and an additional 252 associated avian cases (data received from WHA on 9th April 2024. We also reviewed all of the necropsy reports from 1990 to Nov 23 collated by Phillip Island Nature Park and contacted experts who have been involved with Sphenisciformes both in the wild and captivity to gather information on diseases not already reported by other means.

Spheniscifformes species referred to in this disease risk analysis include those listed in Table 4. It is acknowledged that not all penguin species are at the same risk, depending on geographic region, levels of anthropogenic impacts, and presence of disease transmitting vectors. It is possible that the immune system of penguins with longer periods of exposure to human contact has adapted more to the presence of relevant pathogens (Ropert-Coudert et al. 2019). However, the process of hazard prioritisation would take these factors into account as well as presence or absence of the hazard in the region.

Table 4. Penguin species, and their conservation status (from Scheelings 2022)

Species		Population Trend	Conservation Status
Emperor penguin	*Aptenodytes forsteri*	Decreasing	Near Threatened
Royal penguin	*Eudyptes schlegeli*	Stable	Near Threatened
Adelie penguin	*Pygoscelis adeliae*	Increasing	Least Concern
Humboldt penguin	*Spheniscus humboldti*	Decreasing	Vulnerable
Magellanic penguin	*Spheniscus magellanicus*	Decreasing	Least Concern
Southern rockhopper penguin	*Eudyptes chrysocome*	Decreasing	Vulnerable
Fiordland penguin	*Eudyptes pachyrhynchus*	Decreasing	Near Threatened
Yellow-eyed penguin	*Megadyptes antipodes*	Decreasing	Endangered
Snares penguin	*Eudyptes robustus*	Stable	Vulnerable
Macaroni penguin	*Eudyptes chrysolophus*	Decreasing	Vulnerable
Galapagos penguin	*Spheniscus mendiculus*	Decreasing	Endangered
Erect-crested penguin	*Eudyptes sclateri*	Decreasing	Endangered
African penguin	*Spheniscus demersus*	Decreasing	Endangered
Chinstrap penguin	*Pygoscelis antarcticus*	Decreasing	Least concern
King penguin	*Aptenodytes patagonicus*	Increasing	Least concern
Gentoo penguin	*Pygoscelis papua*	Stable	Least concern
Little penguin	*Eudyptula minor*	Stable	Least concern
Northern rockhopper penguin	*Eudyptes moseleyi*	Decreasing	Endangered

An important component of this DRA was an understanding of LP behaviour and other species that occupy the same ecological niche. Little penguins exhibit communal behaviours such as a large repertoire of social calls and 'rafting' together at sea, which suggest they are adapted to living in large colonies. Therefore, we can assume high host density and direct contact would facilitate exposure and infection transmission. We also needed to identify sympatric avian species that may act as reservoirs of infection for the LPs on Phillip Island (Millowl). The following species are known to co-inhabit penguin nesting sites (Sheelings 2022; Ekanayake et. Al. 2015a,b) (P. Dann pers comm); little raven are known to have frequent interactions with LPs and engage in nest inspection and predation.

· Short-tailed shearwater (*Ardenna tenuirostris*)
· Crested tern (*Thalasseus bergii*)
· Pacific gull (*Larus pacificus*)
· Kelp gull (*Larus dominicanus*)
· Silver gull (*Chroicocephalus novaehollandiae*)
· Cape Barren goose (*Cereopsis novaehollandiae*)
· Purple swamphen (*Porphyrio porphyrio*)
· Sooty oystercatcher (*Haematopus fuliginosus*)
· Little raven (Corvus mellori)
· Hooded plover (*Thinornis rubricollis*)

In order to better quantify the risk that sympatric avian species may have on LPs at Phillip Island (Millowl), it was important to consider individual life history traits that may play a role in pathogen transfer. This is especially pertinent for species that have the possibility of contact with domestic fowl, or travel to areas when diseases of concern are endemic.

The migratory behaviour of the above-mentioned species is listed in Table 5 below (Scheelings 2022) (P. Dann pers comm):

Table 5. Migratory behaviour of species in ready contact with LPs at Phillip Island (Millowl) (from Scheelings 2022)

Species	Migration
Short-tailed shearwater	Trans-hemispheric migrant to northern Pacific particularly Sea of Japan and Bering Sea
Crested tern	Dispersal of young along east coast of Australia
Pacific gull	Resident – maybe some exchange with Corner Inlet and Wilsons Promontory
Kelp gull	Some local dispersal to Mornington Peninsula
Silver gull	Dispersal around Western Port when not breeding
Cape Barren goose	Resident – no migration
Purple swamphen	Resident – no migration
Sooty oystercatcher	Dispersal to Wilsons Prom during autumn/winter
Hooded plover	Dispersal of adults to other parts of Phillip Island and young east along Bass Coast

A full list of resident species on Phillip Island (Millowl) is also included (see Appendix 1).

3.10.1 Formulation of the hazard list

A hazard for the purposes of this DRA constituted known (to date) infectious and non-infectious diseases of wild and captive LPs in the Australasian region as well as wild Sphenisciformes and those held in captive collections globally. This broad scope was included as there has been limited health and disease surveillance undertaken in the Phillip Island (Millowl) population, and we were keen to identify health hazards to which Sphenisciformes would be susceptible, then prioritise associated risk based on likelihood of exposure in the region and consequences to the population. This approach would ensure we identified hazards which are globally present but perhaps yet to be screened for at Phillip Island (Millowl) (data deficient) to improve the knowledge gaps and level of uncertainty in hazard assessment.

Hazards were identified and justified according to the following questions, developed from Jakob-Hoff et al. 2014*a*):

1) '*What can cause disease, or impact the population?* Answered by identification of infection and / or disease in **host range** species and identification of **impact;**
2) '*How can this happen?*' Answered by identification of **transmission** pathways; and
3) '*What are the potential consequences?*' Answered by identification of **impact** and **consequences** (see Figure 3) (Jakob-Hoff et al. 2014*a*).

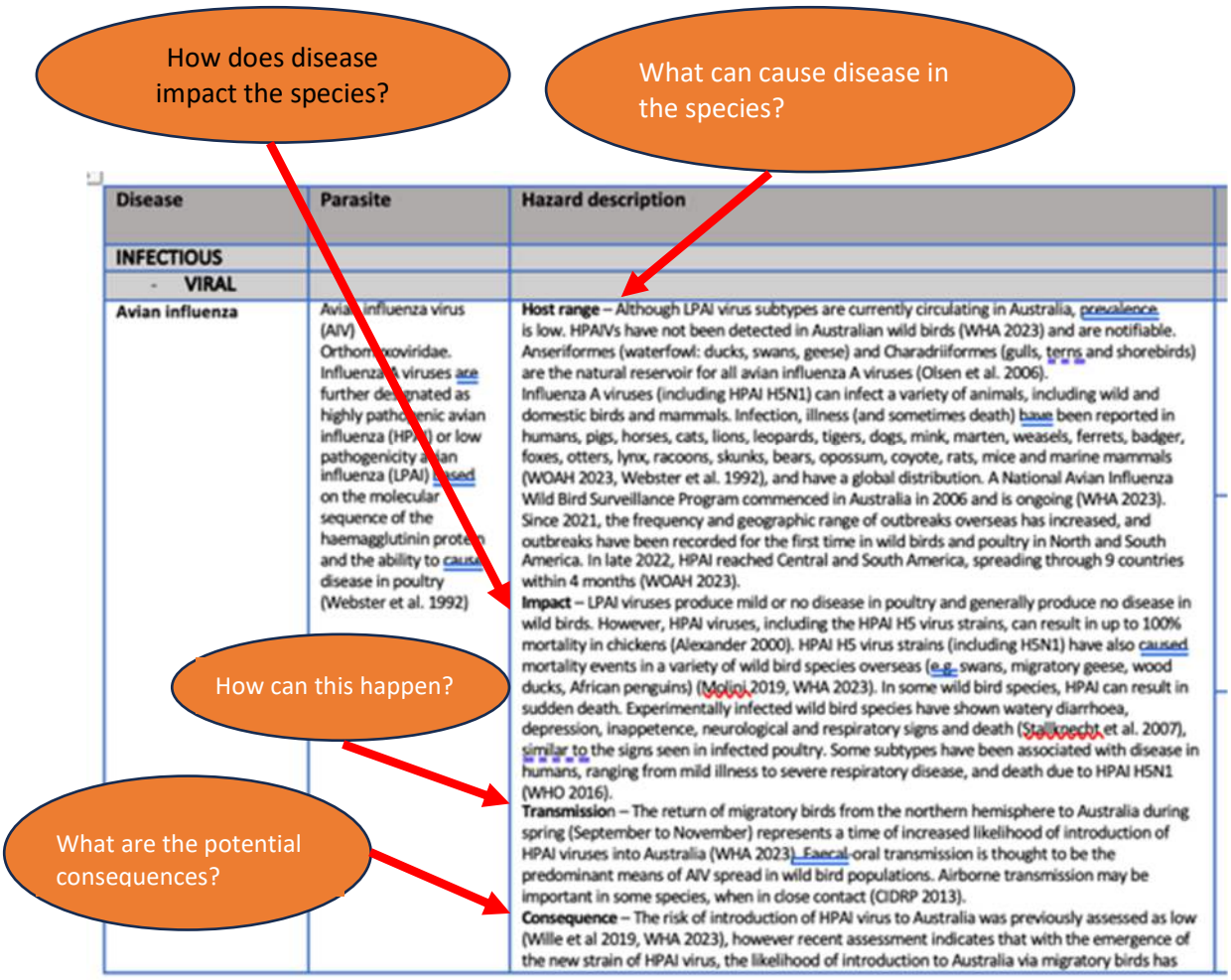


Figure 3. Method for identifying and justifying hazards

3.10.2 Methodology of hazard prioritisation

We developed a method of prioritising hazards for further assessment, as a long list of hazards had been identified in the first step of the process. We initially Identified all possible hazards of concern and categorised into 'infectious' and 'non- infectious'.

We estimated the **likelihood of contact** between hazards and the previously described population groups of interest at Phillip Island (Millowl) and the **consequences** to them if contact were to occur.
A two-step prioritization of hazards was undertaken:

Step 1: For each hazard, we considered whether a risk existed for each population group

Species Group 1	Little penguins at Phillip Island (Millowl)
Species Group 2	Wildlife resident at the site (including introduced or feral species) and migrating wildlife
Species Group 3	People and domestic animals (pets)
Species Group 4	Livestock

Step 2: Where a risk existed for a population group, we scored for **likelihood that an individual animal could harbour a disease hazard and assist in its transmission to another animal (Table 6).**

Table 6: Terminology and classification system for likelihood of exposure to hazards

Terminology and classification system for likelihood of hazard entry, establishment and spread	
Very low	Very unlikely to occur – hazard has not been recorded in the geographic range of the species at Phillip Island (Millowl)
Low	Hazard has been recorded in the geographic range of LPs at Phillip Island (Millowl), but it is unlikely this hazard would occur or become established
Moderate	Hazard occurs in other, similar ecosystems or geographically / climatically similar sites and is able to persist at geographically similar sites, but has not been recorded at Phillip Island (Millowl)
High	Hazard is known or expected to occur / persist in the Phillip Island (Millowl) population of LPs.

Step 3: We then scored for the **consequences of disease to a population group**, in a process similar to that used in the eastern barred bandicoot DRA (Jakob-Hoff et al. 2016) and the Shark Bay bandicoot DRA (Vaughan-Higgins et al. 2019) (Table 7).

Table 7. Terminology and classification system for consequences for LPs and extant wildlife

Terminology and classification system for consequences for LPs and extant wildlife	
Negligible	No detectable effects on the individual, no detectable population consequences, no socio-economic consequences.
Low	Individual morbidity or mortality, but no detectable population consequences, minimal socio-economic consequences in the short or long term. Low impact on welfare
Moderate	Temporary detectable population decline without risk of extinction from this disease, low socio-economic consequences. No long-term population impact on the expected population growth / population levels of birds
High	High risk of local extirpation, due to significant population decline at unsustainable levels, moderate to major socio-economic consequences. Changed status listing of bird species, impacts multiple bird species, flow on impacts to ecosystem processes. Has a high impact on welfare – many animals affected and prolonged suffering.
Proposed classification for consequences for humans and domestic animals	
Low	No effects
Moderate	Self-limiting morbidity, not requiring treatment
High	Individual morbidity, requiring treatment or not self-limiting.
Proposed classification for consequences to livestock	
Low	Minimal effects
Moderate	Short-term treatment, minimal welfare impacts and economic significance
High	Individual morbidity, significant treatment, welfare & economic impacts

We then considered the combined likelihood and potential direct and indirect consequences of hazards to decide which should be subjected to full risk assessment based on the following risk matrix (Table 8).

Table 8. Risk matrix demonstrating the combined likelihood of entry, establishment and spread and consequence of entry, establishment and spread in a defined population.

Likelihood of entry establishment and spread in the specified population	Consequence of entry, establishment and spread in the population				
		Negligible	**Low**	**Moderate**	**High**
	Very Low	Very Low	Very Low	Low	Low
	Low	Very low	Low	Low-Moderate	Moderate
	Moderate	Low	Low-Moderate	Moderate	High
	High	Low	Moderate	High	High

Uncertainty

We also classified levels of uncertainty in the hazard prioritisation steps as per Knox et al. (2020) adapted from Wieland et al. (2011) (Table 9).

Table 9. Proposed classification of level of uncertainty adapted from Wieland, Dhollander et al. (2011)

Proposed classification of level of uncertainty adapted from Wieland, Dhollander et al. (2011)	
Low	Solid and complete data available; strong evidence provided in multiple references
Medium	Some but no complete data available with some information gaps; evidence provided in small number of references.
High	Scarce or no data available with significant information gaps; evidence is not provided in references but rather in unpublished reports, based on observations, or personal communication.

If there was uncertainty, we reached our final hazard outcome by adopting the precautionary principle (IUCN 2007). In many cases, current knowledge of the epidemiology and prevalence of LP disease is incomplete or lacking, creating uncertainty regarding the likelihood or consequences of risk of a disease hazard. Where such limitations on available evidence were encountered, the "precautionary principle" was applied, whereby a risk was assumed to exist (and require management), until proven otherwise. It also meant that a higher rather than lower risk rating was applied as a 'precaution'.

For example:

L (likelihood) and L (consequence) = Low priority for risk assessment
M (likelihood) and M (consequence) = Medium
H (likelihood) and H (consequence) = High
L (likelihood) and M (consequence) = Medium
M (likelihood) and L (consequence) = Medium

Eighty hazards were identified and were summarised in tabular form and colour coded according to risk status: green – low risk hazard, orange – medium risk hazard, red – high risk hazard (Table 10). The full hazard identification and prioritisation list is provided in Table 12.

Where an additional hazard was proposed in the last round of feedback (i.e. antimicrobial resistance), a quick risk assessment was performed based on available literature without further consultation with experts.

4 Results

Eighty hazards were identified in total. This comprised five high risk hazards (HPAI, oiling, predation (marine and terrestrial) and wildfire). Nineteen medium risk hazards (Saumarez reef virus, APMV1, avian chlamydia, avian cholera, Babesiosis, Plasmodium infection, Toxoplasmosis, *Contracaecum* infection, *Mawsonotrema eudyptulae* infection, ticks, algal toxins, persistent organic pollutants and other emerging contaminants, per- and polyfluorinated substances, environmental stressors including climate change, habitat loss (marine and terrestrial), reduction in food supplies, starvation, thermal stress and tourism) were identified.

A summary of the hazard prioritisation is provided in Table 10.

Table 10. Summarised hazard identification and prioritisation for the little penguin DRA

Disease	Parasite	Hazard population	Likelihood rating	Consequence rating	Uncertainty rating	Final rating
VIRAL						
Arboviruses	Barmah Forest Virus	1	L	L	M	L
	Kunjin	1	L	L	L	L
		3	L	M	L	M
	Murray Valley Encephalitis Virus	1	L	L	L	L
		3	L	M	L	M
	Ross River Virus	1	L	L	L	L
		3	L	M	L	M
	Saumarez reef virus	1	L	M	L	M
	West Nile Virus*	1	VL	M	L	L
		2	VL	M	L	L
		3	VL	M	L	M
Avian encephalomyelitis virus 'Pingu virus"	Picornaviridae	1	L	L	M	L
Avian infectious bronchitis virus	Coronaviridae	1	L	L	M	L
High pathogenicity avian influenza	High pathogenicity avian influenza virus *	1	L	H	L	H
		2	L	H	L	H
		3	L	H	L	H
		4	L	H	L	H
Avian poxvirus	Avipoxvirus	1	L	L	L	L
Avian reovirus	Reoviridae	1	L	L	M	L
Gyrovirus -	Yellow eyed penguin Gyrovirus, Respiratory Distress syndrome	1	VL	M	M	L
Infectious bursal disease virus	Birnavirus	1	L	L	M	L
Infectious laryngotracheitis virus Herpes disease	Herpesviridae Avian herpesvirus 1	1	L	L	M	L
Low pathogenicity avian influenza	Lowly pathogenic avian influenza virus	1	L	L	L	L
		2	L	L	L	L
		3	L	L	L	L
Avian paramyxoviruses	Avian paramyxovirus (APMV)1 – virulent*	1	VL	H	L	M
		2	VL	H	L	M
		4	VL	H	L	M
	Avian paramyxoviruses - avirulent	1	L	L	L	L
		2	L	L	L	L
Penguin diphtheria	*Yellow eyed penguin megrivirus, picornavirus*	1	VL	M	M	L
Puffinosis	Suspected viral but unknown	1	L	M	H	L
BACTERIAL						
Avian chlamydia	*Chlamydia psittaci, Chlamydia abortus*	1	L	M	M	L
		2	L	L	M	L
		3	L	M	L	M
Avian cholera	*Pasteurella multocida*	1	L	H	L	M
		2	L	H	L	M
Avian mycobacteriosis		1	M	L	L	L

		2	M	L	L	L
		3	M	L	L	L
E.coli infection	*E.coli*	1	M	L	L	L
		2	M	L	L	L
		3	M	L	L	L
Erysipelas	*Erysipelothrix rhusiopathiae*	1	VL	M	L	L
		3	VL	M	L	L
Mycoplasmosis	*Mycoplasma* spp.	1	L	L	M	L
Necrotic enteritis	*Clostridium* spp. *E.coli*	1	L	M	L	L
Pseudomonas infection	*Pseudomonas* spp.	1	VL	M	L	L
Salmonellosis	*Salmonella* spp.	1	M	L	L	L
		2	M	L	L	L
		3	M	L	L	L
FUNGAL						
Aspergillosis	*Aspergillus* sp	1	L	M	L	L
Candidiasis	*Candida albicans*	1	L	L	L	L
ENDOPARASITES						
-CESTODES						
Tetrabothrius infection	*Tetrabothrius sp.* and *Tetrabothrius lutzi*	1	M	L	M	L
-HAEMOPARASITES						
Haemosporidian	*Haemoproteus* sp.	1	L	L	M	L
	Leukocytozoon sp.	1	L	L	M	L
	Plasmodium sp.	1	L	M	M	M
Piroplasmid	*Babesia* sp.	1	M	L	M	M
Kinetoplastid	*Trypanosoma eudyptulae*	1	L	L	L	L
-PROTOZOANS						
Coccidiosis	*Eimeria* sp.	1	M	L	M	L
Cryptosporidium	*Cryptosporidium* sp.	1	L	M	M	L
		3	L	M	L	L
Giardia	*Giardia* sp.	1	L	L	L	L
		3	L	L	L	L
Trichomoniasis	*Trichomonas gallinae*	1	M	L	L	L
Sarcocystis	*Sarcocystis spp.*	1	L	L	L	L
Toxoplasmosis	*Toxoplasma gondii*	1	L	M	M	M
-NEMATODES						
Capillariasis	*Capillaria* spp.	1	L	L	L	L
Contracaecum - helminths	*Contracaecum eudyptulae*	1	M	M	L	M
		3	VL	L	L	L
-TREMATODES						
Trematodiasis	*Cardiocephaloides physalis*	1	L	L	L	L
Trematodiasis	*Galactosomum angelae*	1	L	L	M	L
Trematodiasis	*Mawsonotrema eudyptulae*	1	M	M	M	M
Renal fluke	*Renicola* sp.	1	L	L	M	L
ECTOPARASITES						
Fleas	*Parapsyllus* sp	1	L	L	L	L
		2	L	L	L	L
Lice	*Austrogonoides waterstoni*	1	L	L	L	L
Mites	*Ingrassia eudyptula, & Veigaia* sp.) and others	1	L	L	M	L
Ticks	*Ixodes eudyptidis, Ixodes uriae*	1	L	L	M	M
		2	L	L	M	L
CONGENITAL/DEVELOPMENTAL						
Beak malformation, carpal rotation, ventricular septal defects, splay leg		1	**L**	**L**	**L**	**L**
DEGENERATIVE						
Cataracts		1	L	L	M	L
Degenerative joint disease		1	L	L	M	L
INTOXICATION						
Algal toxins		1	L	M	L	M
		2	L	M	L	M
		3	L	M	L	M
		4	L	M	L	M
Heavy metal intoxication		1	L	L	L	L
Industrial chemical intoxication - oiling		1	L	H	L	H
		2	L	H	L	H

Persistent organic pollutants (POPs) and other emerging contaminants		1	L	L	M	M
		2	L	L	M	M
Per- and Polyfluorinated Substances (PFAS)		1	L	L	M	M
		2	L	L	M	M
ENVIRONMENTAL						
Antimicrobial resistance genes		3	VL	H	M	L
		4	VL	H	M	L
Botulism		1	VL	L	L	L
Environmental stressors including climate change		1	L	M	M	M
Habitat loss	Marine– ocean acidification	1	M	M	L	M
	Terrestrial - trampling by humans / livestock, introduced species, Cape Baron geese, coastal erosion, dune cliffing, invasive weeds, housing, sea level rise	1	M	M	L	M
Predation	Marine	1	M	H	L	H
	Terrestrial	1	M	M	M	H
Reduction in food supplies		1	L	M	L	M
Starvation		1	L	M	L	M
Storm damage		1	L	L	L	L
Thermal stress	Marine - SST & marine heatwaves, warmer oceans	1	L	L	L	L
	Terrestrial – decreased rainfall & humidity	1	H	L	L	M
Wildfire		1	H	H	L	H
OTHER						
Cardiac (heart) disease		1	L	L	L	L
Entanglement		1	L	L	L	L
Gastrointestinal foreign bodies		1	L	L	L	L
Hepatic (liver) disease		1	L	L	L	L
Neoplasia		1	L	L	L	L
Pododermatitis (bumblefoot)		1	L	L	L	L
Tourism		1	H	L	L	M
Trauma		1	M	M	M	L

*confirmed as exotic to Australia, World Organization for Animal Health (WOAH) Notifiable disease

Oil, wildfire, and predation (marine and terrestrial) were the most significant non-infectious hazards identified, however, PINP already has excellent contingency plans in place for these hazards. Habitat loss (marine and terrestrial), reduction in food supplies, thermal stress, starvation, and tourism also have robust management plans in place through PINP, so further risk assessment was not undertaken.

Six moderate-high risk human health hazards were identified: HPAI, West Nile virus (neither currently present in Australia), Kunjin virus, Murray Valley encephalitis, Ross River virus, and avian chlamydia. Full risk assessments were not conducted for Kunjin virus, Murray Valley encephalitis, or Ross River virus, as the risk management for humans was detailed in the similar arbovirus Saumarez reef virus disease risk assessment. However, please note, while broad recommendations can be summarised, specific information on treatment of zoonotic disease should be referred to a human health specialist.

Full risk assessments were completed for 15 identified medium and high risk hazards, comprising HPAI, Saumarez reef virus, APMV1, avian chlamydia, avian cholera, Babesiosis, infection with *Plasmodium* sp., toxoplasmosis, infection with *Contracaecum* sp., infection with *Mawsonotrema eudyptulae* , ticks, algal toxins, persistent organic pollutants and other emerging contaminants, per- and polyfluorinated substances and environmental stressors, including climate change.

A summary of the number of hazards within each category of infectious and non-infectious hazards is provided below (Table 11).

Table 11. Summary of infectious and non-infectious hazards and categories in the LP DRA.

Viral	19	Congenital/developmental	1
Bacterial	9	Degenerative	2
Fungal	2	Environmental	12
Endoparasites	18	Intoxication	5
Ectoparasites	4	Other	8

The detailed hazard descriptions and assessments can be seen in section 4.1.

4.1 Detailed hazard descriptions

Table 12. Hazard identification, justification, and prioritisation for risk assessment noting (L) likelihood, (C) consequence, (U) uncertainty (O) overall rating for the LP Disease Risk Analysis. The descriptions text has been adapted and/or quoted from the original sources cited.

Disease	Parasite	Hazard description	Species Group*	L	C	U	O
INFECTIOUS							
VIRAL							
Arboviruses Arboviruses are distributed globally and belong mainly to the genera alphavirus (family *Togaviridae*), flavivirus (family *Flaviviridae*), nairovirus (family *Bunyaviridae*), phlebovirus (family *Bunyaviridae*), orbivirus (family *Reoviridae*), coltivirus (family *Reoviridae*) and vesiculovirus (family *Rhabdoviridae*).	Arboviruses - Those circulating in Australia include Murray Valley Encephalitis (MVEV), Kunjin Virus (WNV lineage 1), Barmah Forest virus (BFV) and Ross River Virus (RRV). West Nile Virus (USA strains NY99 and WN02) belonging to lineage 1 are exotic to Australia, however a closely related virus, Kunjin encephalitis virus (KUN) is present, though has not produced the same disease impact as WNV. (WHA 2016).	**Host range** – Group of viruses transmitted through a range of insect vectors. Presence of insect vectors determines the distribution of the diseases. Wild birds considered primary hosts, carriers and reservoir for some of these viruses, though no overt disease from flaviviruses KUN, MVEV in Australian native birds reported (Ladds 2018). At least 32% of penguins sampled at Phillip Island (Millowl) (Morgan et al. 1985) had group specific antibodies for flaviviruses. These viruses multiply in arthropods with vertebrates becoming infected through a bite from an infected vector. Incidence of disease higher in northern Australia, in some coastal regions and along some river systems (Harley *et al.* 2001). MVEV, KUN, RRV and BFV all notifiable diseases in humans. Macquarie Island penguins (rockhopper, royal and king penguins) harbour arboviruses from at least four of the seven arbovirus-containing genera, with related viruses often found in the northern hemisphere). A flavivirus designated Saumarez reef virus, and an unnamed virus isolated from ticks on Macquarie Island, Southern Ocean were reportedly pathogenic causing disease and mortality in Phillip Island (Millowl) LPs inoculated with the viruses (Morgan et al. 1985). There are no records of arbovirus infection in little penguins in the eWHIS record inspected.					
	Flavivirus Ross River virus	<u>RRV:</u> **Host range -** Marsupials are better amplifiers of RRV than placental mammals, which are in turn better amplifier hosts than birds (Harley et al. 2001, WHA 2015). Australian birds including the little corella (*Cacatua sanguinea*), magpie larks (*Grallina cyanoleuca*), Australian brown flycatcher (*Microeca fascinans*), and masked finch (*Poephila personata*) have been reported as asymptomatic hosts (Ong et al. 2021). Macropods are suspected of playing a significant role as reservoir hosts of the virus. Brushtail possums (*Trichosurus* spp.) and horses are likely involved in transmission in the urban environment (WHA 2015). RRV is the most common mosquito-borne infection in Australia with approximately 5,000 human cases reported annually (Russell, 2002).	1	L	L	M	L

		Impact - is capable of causing severe polyarthritis, fever, myalgia and rash in human cases of clinical disease. Most cases, however, are sub-clinical in both humans and animals (WHA 2015) **Transmission -** Mosquito-vertebrate transmission cycle, at least 40 spp. of mosquito capable of harbouring infection (Russell, 2002). **Consequence** – Very low likelihood of exposure in free-living LP population, unless vector present, however, this is unlikely owing to the southern location of Phillip Island (Millowl). Although BFV endemic in Victoria. Native birds and wildlife are not usually clinically affected, therefore likelihood is very low and consequence low, however, can cause disease in humans. The consequence of RRV to humans and other susceptible non-reservoir species is likely moderate, however, the likelihood of LPs contributing to this risk based on current evidence is low. Can be managed by mosquito control and existing protocols e.g. long clothing, insect repellent to reduce to an overall low risk.					
			3	L	M	M	M
	Flavivirus - Kunjin virus	<u>Kunjin virus:</u> **Host range** - crows, magpies (Family Corvidae), house sparrows (*Passer domesticus*), house finches and other passerines develop the highest concentrations of virus in the blood and have the longest duration of viraemia (Phalen and Dahlhausen 2004). Wading birds (in particular the Nankeen Night heron, (*Nycticorax caledonicus*) are considered key natural reservoirs for Kunjin virus (Prow 2013). Other hosts: Humans and other primates; equids such as horses and donkeys; artiodactyls such as cattle, pigs, llama and deer; carnivores such as dogs, cats, seals and cetaceans; bats; rabbits; rodents; elephants; rhinoceroses, crocodiles and alligators (WHA 2016a). Kunjin is endemic to northern Australia but encroaches southward when heavy rains facilitate an increase in water bird (amplifying host) and mosquito (vector) density. **Impact** - in 2011 an outbreak of neurological disease occurred in horses in south-eastern Australia with more than 1000 equine cases and 10-15% mortality (Frost et al. 2012). On average, Kunjin virus infected 1.4 humans annually over the period 2010-2014 within Australia (WHA 2016a). Human infection with Kunjin virus typically produces a mild disease consisting of lymphadenopathy, fever, lethargy, rash and muscle weakness (WHA 2016). In horses, clinical signs include ataxia, incoordination, wide stance in forelimbs, weakness, altered temperament, mild fever (inconsistent), blindness, muscle trembling, seizures, facial paralysis, dullness, recumbency and death (Tee *et al.* 2012). Birds act as a reservoir and amplifying host, but rarely show signs of disease (WHA 2016a). **Transmission** – Bird-mosquito-bird transmission cycles – mainly *Culex* spp. Non-vector transmission via ingestion of infected mosquitoes, infected prey animals and contaminated water has also been demonstrated (Phalen and Dahlhausen 2004). **Consequence** – Very low likelihood of exposure in free-living LP population, unless vector present, however, this is unlikely owing to the southern location of Phillip Island (Millowl). Native birds and wildlife are not usually clinically affected, therefore likelihood is very low and consequence low, however, can cause disease in humans. The consequence of Kunjin to humans and other susceptible non-reservoir species is likely moderate, however, the likelihood of LPs contributing to this risk based on current evidence is low. Can be managed by mosquito control and existing protocols eg long clothing, insect repellent to reduce to an overall low risk.	1	L	L	**M**	**L**
			3	L	M	**M**	**M**

	Flavivirus Murray Valley encephalitis virus	MVEV: **Host range -** cormorants, night herons, brolgas, (primary reservoir hosts) eastern grey kangaroos, rabbits (amplifying hosts) agile wallabies, cattle, horses and feral pigs shown to have serological evidence of infection (Marshall et al. 1982a). MVEV causes serious disease in humans and outbreaks in humans have occurred in southern Australia (Spencer et al. 2001). **Impact** – Wildlife not known to be clinically affected by infection (WHA 2016b). Clinical signs in humans include fever, headache, nausea and vomiting, progressing to neurological signs associated with meningitis and encephalitis (Knox et al. 2012). Affected horses display neurological symptoms including depression, weakness (particularly in hind limbs), ataxia, wide-based stance, drooping head, sweating, facial paralysis, proprioceptive deficits and recumbency (Gordon et al. 2012; Holmes et al. 2012). **Transmission** - Mosquito-bird transmission cycle: mosquitoes *Culex annulirostris* are the primary vectors and birds are the amplifying hosts. Wading birds, in particular the rufous (Nankeen) night heron (*Nycticorax caledonicus*) are considered the major amplifying hosts. **Consequence** – Very low likelihood of exposure in free-living LP population, unless vector present, however, this is unlikely owing to the southern location of Phillip Island (Millowl). Native birds and wildlife are not usually clinically affected, therefore likelihood is very low and consequence low, however can cause disease in humans. The consequence of MVE to humans and other susceptible non-reservoir species is likely moderate, however, the likelihood of LPs contributing to this risk based on current evidence is low. Can be managed by mosquito control and existing protocols e.g. long clothing, insect repellent to reduce to an overall low risk.	1	L	M	**L**	**L**
			3	L	M	M	**M**
	Flavivirus Barmah Forest virus	BFV: **Host range** – It is not established, but it is likely, that macropods and other marsupials are the principal hosts for the virus. BFV antibodies have been found in brushtail possums, kangaroos, cattle, horses and sheep (Kay et al. 2007, Ong et al. 2021, Victoria Health 2021). **Impact –** is capable of causing severe polyarthritis, fever, myalgia and rash in human cases of clinical disease. Most cases, however, like RRV are sub-clinical in both humans and animals (WHA 2015). Outbreaks of BFV disease sometimes occur concurrently with RRV disease, making diagnosis difficult (Victoria Health 2021). **Transmission -** Mosquito-vertebrate transmission cycle**.** Like Ross River virus disease, BFV disease appears after heavy rains that facilitate the breeding of mosquito vectors. There is no evidence of transmission from person to person. Considered endemic in Victoria. **Consequence** – Very low likelihood of exposure in free-living LP population, unless vector present, however, this is unlikely owing to the southern location of Phillip Island (Millowl). Although BFV endemic in Victoria. Native birds and wildlife are not usually clinically affected, therefore likelihood is very low and consequence low, however can cause disease in humans. The consequence of BFV to humans and other susceptible non-reservoir species is likely moderate, however, the likelihood of LPs contributing to this risk based on current evidence is low. Can be managed by mosquito control and existing protocols e.g. long clothing, insect repellent to reduce to an overall low risk.	1	L	L	M	**L**
			3	L	M	**M**	**M**

	Flavivirus Saumarez reef virus	<u>The Saumarez reef virus (SRV):</u> **Host range –** Experimental transmission study reported in Phillip Island (Millowl) LPs (Morgan et al. 1985). A flavivirus designated Saumarez reef virus, and an unnamed virus isolated from ticks on Macquarie Island, Southern Ocean were reportedly pathogenic causing disease and mortality in Phillip Island (Millowl) LPs inoculated with the viruses (Morgan et al. 1985). **Impact** -The Saumarez reef virus caused death 9-13 days post inoculation in LPs. Lesions included gastrointestinal haemorrhage, hepatic periacinar necrosis and severe necrotising enteritis (Ladds 2009). **Transmission** – direct inoculation (for research study) naturally via presumed tick bite. Ticks were considered the most likely vector of infection as flavivirus strains have been isolated from Ixodid ticks (*Ixodes uriae)* **Consequence** – Has caused death in Phillip Island (Millowl) LP population when directly inoculated. Low likelihood of exposure and transmission to other penguins as requires presence of vector. Potential disease risk for LPs being exposed in the wild if vector species present and of moderate consequences to population.	1	L	M	**L**	**M**
West Nile Virus **WOAH listed pathogen**	Flavivirus West Nile Virus USA strains NY99 and WN02) belonging to lineage 1.	<u>West Nile virus</u> **Host range –** Primary host birds, other hosts include humans and other primates; equids such as horses and donkeys; artiodactyls such as cattle, pigs, llama and deer; carnivores such as dogs, cats, seals and cetaceans; bats; rabbits; rodents; elephants; rhinoceroses, crocodiles and alligators. Infection and deaths from WNV have been reported in Humboldt and African penguins, common loons, pie-billed grebes, Clark's grebes, eared grebes, American white and brown pelicans, double-crested, and Guanay cormorants (Stidworthy and Denk 2018). The USA strains of the virus are not present in Australia. **Impact** – Since 1999, WNV has become a significant emerging zoonotic disease, most notably in the USA, causing severe neurological disease and death in many animal species, especially humans, horses and birds mostly among members of the Corvidae (WHA 2016a). The mechanisms for the emergence of WNV likely involve increased global travel, climatic and ecological factors, and novel viral genotypes (Travis 2008). WNV has been associated with morbidity and mortality in Humboldt and African penguins both in captivity and free ranging (Davis et al. 2007). Many zoological facilities subsequently initiated WNV vaccination protocols to at risk or endangered avian species. Clinical signs in penguins can range from anorexia, weakness, abnormal behaviour, vomiting, and dyspnoea from excessive pulmonary and tracheal secretions, recumbency, and sudden death (Stidworthy and Denk 2018). Experimentally infected American crows died after four to eight days. However, experimentally infected Australian little ravens displayed only mild signs of lethargy and reduced food consumption but recovered fully by ten days post infection (Bingham and Lunt 2010). Infected people may have no symptoms, or mild flu-like symptoms. In rare cases, infection can lead to serious complications such as meningitis and encephalitis, paralysis and poliomyelitis. **Transmission** – Mosquitoes of the genus *Culex* are the primary vector of WNV and avian hosts are the main reservoir (WHA 2016a). **Consequence** – Very low likelihood of exposure in free-living LP population, as USA lineage not reported in Australia. With predictions in climate change possible that vector distribution may	1	VL	H	**L**	**L**
			2	VL	H	**L**	**L**
			3	VL	H	**L**	**L**

		change. Can rarely cause significant disease in humans. There are commercially available equine vaccines being used to vaccinate birds. Overall, of low consequence to populations as not reported in Australia.					

Disease	Parasite	Hazard description	Species Group*	L	C	U	O
Avian encephalomyelitis virus (AEV) 'Pingu' virus	Picornoviridae	**Host range –** Seropositivity to AEV was identified in wild Namibian and Western Cape African Penguin samples and in penguins admitted for rehabilitation at the Southern African Foundation For the Conservation Of Coastal Birds (SANCCOB); at 2.9% prevalence (Parsons et al. 2016). Karesh et al. (1999) reported AEV antibodies in southern rockhopper penguins in Argentina, with 3% seroprevalence. A novel picornavirus named 'Pingu' virus was isolated from Gentoo penguins at King George Sound Antarctica (deSouza et al. 2019). Four novel viruses belonging to the family *Picornaviridae* were also recently reported in healthy Magellanic penguins off the coast of Chile (Hayer et al. 2020). **Impact –** Picornavirus infections in birds are known to cause, or be associated with, a range of diseases, including avian encephalomyelitis in chickens, hepatitis in ducks and turkeys, malabsorption in chickens and turkeys, proventriculitis in chickens, and keratin disorder in chickadees (deSouza et al. 2019). Serological evidence of infection reported, with no associated clinical signs or evidence of disease (Parsons et al. 2016) on necropsy of penguins. In chickens seldom causes clinical disease in adults but can lead to significant decreases in egg production and hatchability; however, in young chickens, AEV can produce paralysis, ataxia and muscular dystrophy (Parsons et al. 2016). 'Pingu' virus was isolated from cloacal swabs of healthy penguins apparently without symptoms or disease, suggesting the virus might be endemic and non-pathogenic in penguins. Hayer et al. (2020) reports evidence to suggest that picornaviruses are not exclusively disease causing, and that picornavirus species detected in wild birds are often not associated with any signs of disease. **Transmission –** Faecal-oral route **Consequence –** Not reported in Australian LPs, therefore low likelihood of occurrence and transmission. Likely to be susceptible however no evidence of testing undertaken in Australasian region, therefore medium level of uncertainty. Overall, of low consequences to penguin populations as not typically associated with clinical signs or disease.	1	L	L	**M**	**L**
Avian infectious bronchitis virus (IBV)	Coronaviridae	**Host range –** IBV is ubiquitous in most parts of the world in regions with intensive poultry production (Milek and Blicharz-Domańska 2018). Coronaviruses have been detected in 108 wild bird species. Specifically, members of the Anseriiformes (ducks, geese, swans) are important hosts for gammacoronaviruses, Charadriiformes (gulls and shorebirds) are hosts for both gamma- and deltacoroanviruses, and Pelecaniiformes (specifically herons and egrets) are important hosts for deltacoronaviruses. Coronaviruses have been found in wild birds on every continent, including	1	L	L	**M**	**L**

		deltacoronaviruses in Chinstrap, Gentoo and Adelie penguins in Antarctica (Wille and Holmes 2020). Seropositivity to IBV was identified in Namibian and Western Cape samples of African Penguins and in penguins admitted for rehabilitation at SANCCOB; overall seroprevalence was relatively low (3.6%) (Parsons et al. 2016). Karesh et al. (1999) reported a seroprevalence between 23% and 47% in southern rockhopper penguins in Argentina. **Impact –** Coronaviruses (CoVs) mainly cause enteric and/or respiratory signs, however, apart from infectious bronchitis virus in chickens, the only avian species in which CoV has been definitively associated with disease are the turkey, pheasant and guinea fowl (DeWit and Cook 2020). IBV causes huge economic losses in domestic fowl (*Gallus gallus*). However, there are many reports of IBV presence in other bird species, which indicate that the virus can cross the species barrier. IBVs have been detected in healthy wild birds, demonstrating that they may act as the vector between domestic and free-living birds (Milek and Blicharz-Domańska 2018). DNA from coronaviruses has been detected in the tissues of washed up carcasses of Magellanic penguins in Brazil; however, it is not certain if the presence of the virus was associated with disease (Niemeyer et al. 2012). Few studies have tested penguins for antibodies against IBV. **Transmission –** primarily spread via respiratory droplets that contain the virus, with infections occurring via the nose, eyes and mouth. **Consequence –** Coronavirus has been identified in penguins, and antibodies against avian coronavirus have been seen in several wild penguin species but it is yet to be detected in Australian LP population. LPs likely to be susceptible and may have been exposed but suspect of low consequence to the population unless other significant population pressures / disease causing agents. Few studies have tested penguins for antibodies against IBV.					
Avian influenza **HPAI - WOAH listed pathogen**	Avian influenza virus (AIV) Orthomyxoviridae. Influenza A viruses are further designated as high pathogenicity avian influenza (HPAI) or low pathogenicity avian influenza (LPAI) based on the molecular	**Host range** –HPAIs have not been detected in Australian wild birds (WHA 2023a) and are notifiable. The risk of introduction of HPAI virus to Australia was previously assessed as low (Wille et al. 2019b, WHA 2023a), however, recent assessment indicates that with the emergence of the new strain of HPAI virus, the likelihood of introduction to Australia via migratory birds has increased. Anseriformes (waterfowl: ducks, swans, geese) and Charadriiformes (gulls, terns and shorebirds) are the natural reservoir for all avian influenza A viruses (Olsen et al. 2006). Influenza A viruses (including HPAI H5N1) can infect a variety of animals, including wild and domestic birds and mammals. Infection, illness (and sometimes death) have been reported in humans, pigs, horses, cats, lions, leopards, tigers, dogs, mink, marten, weasels, ferrets, badger, foxes, otters, lynx, racoons, skunks, bears, opossum, coyote, rats, mice and marine mammals	1	L	H	L	H

	sequence of the haemagglutinin protein and the ability to cause disease in poultry (Webster et al. 1992)	(WOAH 2023a, Webster et al. 1992), and have a global distribution. A National Avian Influenza Wild Bird Surveillance Program commenced in Australia in 2006 and is ongoing (WHA 2023a). Since 2021, the frequency and geographic range of HPAI (H5N1)outbreaks overseas has increased. In late 2022, H5N1 reached Central and South America, spreading through 9 countries within 4 months (WOAH 2023b). H5N1 has now been detected in the brown skua population in South Georgia, the first known cases in the Antarctic region Antarctica (Oct 23 2023, WOAH 2023b) **Impact** –HPAI viruses, including the HPAI H5 virus strains, can result in up to 100% mortality in chickens (Alexander 2000a). HPAI H5 virus strains (including H5N1) have also caused mortality events in a variety of wild bird species overseas (e.g. swans, migratory geese, wood ducks, African penguins and a wide variety of other seabirds) (Molini 2019, WHA 2023a). In some wild bird species, HPAI can result in sudden death. Experimentally infected wild bird species have shown watery diarrhoea, depression, inappetence, neurological and respiratory signs and death (Stallknecht et al. 2007), similar to the signs seen in infected poultry. Some subtypes have been associated with disease in humans, ranging from mild illness to severe respiratory disease, and death due to HPAI H5N1 (WHO 2016). **Transmissio**n – The return of migratory birds from the northern hemisphere to Australia during spring (September to November) represents a time of increased likelihood of introduction of HPAI viruses into Australia (WHA 2023a). In relation to LPs, the most likely route of infection was from migrating shearwaters returning from wintering in the northern hemisphere (Wille et al 2022; WHA et al. 2023a). Faecal-oral transmission is thought to be the predominant means of AIV spread in wild bird populations. Airborne transmission may be important in some species, when in close contact (CIDRP 2013). **Consequence** –The consequences are hard to predict but, based on overseas experience, could be devastating for wild birds, as well as poultry, and potentially also for some marine mammals. Currently low likelihood of exposure exists from contact between LPs and infected migratory shorebirds at Phillip Island (Millowl). Medium likelihood of carriage and transmission given species susceptibility and not in close proximity to poultry production facilities. HPAI not present in Australia, therefore very low current disease risk for humans contracting the zoonotic disease from LPs. Most illness and deaths associated with AIV infection in humans occurred after close contact with infected poultry or with objects contaminated by their faeces (WHA 2018) which will not occur if strict biosecurity is undertaken, and access to wild birds and penguins is minimised should disease incursion with HPAI occur in Victoria. Going through the eWHIS record, there were 79 records (some reflecting multiple penguins) where Influenza A was tested for; all were negative.	2	L	H	L	H
			3	L	M	L	H
			4	L	H	L	H

Disease	Parasite	Hazard description	Species Group*	L	C	U	O
Avian influenza **LPAI**	Avian influenza virus (AIV) Orthomyxoviridae. Influenza A viruses are further designated as high pathogenicity avian influenza (HPAI) or low pathogenicity avian influenza (LPAI) based on the molecular sequence of the haemagglutinin protein and the ability to cause disease in poultry (Webster et al. 1992)	**Host range** – Low pathogenicity avian influenza viruses are considered part of the natural viral community in wild birds in Australia. LPAI viruses have been identified in Australian Gruiformes, Pelecaniformes, Procellariiformes, Anseriformes and Charadriiformes. However, mortality due to AIVs has not been reported in wild birds (either native or feral) in Australia (WHA 2023a). A National Avian Influenza Wild Bird Surveillance Program commenced in Australia in 2006 and is ongoing (WHA 2023e). **Impact** – Low pathogenicity AI strains are unlikely to cause disease in wild birds or other wildlife species but are important due to the potential of H5 and H7 subtypes to mutate into HPAI forms when introduced to poultry (WHA 2023a). A wide range of domestic species are susceptible to AIVs including poultry, swine, horses, dogs and cats. Domestic animals should not be allowed access to sick birds or bird carcasses. Some humans have become infected with LPAI. Most illness and deaths associated with AIV in humans occur after close contact with infected poultry or with objects contaminated by their faeces. **Transmissio**n – The return of migratory birds from the northern hemisphere to Australia during spring (September to November) represents a time of increased likelihood of introduction of LPAI viruses into Australia (WHA 2023a). In LPs, Scheelings (2022) commented that the most likely route of infection was from migrating shearwaters returning from wintering in the northern hemisphere. Faecal-oral transmission is thought to be the predominant means of AIV spread in wild bird populations. Airborne transmission may be important in some species, when in close contact (CIDRP 2013). **Consequence** – Likely to have low consequence for wild birds, unless entering poultry and mutating to HPAI. Most illness and deaths associated with AIV infection in humans occurred after close contact with infected poultry or with objects contaminated by their faeces (WHA 2018), which will not occur if strict biosecurity is undertaken, and access to wild birds and penguins is minimised should disease incursion and mutation to HPAI occur in Victoria.	1	L	L	L	L
			2	L	L	L	L
			3	L	L	L	L
Avian pox	Avipoxvirus	**Host range** – infects most bird species globally (WHA 2012). Avipoxvirus infections have been documented in African, Humboldt, gentoo, rockhopper, and Magellanic penguins, but all species probably susceptible (Stidworthy and Dent 2018). Described in an Australian LP, but no major outbreaks reported (Ladds 2009). **Impact** –Most infections are cutaneous, affect fledgling birds on the nest, and result in proliferative and ulcerative lesions located on facial skin, beak, and legs (Stidworthy and Dent 2018). Three syndromes commonly reported: Skin form –most common presentation - localised swellings, to warty growths, typically found on areas of unfeathered skin, especially the feet, legs and around the face. Typically self-limiting, with most birds making a full recovery after the lesions become necrotic and shed, though mortality can arise if lesions become severe and affect vital areas (e.g. vision, ability to feed) or through secondary infections (Eden 2021)	1	L	L	L	L

		Diphtheritic form -lesions develop in the oral cavity and upper airways, and present as moist necrotic lesions that may progress to coalesce and form a lining of caseous material. This form is less common in wild birds. Systemic form – lesions can be found through internal organs, including areas of liver necrosis. This form is rarely reported in wild birds. Avian poxvirus is a key disease threat for some island-based bird species, which are less likely to have co-evolved with these viruses and may be naïve (van Ripper III and Forrester 2007). **Transmission** – Transmission is primarily through direct contact or vectors (biting insects such as mosquitos and flat-flies), though indirect contact with contaminated objects can also spread virus (WHA 2012). Transmission in wild birds is greatest when environmental factors such as temperature, moisture and humidity favour vector activity (van Ripper III and Forrester 2007) **Consequence** –The risk of avian pox virus contributing to significant population decline in Phillip Island (Millowl) little penguins was considered to be low, as infection with pox viruses in wild penguins are rare and have not resulted in mortality events, likely to have co-evolved with this virus and therefore possibly less susceptible to disease. There was one record of avipox in little penguins in the eWHIS record, a juvenile wild LP from St Kilda, Vic, with concurrent babesiosis.					

Disease	Parasite	Hazard description	Species Group*	L	C	U	O
Avian reovirus	Reovirus	**Host range –** Serological evidence reported from Sphenisciformes in Argentina (Karesh et al. 2009). Reported in wild birds from several continents including the Americas, Australia and Eurasia and are likely ubiquitous (Hollmen and Docherty 2007). Antibodies detected in wild African penguins sampled in Namibia and the Western Cape, with a low overall seroprevalence (0.9%) (Parsons et al. 2016). There is a limited number of studies conducted on reoviruses in free ranging birds. **Impact –** Reovirus-like agents with some similarity to the reference chicken reovirus strain were isolated in African penguins that died at a zoo in the United Kingdom. However, it was unclear what role the virus played in death (Gough et al. 2002). Clinical signs and pathology vary greatly, many are asymptomatic however, secondary or concurrent infections may play an important role in increasing host susceptibility. Has been associated with mortality events in Common Eiders and American Woodcock (Hollmen and Docherty 2007). In chickens has been associated with arthritis, tenosynovitis, enteritis, growth retardation, bursal and thymic atrophy and respiratory disease. Determining whether reovirus is an aetiological agent or an opportunistic or coincidental finding in a wildlife mortality event can be challenging and have been implicated as a potential disease agent associated with translocation of birds (Rigby et al. 1981). **Transmission –** Horizontal and vertical transmission with faecal-oral transmission the most likely route **Consequence –** Reovirus have been identified in penguins, and antibodies against avian reovirus have been seen in several wild penguin species but yet to be detected in Australian LP population. LPs likely to be susceptible and may have been exposed but suspect of low consequence to the population unless other significant population pressures / disease causing agents.	1	L	L	**M**	**L**
Infectious bursal disease (IBDV).	Birnavirus There are 2 known serotypes (1 & 2) Serotype 1 is pathogenic in chickens.	**Host range –** IBDV-2 antibodies isolated in captive African and Macaroni penguins (Gough et al. 2002). Antibodies against IBDV were detected in wild African penguins sampled in Namibia and the Western Cape and in penguins admitted for rehabilitation at SANCCOB; overall seroprevalence was relatively low (2.7%) (Parsons et al. 2016). Birnavirus antibodies have been detected in a variety of avian species including Antarctic penguins (Gardner et al. 1997). Not reported in Australian LPs. **Impact –** Birnavirus and reovirus isolated from 21 adult African penguins, and 5 Macaroni penguins on post-mortem exam at a UK Zoo in 1999 (Gough et al. 2002). Presence of virus thought to be immunosuppressive but not definitive cause of death. The etiologic agent of infectious bursal disease is the infectious bursal disease virus (IBDV), in the family Birnaviridae. The serotype 1 viruses cause disease in young chickens. Flock morbidity rate is typically 100%, and mortality rate can range from 5% to greater than 60% depending on the strain of virus and breed of chicken (Dey et al. 2019). No clinical signs of disease have been observed in any of the seropositive penguin species in the wild (Parsons et al. 2016). **Transmission –** Infectious bursal disease is highly contagious. IBDV is shed in the faeces and transferred by fomites.	1	L	L	**M**	**L**

		Consequence - IBDVs have been identified in avian species, including penguins, and antibodies against IBDV have been seen in several wild avian species. The contribution of IBDV to disease in these wild birds is uncertain. No record of infection in wild LPs in Australia likely to be of low / unknown consequence to the population.					
Yellow eyed penguin Gyrovirus Respiratory distress syndrome (RDS), Chicken anaemia virus	Gyrovirus	**Host range –** Gyroviruses cause disease in avian hosts, domestic cats, mice, ferrets and humans.. In yellow eyed penguin chicks associated with acutely fatal respiratory distress syndrome (RDS), (Wierenga et al. 2023). Not reported in other penguin species. **Impact –** The best-known virus in this genus is Chicken anaemia virus, which causes anaemia, poor growth and severe immunosuppression in young chicks. Chicken anaemia virus is found worldwide and causes significant mortality among chicks not protected by maternal antibodies. In yellow eyed penguins gyrovirus associated with respiratory distress syndrome with a mortality rate of over 90%, and chicks typically succumbed to the disease within the first week of life. Affected birds demonstrated a progressive increase in respiratory rate and effort. As the disease progressed birds become weak and often recumbent, with pale mucous membranes and evidence of hypothermia despite the provision of external heat. Terminally, chicks presented with coelomic distension, presumably due to overinflation of airsacs as a result of agonal gasping, and birds were visibly cyanotic with a reduced level of consciousness. In addition to the 28 birds that died in hospital, one died enroute to hospital, one was euthanised due to a limb deformity and a further 13 neonates died at the nest during this same period (Wierenga et al. 2023). **Transmission -** can be transmitted both horizontally and vertically and, as a small circular DNA virus, is relatively stable and resistant to most disinfectants (Wierenga et al. 2023). **Consequences –** Not reported in Australian LPs, only reported in yellow eyed penguins which are an endangered species facing differing pressures. However, if exposed consequences to population may be moderate however uncertainty rating is moderate as only one case report exists overall of low disease risk.	1	VL	M	**M**	**L**
Herpes disease	Herpesvirus-like infection, *Avian herpesvirus 1* **Infectious laryngotracheitis virus (Herpesviridae)**	**Host range** – described in African penguins in captivity and rehabilitation. Associated lesions resemble those of infectious laryngotracheitis (*Gallid herpesvirus 1* syn: *Avian herpesvirus 1*) (Stidworthy and Denk 2018). A novel avian *alpha herpesvirus*, preliminarily designated *Spheniscid herpesvirus 1*, has been isolated and characterized in Humboldt and African penguins (Pfaff et al. 2017). Parsons et al. (2016) reported no positive samples in serological testing for ILTV in 578 African penguins. Not reported in Australian LPs. **Impact** – Affected chicks presented with poor weight gain, airsacculitis, and congested, oedematous, and firm lungs (Parsons et al. 2015) **Transmission** – occurs via the faecal-oral route, although adult birds rearing chicks may pass on infection via regurgitated food. There is also evidence of transmission via feather dander. Individual birds which are infected early in life and which have recovered from the disease can become persistent carriers and intermittently shed virus into the environment. **Consequence** –Low potential disease risk for LP s being exposed in the wild and developing disease. If infected low likelihood of exposing, low likelihood of carriage and transmission given lack of prior positive disease testing and lack of clinical signs but medium consequence of disease to population. Overall, of low risk to LP population	1	L	M	**M**	**L**

Disease	Parasite	Hazard description	Species Group*	L	C	U	O
Newcastle disease and Avian paramyxoviruses **WHOA listed pathogen** *Avian orthoavulavirus 1* (AOaV-1), formerly known as *Avian avulavirus 1* (AAvV-1), *avian paramyxovirus 1* (APMV-1) and *Newcastle disease virus* (NDV), belongs to the family *Paramyxoviridae*, subfamily *Avulavirinae*, genus *Orthoavulavirus* (I nternational Committee on Taxonomy of Viruses (ICTV) 2019) Due to multiple name changes in recent years, the terms APMV-1 and Newcastle disease virus (NDV) still appear in the majority of scientific literature.	Avian paramyxovirus (APMV) A group of 13 serotypes which includes the notifiable disease Newcastle Disease, caused by virulent strains of APMV-1. Avian paramyxovirus (APMV-1) – virulent.	**Host range** – The virus is able to infect all orders of avian species, and virulent strains can cause significant clinical signs. Newcastle disease (APMV1) can have devastating effects on the poultry industry due to the high morbidity and mortality associated with the virulent strain. Epizootics continue to occur on a regular basis in Central and South America, Africa, and Asia, while sporadic epizootics occur in Europe (Hines and Miller 2012). Penguins that were seropositive for NDV have been demonstrated in the Antarctic (Morgan and Westbury 1981), Argentina (Karesh et al. 1999), Macquarie Island (Morgan et al. 1981) and South Shetland Islands (Thomazelli et al. 2010). Thomazelli et al. (2010) determined that the strains detected in penguins at the South Shetlands Islands had low pathogenicity. NDV infection has also been demonstrated in captive penguins in the United States (Pierson and Pfow 1975), where a velogenic neurotropic strain was identified in birds being shipped from Scott Base in the Ross sea to the states resulting in multiple deaths. Infection and disease was also reported and in a captive king penguin (Krauss 1963). It is clear that penguins are susceptible to this virus and that some NDV strains, presumably those with low pathogenicity, circulate in wild penguin populations. The virus can infect nonavian hosts, including primates (humans, monkeys), rabbits, and pigs. Human infection may result in transient conjunctivitis and/or flu-like signs (AHA 2014; OIE 2019) and more recently a fatal case of neurological infection caused by PPMV-1 has been reported in a child (Hurley et al. 2023). **Impact** – Clinical signs of Newcastle disease in chickens include drop in egg production, respiratory distress, listlessness, weakness, and central nervous system symptoms (Hines and Miller 2012). Paramyxovirus seropositivity is widespread in free-ranging penguins but disease is rare (Stidworthy and Denk 2018). Recurrent, seasonal high mortality events in double-crested cormorants, attributable to virulent Newcastle Disease Virus (NDV); Avian paramyxovirus-1, occurred in breeding colonies in Canada and USA since 1992 (White et al. 2015) with deaths of 20,000 or more and up to 90% mortality. Often associated with neurological signs. APMV-1 infection not reported in cormorants in Australia. Wild birds usually do not demonstrate classic clinical signs of virulent paramyxovirus infection however birds in a weakened state (e.g. during annual migrations), co-infections or adverse environmental conditions may develop clinical signs and die. Note that wild waterfowl in Australia do not migrate (WHA 2016) **Transmission** – Highly contagious spread via inhalation and ingestion. Environmental transmission (e.g. via nesting hollows or nesting material) is also suspected, due to the long term persistence of the virus (Eastwood et al. 2019). **Consequence** - Activation of infection reported with immunosuppression of the host. Significance of antibody response unknown however disease has occurred in Adelie penguins believed to have become infected in the wild and in a captive King penguin (Krauss et al. 1963). Most chronically infected birds eventually die as a result of impaired eating and/or secondary infections due to the immunosuppressive nature of the infection (Raidal et al. 2015). Overall, of medium population consequence if infected by a pathogenic strain and could predispose penguins to opportunistic infections. Could also cause significant poultry disease with potential trade implications from gulls which visit mainland poultry facilities.	1	L	M	**M**	**M**
			2	L	M	**L**	**M**
			3	L	M	**M**	**M**
			4	L	H	**L**	**M**

		There are 49 records of testing for avian paramyxovirus in the eWHIS record (some referring to multiple penguins), and there were no positive cases in little penguins, or any of the other penguins tested.					
	Avian paramyxoviruses (avirulent)	**Host range** – The virus is able to infect all orders of avian species. Avian paramyxoviruses are widespread among Adelie penguins in Antarctica (Morgan and Westbury 1988). Penguin species, including Antarctic penguins, have been described as potential reservoirs for several avian orthoavulaviruses, which could have the potential to infect other avian hosts (Wille et al. 2019a). Previous studies have described the presence of APMV-1, APMV-3, APMV-7, APMV-8, and other as-yet uncharacterized avulaviruses in Antarctic penguins (Neira et al. 2017). **Impact** – Paramyxovirus seropositivity is widespread in free-ranging penguins but disease is rare (Stidworthy and Denk 2018). The pathogenesis of APMV serotypes (excluding APMV-1) is not well understood. APMV-2, -3, -4, -6, and -7 cause decreased egg production in affected production birds. Mortality rate of budgerigars infected with APMV-5 can be 95-100%. Poultry operations are at risk for infection with several serotypes of APMV that can cause significant production loss. APMV-2, -3, -6, and -7 are most commonly associated with outbreaks in commercial poultry operations (OIE 2021) **Transmission** – The mechanism of transmission is not well-established for APMV serotypes other than APMV-1. It is suspected that contact with excreta from infected birds may spread the viruses (OIE 2021). **Consequence** - Penguins are susceptible and infected by this virus and some NDV strains, presumably those with low pathogenicity, circulate in wild penguin populations. Overall, of low population consequence.	1	L	L	**L**	**L**
			2	L	L	**L**	**L**
Penguin diptheria	Yellow eyed penguin megrivirus, picornavirus	**Host range –** seasonally recognized in endangered yellow-eyed penguins in New Zealand only. Yellow-eyed penguins are significantly impacted by avian diphtheria with reports in 2004 of 90% of New Zealand mainland penguins having contracted the infection, and over 50% resulting in fatality (Alley et al. 2017). **Impact -** Diphtheritic stomatitis, characterized by a thick fibrinopurulent exudate in the oral cavity and signs including inappetence and significant weight loss, is responsible for significant mortality among the young chicks. Bacterial plaques form in the mouth and can be inhaled, causing aspiration pneumonia, or result in a blockage which prevents feeding and causes dehydration. These chicks are treated with antibiotics, amoxicillin-clavulanic acid or enrofloxacin, but do not always recover from the infection. Saunderson et al. (2021) identified a novel Corynebacterium species associated with diphtheritic stomatitis in yellow-eyed penguins with potential virulence genes that are likely involved in pathogenesis. May also have an underlying viral aetiology which predisposes to secondary Corynebacterium infection (Alley et al. 2017) **Transmission –** Typically through respiratory droplets, commonly sneezing or possibly vectors means of transmission yet to be definitively identified (Alley et al. 2017) **Consequence –** Only reported in endangered Yellow eyed penguins in NZ so exposure of very low likelihood on Phillip Island (Millowl) LPs however consequences to population are moderate to severe and moderate levels of uncertainty in regards to aetiology and transmission so overall assigned a low risk to the population.	1	VL	H	**M**	**L**

Puffinosis	Type II coronavirus suspected but not confirmed	**Host range –** Disease resembling viral disease 'Puffinosis' in Manx shearwaters (*Puffinus puffinus*) reported in Gentoo penguins at Signey Island Antarctica (MacDonald and Conroy 1971) **Impact –** Several hundred penguin chicks found dead. Penguins were in good body condition but had multiple ulcers 2-4mm diameter on the dorsal aspects of the feet/ Adelie and chinstrap penguins in adjacent colonies were not affected (Clarke and Knowles 1993). **Transmission –**Unknown **Consequence -**One event only, significance uncertain therefore of low population consequence.	1	L	H	**M**	**L**

BACTERIAL							
Disease	**Parasite**	**Hazard description**	**Species Group***	**L**	**C**	**U**	**O**
Avian Chlamydiosis	*C. psittaci* - Avian chlamydiosis refers to the disease in birds and psittacosis refers to disease caused by *C. psittaci* in humans.	**Host range** – Global distribution, all bird species are susceptible to *C. psittaci* infection however disease is rarely reported in wild bird populations (WHA 2017b). Although avian chlamydia increasing in frequency in LPs and Yellow-eyed penguins in New Zealand (B. Gartrell pers comm Nov 2023). Zoonotic pathogen. An outbreak of *C. psittaci* occurred in an outdoor colony of 63 Magellanic penguins at the San Francisco Zoo (Jencek et al. 2012). Serological surveys (complement fixation test) show that wild populations of various penguin species have antibodies to *C. psittaci* (Gallo-Vaulet et al. 2022). However unknown if results reflect exposure to *C. psittaci* or to other antigenically similar organisms. Cameron (1968) reported on the isolation of "a member of the Psittacosis-Lymphogranuloma Venereum group of viruses" from the lungs of an emperor penguin chick found dead in Antarctica, but it is unknown whether this organism was *Chlamydia* sp. or another *Chlamydiales*-like organism. Recent attempts to detect DNA from *C. psittaci* in faecal samples from wild penguins with real-time PCR tests failed, but Isaksson et al. (2015) detected DNA from *Chlamydiales* sp. in cloacal swabs from 7/105 chinstrap penguins sampled in the Antarctic Peninsula. **Impact** – The nature of disease in infected birds will depend on the host and strain of bacteria. Stress and immune function can also influence susceptibility to infection and disease. Clinical signs of disease include lethargy, weakness, reduced appetite, ruffled feathers, weight loss, ocular and nasal discharge, diarrhoea and green coloured urates. More severe cases may be fatal (WHA 2017b). However, there may be no clinical signs and many chronically infected birds show no signs until stressed. Persistent infections normally result in a bird that is either clinically normal or shows only mild signs. In the outbreak of *C psittaci* in 63 Magellanic penguins at the San Francisco Zoo affected penguins presented with inappetence, lethargy, and light green	1	L	M	**M**	**L**
			2	L	L	**M**	**L**

		urates. Hematologic and serum biochemical findings were consistent with chronic inflammation. Penguins did not respond to initial supportive and antimicrobial therapy, and three died. Necropsy results reported hepatomegaly and splenomegaly, and histologic lesions included necrotizing hepatitis, splenitis, and vasculitis. *C psittaci* was confirmed by Gimenez staining, immunohistochemistry, and tissue PCR (Jencek et al. 2012). Diagnosis can be challenging, as *Chlamydia* are often shed intermittently (Stokes *et al.* 2020, Gerlach 1994) particularly in chronically infected birds, which can result in false negative test results. In humans, clinical signs vary from mild to potentially fatal systemic disease with severe pneumonia (CDC 2010). Commonly reported clinical signs include conjunctivitis, non-productive cough, fever, chills, headache, malaise, and myalgia (WHA 2017b). **Transmission** – Transmission mostly occurs via ingestion or inhalation, with infectious material shed in faeces, nasal secretions, and eye secretions. Exposure to infection may arise from close contact with an infected bird or via contamination of the environment with infectious material, enhanced by prolonged or persistent shedding of highly resistant elementary bodies from infected birds. Aerosolisation may be an important route for dissemination of infection in a flock setting, especially around communal feeding areas. Levels of vertical transmission are considered to be low, though transmission from parent to offspring may occur through regurgitated crop content (Andersen and Franson 2007). **Consequence** – LPs could be exposed and infected to chlamydia, however disease development seems dependent on immune status. Most wild birds are asymptomatic yet has caused disease in captive zoo populations and is reportedly increasing in frequency in New Zealand penguin colonies (B. Gartrell pers comm Nov 2023). Low population consequences to both LPs and other avian sp if infected and human consequences vary from mild to fatal depending on immune status. Based on the eWHIS record, avian psittacosis was reported in 1 emaciated juvenile little penguin found dead in Lorne, Victoria.	3	L	L	**L**	**M**
Avian cholera	*Pasteurella multocida*	**Host range** - *P. multocida* is highly infectious, and likely occurs globally in domesticated and wild fowl causing significant and explosive mortality. Over 100 species of wild birds are susceptible to disease with North American wild fowl the most severely impacted (Botzler 1991). Outbreaks of avian cholera leading to significant mortality events in free-ranging birds, are reported in southern rockhopper (Jaegar et al. 2018), Adélie (Leottie et al. 2006) and macaroni (Cooper et al. 2009) penguins, brown and great white pelicans, double-crested and Cape cormorants, common loons, pie-billed, horned, eared, and Western grebes (Stidworthy and Dent 2018). Infected birds can remain carriers for life. Avian cholera has not been reported in Australian penguins to date, but has been found in rockhopper penguins on Campbell Island (New Zealand) (de Lisle et al.	1	L	H	**L**	**M**
			2	L	H	**L**	**M**

		1990). Can cause human infections from an animal bite or scratch, mainly from domestic dogs and cats. Infections can also arise through inhalation of bacteria which is most likely to happen in confined areas of air movement where a large amount of infected material is present (e.g. during disease control operations). **Impact –** Infection by a virulent strain usually leads to acute septicaemic disease with high morbidity and mortality. Birds may die within a few hours of the onset of clinical signs (Friend 1999a). Clinical signs typically are neurological or respiratory in origin. However, the disease may also occur in a chronic form. Birds with chronic avian cholera constitute a major reservoir of disease in a population (Songer and Post 2005). Gross and histological findings include haemorrhage, intestinal hyperemia, hepato- and splenomegaly, and necrotizing hepatitis and splenitis containing intralesional Gram-negative coccobacilli (Stidworthy and Dent 2018). In domestic fowl it has emerged as a disease of significant economic concern to the Australian free-range layer industry (Singh et al. 2013, Scheelings 2022). Not considered a high risk disease for humans although infections are not uncommon (Cromie et al.2012). **Transmission –** Inhalation and ingestion. Ingestion of contaminated water and food sources (Samuel et al. 2007). Diseased birds contaminate their environment, food and water mainly via respiratory excretions. Dead birds and fomites including equipment used by humans may also serve as sources of infection. Predator attacks: Non-fatal predator attacks from wild or domestic animals (dogs, cats, are known to be carriers of high amounts of the bacterium in their oral cavities and underneath their nails). Any chicken that has been in a predator's mouth or scratched by a predator should be treated immediately with appropriate antibiotics. **Consequence** – PINP LPs likely to be susceptible given other penguin species susceptibility and reported epidemic mortality. Fowl cholera is also known to be present in Australian domestic fowl flocks (Singh et al. 2013). Kelp gulls, which are present all year round within the penguin colony at Phillip Island (Millowl) (Dann 2007), have been shown to be important vectors for cholera outbreaks in other species of penguins (Leotta et al.2006). The greatest risk of cholera incursion is likely to come from kelp gulls that travel from the island to mainland sites, where it is possible for them to have contact with domestic chickens in both commercial and private settings (Scheelings 2022). There are no records of avian cholera in the eWHIS record for little penguins.	3	L	M	**L**	**M**
			4	L	H	**L**	**M**
Avian mycobacteriosis (tuberculosis)	Caused principally by bacteria from the *Mycobacterium avium-intracellulare complex* (MAAC). *Mycobacterium avium* subspecies *avium (*MAA) and *M. genavense* (MG) most common mycobacterial infections in birds.	**Host range** –Ubiquitous, with a global distribution. Mycobacterial infections have been described predominately in zoological collections, backyard chickens, and pet birds in Australia (Sangster and Vinette-Herrin 2012). There are only a few reported cases of mycobacterial infection in free-ranging wild birds and none in penguins. Current data suggests that MAA does not cause disease in humans or does so very rarely. *M. genavense* reported in a captive African penguin (Krause et al. 2015) and can causes disease in humans that are severely immunosuppressed (Hoefsloot *et al*. 2013). *M. fortuitum* reported in a captive Southern Rockhopper penguin on necropsy exam (Buscaglia et al. 2020) **Impact** – Whether exposure results in infection will depend on the number of organisms to which the bird is exposed and the host's immune response to infection. Mycobacteria typically cause a slowly progressive and often fatal disease in the birds they infect. Clinical signs are non-specific	**1**	**L**	**M**	**L**	**L**
			2	**L**	**M**	**L**	**L**

		and include reduced appetite, progressive weight loss, ruffled feathers, weakness, diarrhoea, and a distended abdomen (WHA 2013b). Reported in LPs in a US zoo diagnosed via opportunistic CT for a separate research study. Penguins appeared healthy and free of outward signs of disease. However, after the loss of three individuals, mycobacterial disease due to *Mycobacterium avium-intracellulare* complex was confirmed on necropsy exam. Surviving penguins commenced multiple antibacterial treatment (Rivas et al. 2019). *M. genavense* was diagnosed on necropsy exam of a captive African penguin from a private collection in California USA initially presenting with anorexia, and laboured breathing with a history of weight loss and poor appetite (Krause et al. 2015) **Transmission** – Infection with MAA or MG is likely to be the result of ingestion or inhalation of the aerosolized organism. Ingestion of contaminated soil or water is a route of exposure for all birds (Whiley *et al.* 2012). Mechanical spread of infection via ectoparasites (particularly ticks) has also been demonstrated (Converse 2007). **Consequence** - Possibly endemic at Phillip Island (Millowl), and LPs already exposed, so low likelihood and low consequence as it would probably only impact individual animals of lowered immune status. Very low level of risk that wild LPs will be exposed from sympatric species, particularly waterfowl. Similarly, low likelihood of carriage and transmission to humans and disease likely of low consequence to humans unless immunocompromised. There are eWHIS records for 8 captive little penguins with mycobacteriosis; 1 x M. avium complex positive (captive, Doonside, NSW), 1 x non-speciated (captive, Sydney, NSW), 4 x M. intracellulare (captive, Main Beach, QLD), 3 x M. intracellulare (captive, Main Beach, QLD). In addition, there is a record for non-speciated mycobacteriosis in a free-ranging emaciated short-tailed shearwater from Warnambool, Victoria.	**3**	**VL**	**L**	**L**	**L**
E.coli infection	*Escherichia coli, Escherichia albertii*	**Host range** – *E.coli* present in the intestinal tracts of warm-blooded species and in the environment. Has been associated with enteritis in captive penguins (Stidworthy and Denk 2018). **Impact –** *E.coli* – Birds may develop gastrointestinal signs or present with sudden death. Humans exhibit characteristic symptoms of enteric bacterial infections including, abdominal pain, bloating, vomiting, diarrhoea, dehydration and fever. *E. albertii* **-** Similar to *Salmonella,* infection can produce asymptomatic carriers that shed bacteria in their faeces. Like other enteric pathogens it is likely to cause disease in circumstances where hygiene is poor or for wild birds where animals are concentrated. **Transmission –** Faecal-oral - typically infected as a result of oral consumption of contaminated feed or water. Human exposure is most likely to occur as the result of contact with wild birds and possibly by ingestion of contaminated water or food (Gordon 2011). **Consequence** – LPs may carry *E.coli* commensally developing disease if become stressed. Medium likelihood of carriage but of low consequence unless stressed. Stress can increase disease susceptibility, shedding of infectious agents, and severity of clinical signs. Can be managed by recommended biosecurity practices.	**1**	**M**	**L**	**L**	**L**
			2	**M**	**L**	**L**	**L**
			3	**M**	**L**	**L**	**L**

Erysipelas	*Erysipelothrix rhusiopathiae* The disease caused by *E. rhusiopathiae* in animals is called erysipelas, whereas in humans it is referred to as erysipeloid.	**Host range –** Ubiquitous in the environment, Infections most commonly reported in ducks and geese but can occur in multiple avian sp. (Boerner et al. 2004). An outbreak of erysipelas was reported in a colony of captive Humboldt penguins (Xie et al. 2019) and a little penguin (Boerner et al. 2004). Rodents, pigs and raw fish are reservoirs of infection. Zoonotic, but rare to catch from birds. **Impact –** Wild birds are a potential reservoir and usually asymptomatic. Occasional deaths in wild birds are associated with effluent from ponds associated with livestock. Affected birds are typically found dead with little to no prior signs of illness. Pathology may reveal evidence of septicaemia, with congestion of internal organs, multiple internal haemorrhages and a swollen spleen (Ladds 2009). Captive Humboldt penguins (Xie et al. 2019) presented with lethargy, inappetence, regurgitation and sudden death. The likely source of infection was the fish they were fed. In humans frequently presents as an erysipeloid, an acute cellulitis at the portal of entry and uncommonly as a systemic infection typically involving the endocardium (subacute endocarditis) or joints (Galindo-Cardiel *et al*. 2012). **Transmission –** Direct horizontal transmission by asymptomatic carriers such as pigs, turkeys, or contaminated fish food, as well as indirect horizontal transmission by the means of fomites such as contaminated soil or mechanical vectors such as arthropods (Galindo-Cardiel et al. 2012). Infection results from entrance of the organisms through breaks in the skin, through the mucous membranes, and possibly by mechanical transmission via biting insects. Erysipelas typically occurs sporadically after heavy rainfall, and animals are usually infected when they ingest the bacterium. Captive birds may be at more risk when housed in high stocking densities and under physiological or social stresses, such as territory maintenance and cold winters (Ladds 2009). **Consequence –** Low likelihood of exposure as PINP is a national park with minimal access to livestock. Wild LPs unlikely to ingest the bacterium in living fish. Therefore, very low likelihood of exposure but consequences of infection medium but would likely only cause sporadic deaths.	**1**	**VL**	**M**	**L**	**L**
			3	**VL**	**M**	**L**	**L**
Mycoplasmosis	*Mycoplasma spp.*	**Host range –** *Mycoplasma sphenisci* was described in an African penguin showing signs of upper respiratory tract disease in a North American aquarium (Frasca et al. 2005) and *M. lipofaciens* was identified from the lungs of a Fiordland penguin (*Eudyptes pachyrhynchus*) on necropsy with associated bronchopneumonia in New Zealand (Buckle et al. 2013). *M. sphenisci* and other *Mycoplasma* spp. however have also been detected in the faeces of apparently healthy penguins in Antarctica and subantarctic islands (Banks et al. 2009). Reported in wild anseriformes, falconiformes, Galliformes, Gruiformes, Columbiformes and Passeriformes however Mycoplasmosis has not been reported in Australian native birds (Ladds 2009). **Impact –** Causes chronic respiratory disease in domestic poultry and infectious sinusitis in turkeys with resultant significant economic losses from carcass condemnations, reduced feed and egg production and retarded growth (Luttrell and Fischer 2007). The disease presents with coughing, rales, sinus exudate, swollen sinuses and airsacullitis. **Transmission –** Horizontal through direct contact or aerosol droplets between a carrier and a susceptible bird (Luttrell and Fischer 2007). **Consequence –** While LPs may be susceptible, likelihood of exposure to infection seems low although consequences may be medium for the individual would be low for the population. No	**1**	**L**	**L**	**M**	**L**

		current evidence of infection in Australian LPs but suspect minimal targeted surveillance has occurred. Co-infection of *Mycoplasma* sp. with aspergillosis was suspected in two little penguin records from eWHIS.					
Necrotic enteritis	*Clostridium perfingens* and others such as *E.coli*	**Host range –***Clostridium perfringens* and *E.coli*, are most commonly isolated within the intestine and other tissues of birds with necrotic enteritis. Outbreaks of Clostridium enterotoxaemia have caused peracute and acute deaths in multiple penguin species in captivity (Greenwood 2000) **Impact –**Affected birds are in good body condition, but obtunded, dehydrated, regurgitate clear fluid, and have soiled vent feathers as a result of watery diarrhoea. The bird's abdomen may be palpably distended. Alternatively, birds with necrotic enteritis are found dead or moribund (Rose 2005). *Salmonella* and *Clostridium* species are the most significant enteric pathogens of captive penguins (Stidworthy and Dent 2018). In penguins typically found dead in excellent body condition or may present with acute depression and brown, foul-smelling diarrhoea (Stidworthy and Dent 2018) **Transmission –** Necrotic enteritis is a multifactorial disease. Several components must occur simultaneously for commensal *Clostridium* to begin multiplying in large numbers and producing toxins that harm the intestine. Sudden diet change has been suggested as a means of causing intestinal overgrowth with *Clostridium perfringens,* and subsequent necrotic enteritis (Pass 1993). **Consequence -** LPs may carry *Clostridium* and *E.coli* commensally developing disease if become stressed. Medium likelihood of carriage but of low consequence unless stressed. Stress can increase disease susceptibility, shedding of infectious agents, and severity of clinical signs. Can be managed by recommended biosecurity practices. Based on eWHIS, clostridial infection was suspected in an emaciated little penguin with enteritis in Tasmania.	1	L	M	**L**	**L**
Pseudomonas	*Pseudomonas aeruginosa* *Pseudomonas pseudomallei*	**Host range –** Global distribution, reported in psittacines, waterfowl, penguins and any species in contact with contaminated food and water sources. **Impact –** Death attributed to disseminated *Pseudomonas pseudomallei* infection reported in a captive macaroni penguin in Hong King likely subjected to temperature and humidity extremes (MacKnight et al. 1990). Associated with respiratory signs including panophthalmitis, air sacculitis, pneumonia, otitis media in psittacines (Samour 2000). Virulent strains can cause septicaemia and death. **Transmission –** through ingestion of contaminated feed and water or contamination of wounds and injuries (Ladds 2009) and tends to proliferate above 20°C. **Consequence –** Very low likelihood of exposure in wild LPs, if infected likely to be sporadic but could cause moderate consequences to the individual but overall low consequences to population. eWHIS record shows 5 captive little penguins which were found drowned may have been exposed to Pseudomonas in their food and water.	1	VL	M	**L**	**L**

Salmonellosis	*Salmonella* sp. *S.typhimurium and S. anatis)*	**Host range** – Global distribution, all avian spp. pets, livestock susceptible, also zoonotic. *Salmonellae* (*S. typhimurium and S. anatis)* may cause clinical disease in penguins, and some infected birds are asymptomatic carriers (Stidworthy and Dent 2018). **Impact -** Infection can result in a range of outcomes, from asymptomatic carriage to fatal infection. *Salmonella* typically causes sporadic deaths, though has been responsible for outbreaks of mortality in wild bird populations around the world. *Salmonella* and *Clostridium* species are the most significant enteric pathogens of captive penguins (Stidworthy and Dent 2018). Asymptomatic carriage of multiple drug resistant forms reported in Gentoo penguins in Antarctica (Retamal et al. 2017). Birds can shed the bacteria asymptomatically at least for 10 days, which is likely to contribute to the spread of infection to humans and other hosts (Connolly *et al.* 2006). Infected humans may exhibit typical signs of salmonellosis: including diarrhoea, vomiting and fever. **Transmission** – Faecal-oral - typically infected as a result of oral consumption of contaminated feed or water (WHA 2018b), asymptomatic carriers. Vertical transmission through the egg is also an important means of infection in domestic poultry. **Consequence -** Potential disease risk for LPs under other pressures in the wild. Moderate likelihood of exposure with variable consequences to the individual but low population consequences. Consequences generally low unless young, old, or stress-related immunosuppression. Can be managed by recommended biosecurity practices. eWHIS recorded *Salmonella typhimurium* infection from a cloacal swab in a live captive little penguin with no clinical signs.	1	M	L	**L**	**L**
			2	M	L	**L**	**L**
			3	M	L	**L**	**L**

Disease	Parasite	Hazard description	Species Group*	L	C	U	O
FUNGAL							
Aspergillosis	*A fumigatus, A. flavus, A. niger, A. nidulans and A. flavus-oryzae.*	**Host range –** Ubiquitous in the environment, a major cause of mortality in captive penguins and, less frequently, in free-living penguins. Sphenisciformes seem particularly susceptible to infection (Hocken 2002). Reported in two cases of stranded Magellanic penguins on necropsy (Ewbank et al. 2021). In little penguins, mycotic pneumonia reported in (PINP W 140392) (Idexx PINP 241103) on necropsy exam. **Impact –** Clinical manifestations depend on the infective dose, the spore distribution, pre-existing diseases, and the immune response of the host (Dahlhausen et al. 2004). An increased concentration of spores in the environment may predispose to aspergillosis. A warm environment, humidity, poor ventilation (Phalen 2000), poor sanitation, and the long-term storage of feed may increase the number of spores in the air. Factors impairing the bird's immunity can also predispose to mycosis (Beernaert et al. 2000). Infection typically results in a chronic, debilitating disease, with clinical signs of weight loss, reduced appetite, fluffed feathers, voice change and respiratory difficulty, though these signs may seem to appear suddenly and may be move apparent in the advanced stages of disease. Outbreaks of acute mortality have been reported, with affected birds found to be in good body condition yet succumbing to a rapidly developing fatal lung infection (Converse 2007b). Hocken 2002 reports typically diagnosed on post-mortem exam with 1/3 presentations: granulomatous tumors in the lungs and airsacs, white plaques in the airsacs or small nodules in the airsacs. Outbreaks may reflect a heavy load of fungal spores in the environment and are often seen in gregarious species that flock and feed together (Friend 1999b). **Transmission –** Inhalation of ubiquitous fungal spores. **Consequence –** Likely that wild LPs have been exposed to this infection. However environmental stressors may increase susceptibility to disease. If infected low likelihood of exposing other species as Aspergillus is ubiquitous in the environment and is not contagious from bird to bird. Consequences depend on the infective dose, pre-existing disease and immune response. In captivity cleaning and disinfection, good ventilation, and fresh feed is likely to minimise the number of spores birds will be exposed to. In free living populations likely to see disease secondary to other environmental or disease pressures. Aspergillosis was a common finding on the eWHIS record, with a total of 7 affected wild little penguins: 3 x Shoalwater, WA; 1 x Wye River, Victoria; 2 x St Kilda, Victoria; 1 x Edithvale (co-infected with *T. gondii*);	1	L	M	**L**	**L**

Candidiasis	*Candida albicans*	**Host range –***Candida* spp. are yeasts that are commensal within the upper gastrointestinal tract of a variety of birds (Hall and Rose 2021). Candidiasis is a common fungal disease, seen mostly in captive young or immunocompromised birds. Reported rarely in wild penguins one case report only in a wild stranded Magellanic penguin on necropsy (Ewbank et al. 2021). **Impact –**In Magellanic penguin associated with marked chronic necrotizing multifocal to coalescent pneumonia, airsacculitis, and oesophageal/gastric serositis with intralesional fungal structures. It most commonly affects unweaned captive chicks. Birds on broad-spectrum antimicrobials are most at risk. Often, candidiasis is secondary to poor husbandry and an unclean environment. Clinical signs include anorexia, crop stasis, white plaques in the oral cavity, regurgitation, and weight loss (Hall & Rose 2021). Thickening of the crop & may develop a "Turkish-towel" appearance. **Transmission –** Ingestion in food or in water is the usual means for its transmission (Friend 1990b) Contaminated environments, such as litter from poultry and gamebird rearing facilities, refuse disposal areas, discharge sites for poultry operations, and areas contaminated with human waste have all been suggested as sources for *Candidia* exposure for birds. **Consequence –** The infrequent reports of this disease in free-ranging wild birds, low likelihood of exposure unless immunocompromised, leads to disease presentations most likely being sporadic and therefore of low population consequence.	1	L	L	**L**	**L**

Disease	**Parasite**	**Hazard description**	**Species Group***	**L**	**C**	**U**	**O**
ENDOPARASITES							
CESTODES (tapeworms)							
Cestodiasis	*Tetrabothrius sp.* and *Tetrabothrius lutzi*	**Host range –** Reported in multi penguin species including LPs (Clarke and Keane 1993) Magellanic, emperor, king, gentoo as well as, pelicans, Kerguelen and imperial shags, boobies, loons, grebes, waved albatross, Antarctic petrels, greater, Manx and short-tailed shearwaters (Stidworthy and Dent 2018) in Australia, New Zealand, Antarctica and South America. **Impact –** Commonly found on necropsy exam in large numbers in intestinal tissue. Cestodes may be free in the intestinal lumen, clustered within mucosal ulcers, or deeply embedded in the intestinal wall. Histopathologic changes were most severe in adult Gentoo Penguins and included transmural fibrogranulomatous enteritis, haemorrhage, and oedema. Potential to be associated with disease and typically confounded by concurrent disease (Ladds 2009). As observed in many host-parasite systems, parasites may not produce clinical disease until the host becomes stressed or highly infected. May have concurrent signs such as weight loss, decreased appetite and diarrhoea. **Transmission –** Probably marine and other aquatic invertebrates such as crustaceans serving as intermediate hosts (Ladds 2009) **Consequence –** Moderate likelihood of exposure and infection and consequence affected by concurrent disease however likely of low consequence to the population	1	M	L	**L**	**L**

		Cestodes have been reported from over 30 records (some of which are multiple penguins per record) in eWHIS.					
BLOOD PARASITES							
HAEMOSPORIDIANS Avian malaria	*Plasmodium* spp. Although avian malaria is a term that should be used to refer specifically to infections with *Plasmodium* spp. it is often applied to any haemoparasite infection in birds.	**Host range** – Global distribution, infection with these protozoan parasites is common in many species of birds in Australia. Infection can also occur in poultry species. Seven genera of haemoparasites have been reported to infect penguins (Scheelings 2022). Infection with *Plasmodium* sp, has been documented in 13 species of penguins in both wild and captive settings, including king, Humboldt, African, northern rockhopper, yellow-eyed, Snares, chinstrap, little, gentoo, Macaroni, Galapagos, and southern rockhopper (Vanstreels et al. 2016). In a molecular screening of LP ticks (*Ixodes kohlsi)* from PINP, evidence of apicomplexa was found, however, the genus and species was unknown (Ghafar et al. 2023). In wild LPs in the islands in the Gulf of St Vincent, South Australia Burt et al. (2016) reported that *Haemoproteus* spp. was seen in 98% of individuals, and *Plasmodium* spp. detected in 82% of penguins. Interestingly, 80% of all penguins had multiple infections with both *Plasmodium* and *Haemoproteus. Plasmodium* spp. have not yet been reported from Tasmania (Iutruwita) (Wells, M. pers. comm.). Increasing reports of frequency of occurrence in New Zealand penguins (B Gartrell pers comm, Nov 2023). **Impact** - Infections with multiple species and genera of haemosporidia are common and infections may persist for years, although a percentage of infected birds will cure themselves of infection (van Rooyen et al. 2013). These parasites have historically been considered to be host-adapted and to cause little disease in the species that they infected (WHA 2013e). Infection is endemic in large parts of the world and is typically subclinical in birds that are native to these regions. Several penguin species have evolved in non-endemic regions without the selective pressure that these parasites exert and are highly susceptible to infection when moved to endemic regions, for example, in the context of zoological collections or rehabilitation centers (Ings and Denk 2022). In penguins, avian malaria (*Plasmodium*) is one of the most significant parasitic diseases, with mortality rates as high as 50%–80% (Grillo et al. 2016). Disease is common in captive penguin colonies and rehabilitation centers, and also occurs in the wild (Stidworthy and Denk 2018). Disease risk is highest in chicks, juveniles and naive adults, and in animals with outdoor access. Stressors (moult, chick rearing, or poor husbandry) increase mortality. Clinical signs range from acute death to lethargy, anorexia, depression, vomiting, dyspnoea, pale mucous membranes (anaemia), and behavioural separation. Severe forms induce neurological signs including motor incoordination, seizures, and paralysis. Antemortem diagnosis is difficult as animals frequently succumb rapidly without detectable blood parasitaemia (Stidworthy and Denk 2018). Fatal infection is most commonly associated with *Plasmodium relictum* or *P. elongatum*, but cases involving *P. cathemerium, P. juxtanucleare, P. tejerai, P. nucleophilum*, and *P. unalis* have been reported (Clarke and Kerry 1993). In birds that survive, periods of recrudescence during spring or breeding season have been reported (Atkinson 2008a).	1	L	M	**M**	**M**

		Transmission – Seasonal and dependent on population of vectors. In Australia vectors typically active during spring and summer. Birds are infected by the parasites' sporozoites when they are bitten by the infected insect vector the mosquito (Culicidae), particularly *Culex* spp., *Mansonia* spp., *Culiseta* spp. and *Aedeomyia* spp. (Vanstreels et al. 2016). The geographic distribution of *Culex* mosquitoes overlaps with the breeding habitat of penguins in Peru, Chile, Namibia and Australia, and it is therefore plausible that wild penguins in these countries may be infected by *Plasmodium* sp. (Vanstreels et al. 2016). **Consequence** - Highly likely that haemoparasites, the agents of avian malaria, are already present in the Phillip Island (Millowl) population. However, the effects of infection may be heightened if birds become compromised for some other reason, i.e. concurrent illness or environmental stressors. Consequences of infection to the population may range from mild or subclinical to significant mortality. Factors such as climate change, expansion of the range of some species, and possibly the introduction of new haemosporidial species into Australia may contribute to outbreaks, may predispose to an increased prevalence of infection and, additional outbreaks of disease in the future (WHA 2013e). eWHIS reports one suspected Plasmodium/Haemoproteus infection from a little penguin at Lorne, Vic (co-infection with Babesia); an undisclosed number of LPs were infected with Plasmodium in a mass mortality event (175 LPs) at Shoalwater, WA					
	Leukocytozoon spp.	**Host range –** Leukocytozoon has a world-wide distribution with the exception of South America, Central America and the Carribean islands where insect vector species do not occur (Rae 1995). The natural intermediate hosts are Simulid flies or Culicoides mosquitoes (Tudor 1991). *Leucocytozoon tawaki* has been described from Fiordland penguins and a New Zealand LP. *Leucocytozoon* spp. parasites have also been detected in African penguins, yellow-eyed penguins, and macaroni penguins and the endangered yellow-eyed penguin in New Zealand (Argilla et al. 2013). Surveillance for the parasite in a recent Tasmanian study of LPs did not detect any positive cases (Well, M. pers. comm). **Impact –** In wild penguins in which *L tawaki* occurs it is not known to be pathogenic (Peirce, Greenwood and Stidworthy (2005). Leukocytozoon infection with no associated clinical signs was reported in three macaroni penguins in an English zoo (Peirce, Greenwood and Stidworthy (2005). *L tawaki* has been detected in captive African penguins during treatment and rehabilitation following oiling within their home range (Brossy 1993). A yellow-eyed penguin chick had histological evidence of leucocytozoonosis with megaloschizonts in multiple organs throughout its body (Argilla et al. 2013). Clinical signs of leucocytozoon infections include visible hepatomegaly, dehydration, anorexia, depression, haemoglobinuria, haemolyic anaemia and acute death may also be reported (Rae 1995, Greiner and Ritchie 1994). **Transmission** – Seasonal and dependent on population of vectors. In Australia vectors are typically active during spring and summer. Birds are infected by the parasites' sporozoites when they are bitten by the infected insect vector. Leukocytozoon is transmitted by simulid black flies and transmission would require vector contact between infected and uninfected penguins. **Consequence** – LPs susceptible and vector present in Australia. Possibly already present in the Phillip Island (Millowl) population. However, the effects of infection may be heightened if birds become compromised for some other reason, i.e. concurrent illness or environmental stressors.	1	L	L	**M**	**L**

		Consequences of infection to the population may range from mild or subclinical to significant mortality. eWHIS reports suspected Leucocytozoon infection from wild Fjordland crested penguins at Aireys Inlet, Vic; Torquay, Vic; and Warnambool, Vic. None were reported from LPs.					
PIROPLASMIDS	*Haemoproteus* spp.	**Host range –**Two captive African penguins and two Magellanic penguins were found to be positive for *Haemoproteus* infection in two open-air aquariums in Japan (Inamuru et al. 2020). Inamuru et al. (2020) then went on to demonstrate penguins as competent hosts reporting successful replication in the host. Mortalities in LPs at Penguin Island in WA were attributed to a fulminant toxoplasmosis, with a concurrent *Haemoproteus* infection in some cases (Campbell et al. 2022). Four LPs from a cluster of 12 penguins that died of toxoplasmosis at Penguin Island WA (Campbell et al. 2022) were also infected with *Haemoproteus* spp. which were the first known recordings for LPs in WA (Cannell et al. 2013). It was postulated that biting midges on Penguin Island were the most likely vector of the blood-borne *Haemoproteus* spp., and that the strong La Niña period across 2010/2011 caused heavy rainfall during the spring and summer and a record number of hot days, subsequently leading to ideal conditions for midges or other vectors of the parasite. Recent surveillance for the parasite in Tasmanian LPs did not find any positive cases (Wells, M. pers. comm.). **Impact –** *Haemoproteus* spp. are generally considered the least pathogenic of avian haemosporidians; however, there are cases of lethal haemoproteosis (Atkinson and Van Riper III, 1991). The virulence of *Haemoproteus* spp. overall remains unknown (Vanstreels et al. 2016). However infected birds can be a source of infection for other penguins, including young or weak individuals which may show clinical symptoms. **Transmission** – Seasonal and dependent on population of vectors. In Australia vectors typically active during spring & summer. Birds are infected by the parasites' sporozoites when they are bitten by the infected insect vector biting midges, and Hippoboscid flies. The distribution of biting midges overlaps that of penguins breeding in Namibia, South Africa and Australia, possibly providing opportunities for *H.* (*Parahaemoproteus*) sp. inoculation. Similarly, captive penguins in areas of North and South America, southern Europe, Africa, Asia and Oceania could also be exposed (Vanstreels et al. 2016) **Consequence -** LPs susceptible and vector present in Australia. Possibly already present in the Phillip Island (Millowl) population. However, the effects of infection may be heightened if birds become compromised for some other reason, i.e. concurrent illness or environmental stressors. Consequences of infection to the population may range from mild or subclinical to mortality.	1	L	L	**M**	**L**

KINETOPLASTIDS		eWHIS records *Haemoproteus* sp. infection in 17 records of LPs (some records include multiple LPS). In one record of four deaths from Shoalwater, WA, the infection was believed to be the cause of death.					
	Babesia spp.	**Host range** – *Babesia* spp. are tick-borne protozoan parasites, and 16 avian-infecting species have been described, including one species (*Babesia peircei*) that infects penguins (Vanstreels et al. 2018). van Rensburg (2010) observed round intracytoplasmatic inclusions compatible with *Babesia* sp. in the blood smears of LPs at Tiritiri Matangi Island (9/79 = 11.4%) (Hauraki Gulf, NZ). *Babesia* sp. was identified in seven wild LPs, with positive individuals recorded in New South Wales, Victoria and Tasmania with prevalence estimated between 3.4% and 4.5%. A recent surveillance study of Tasmanian LPs suspected 70 out of 300 samples to be positive, though only 4 were confirmed through sequencing (Wells, M. pers. comm.). Sequencing revealed the same species as that recorded in Australian LPs by Vanstreels (2015), as well as being consistent with the species found in red-billed gulls, white-fronted terns and Australasian gannets in New Zealand (Paparini et al. 2014). Gene sequencing confirmed the identity of the parasite and demonstrated close relatedness to *Babesia poelea* from boobies (*Sula* spp.) and *B. uriae* from murres (*Uria aalge*). (Vanstreels et al. 2015) **Impact -** None of the Babesia-positive penguins in Vanstreels et al. (2015) study presented with signs of disease, confirming earlier suggestions that chronic infections by these parasites are not clinically significant to otherwise healthy LPs. However, Babesia infection may lead to mild anaemia in LPs (Sergent et al. 2004) and is associated with mild anaemia, leukocytosis and impairment of hepatic function in African penguins (Parsons et al. 2016). **Transmission** – Seasonal and dependent on population of vectors. In Australia vectors typically active during spring & summer. Birds are infected by the parasites' sporozoites when they are bitten by the infected tick vector. **Consequence -** Highly likely that haemoparasites, are already present in the Phillip Island (Millowl) population. However, the effects of infection may be heightened if birds become compromised for some other reason, i.e. concurrent illness or environmental stressors. Consequences of infection to the population may range from mild or subclinical to mortality. Babesiosis was recorded in eWHIS in one record from a wild LP at Lorne, Vic; and one juvenile LP at St Kilda, Vic.	1	M	L	**M**	**M**

	Trypanosomes spp.	**Host range –**Although originally reported in Australia in LPs, *Trypanosoma eudyptulae* was not subsequently detected in LPs in extensive surveys on the southeast coast (Vanstreels et al. 2015). Similarly, a recent (2020-2023) disease survey of Tasmanian LPs did not reveal any Trypanosome infections (Wells, M. pers. comm.). The initial report of detection was made by Jones and Woehler (1989), who described the parasite based on blood smears collected from wild little penguins at Marion Bay, Tasmania. This colony was devastated by wildfire and is no longer present. **Impact –** There is little evidence of pathogenic effect from Trypanosomes (Ladds 2009) and little is known about *T. eudyptulae* and its virulence (Vanstreels et al. 2016) **Transmission** – Seasonal and dependent on population of vectors. However, this parasite's invertebrate hosts remain unknown (Vanstreels et al. 2016). **Consequence** - There is little evidence of pathogenic effect from Trypanosomes (Ladds 2009). Little is known about *T. eudyptulae*, therefore it is difficult to evaluate any impacts on the conservation of LPs (Vanstreels et al. 2016) but is overall thought to be of low consequence.	1	L	L	**M**	**L**
Disease	**Parasite**	**Hazard description**		L	C	**U**	**O**
PROTOZOANS							
Coccidiosis	Coccidia known to infect Sphenisciformes include species of *Eimeria*, *Isospora*, and *Cryptosporidium*.	**Host range** – Global distribution, typically species-specific infection. Disease of significance to poultry industry and birds held in high stocking density, owing to high environmental load. Coccidia infections in wild birds rarely cause clinical signs; however, disease and mortality can occur with predisposing environmental and host conditions (Kay et al. 2022). In Sphenisciformes renal coccidiosis reported in wild LP on necropsy exam (Obendorf and McColl 1980, Hocken 2002). **Impact** – Frequently asymptomatic, disease in wild populations rarely reported, though disease can arise in some situations, with some parasite species infecting tissues other than the intestine (e.g. renal coccidiosis in LPs in Victoria (Obendorf and McColl 1980). In LP, renal coccidia may be particularly pathogenic, causing blockage of renal collecting ducts, chronic interstitial nephritis, inflammation and intrarenal ureteritis (Obendorf and McColl 1980). *Eimeria pygosceli* (*Eimeria* sp. and *Isospora* sp. identified in faecal samples from 360 examined Adelie, chinstrap and gentoo penguins (Golemansky 2003). The total prevalence of coccidian parasites was high: about 35% in all of examined penguins, but no host specificity observed possibly owing to the close phylogenetic relationships, common habitats and nesting territories, similar feeding and reproductive biology of the 3 penguin species. Pathology of coccidians requires further study to assess significance in penguin species (Kay et al. 2022) healthy individuals do not usually exhibit disease. **Transmission** – Faecal-oral transmission route, transmitted by contamination of water and feed by the infected bird droppings. **Consequence –** Highly likely that Phillip Island (Millowl) LPs have been exposed and infected with coccidia. However, the effects of infection may be heightened if birds become compromised for some other reason, i.e. concurrent illness or environmental stressors. Consequences of infection	1	M	L	**L**	**L**

		to the population may range from mild or subclinical to significant mortality, mortality is likely to be sporadic if present, therefore overall of low population consequence. Coccidiosis, especially renal coccidiosis, was reported in seven separate LP records on eWHIS, including animals from Portsea, Vic; Arthur River, Tas (renal coccidiosis); Torquay, Vic (renal), Aireys Inlet , Vic (renal); Lorne, Vic (renal); Rye, Vic (renal),					
	Cryptosporidium	**Host range** –*Cryptosporidium* spp. are parasitic intracellular protozoa that infect the digestive, respiratory, and urinary tracts of vertebrates. The disease affects many different avian species across all continents, and >25 species and genotypes of *Cryptosporidium* have been documented infecting birds. One report only of an outbreak of cryptosporidiosis in a colony of African penguins chicks Hurtado et al. (2020) not reported in other captive or wild penguins. **Impact** – Hurtado et al. (2020) reported on an outbreak of cryptosporidiosis in African penguin chicks admitted to a rehabilitation center in South Africa from February 2012 to October 2013. Eighteen cases were confirmed through histopathology. The most frequent clinical signs were regurgitation (78%), dyspnoea (72%), decreased weight gain or weight loss (72%), and lethargy (50%). Clinical signs began 8−46 d after hatching or admission (median: 13 d), and death followed 1−41 d after the onset of clinical signs (median: 13.5 d). *Cryptosporidium* spp. are frequently considered opportunistic pathogens that take advantage of the immunocompromised status of their hosts (Leitch and He 2012). Hurtado et als (2020) findings raise concern that *Cryptosporidium* sp. may be a conservation-significant pathogen for penguins, potentially causing substantial chick mortality as well as sublethal effects in surviving birds, such as a permanently impaired immune system. **Transmission** – Faecal-oral transmission route. The initial source may have been seabirds admitted for rehabilitation, free-ranging birds that occasionally visit the facilities, water or food-borne sources, or contaminated equipment or staff (Hurtado et al. 2020). **Consequence** - Likelihood of exposure and transmission for LPs low. Consequences typically low for the individual although could be medium if immunocompromised. Likely of low population consequence unless strain differences. For humans, low likelihood and low consequence if appropriate hygiene methods undertaken, although possibly medium if immunocompromised.	1	L	M	**M**	**L**
			3	L	M	**L**	**L**
Flagellates Giardia Trichomoniasis	*Giardia*	<u>*Giardia*</u> **Host range** – Giardia infection is common in a wide range of avian and mammalian hosts. Not all animal isolates cause disease in humans (Jakob-Hoff et al. 2016). **Impact** –Typically causes an asymptomatic carrier infection, but abdominal discomfort and watery diarrhoea can occur in immune-competent patients. However, infection may be chronic and life threatening in those that are immunocompromised (Greiner and Ritchie 1994). Younger birds appear more susceptible to infection, and co-infection with other pathogens, can occur (Ladds 2009). **Transmission** – Faecal-oral transmission route. **Consequence** – Likelihood of exposure and transmission for LPs low, although subclinical carrier status can occur. Consequences typically low for the individual although could be medium if immunocompromised. Likely of low population consequence unless strain differences. For humans, low likelihood and low consequence if appropriate hygiene methods undertaken, although possibly medium if immunocompromised.	1	L	L	**L**	**L**
			3	L	L	**L**	**L**

	Trichomonas gallinae	*Trichomonas gallinae* **Host range** – Global distribution in avian hosts, primarily a pathogen of pigeons and birds of prey (Forrester and Foster 2008), however, can infect a wide host range, including LPs (a commensal protozoan within the alimentary tract, sometimes causing tissue necrosis and secondary infections). Trichomoniasis appears to be a relatively common disease of both captive and wild birds in Australia (WHA 2014). Reported in LP (PINP DPI 220822) **Impact** – A commensal protozoan within the alimentary tract, sometimes causing tissue necrosis and secondary infections (Wiebkin 2011). Dyspnoea and asphyxiation may result if the lesions block the trachea (WHA 2014). Outbreaks of disease in wild bird populations are uncommon, and disease most often seen in pigeons, with spill over to birds of prey (WHA 2014). **Transmission** – Directly from an infected bird, without the development of a cyst stage, and may occur during communal feeding, courtship and/or rearing of young. **Consequence –** Potential disease risk for LPs being exposed at Phillip Island (Millowl). However, unlikely to transmit to other birds unless occupy similar ecological niche. Likelihood of carriage and transmission for LPs low, although subclinical carrier status can occur. Consequences typically low although could be medium if immunocompromised.	1	M	L	**L**	**L**
Disease	**Parasite**	**Hazard description**		L	C	**U**	**O**
Sarcocystis	*Sarcocystis*	**Host range** – The disease has been described in captive parrots and other orders of birds in captivity, such as Psittaciformes, Passeriformes, Columbiformes, Suliformes and Strigiformes, but is rarely observed in free-living birds (Acosta et al. 2018). Reported in penguins (gentoo, chinstrap), Australian pelican, northern gannet (encephalitis), little pied cormorants, and hoary-headed grebes (Stidworthy and Dent 2018). Muscle cysts from intermediate host infection with *Sarcocystis* spp. are a common incidental finding in bird, marsupial and eutherian mammal necropsies in Australia (Spratt and Beveridge 2018). Acute disease in Australian native birds has been reported primarily from those in captivity overseas (Ladds 2009). **Impact** – Infection often subclinical. When signs occur, may include lethargy, fluffed appearance, reduced appetite, severe dyspnoea, yellow pigmented urates, disorientation and seizures. Sudden death without prior signs of illness is a common presentation of Sarcocystis. S*arcocystis falcatula* is a well-known cause of fatal pneumonia in Old World psittacines. Fatal sarcosystosis due to *S. falcatula* reported in three captive penguins under managed care, one African penguin and two Southern rockhopper penguins (Kirejczyk et al. 2019). Randomly distributed foci of necrosis, inflammatory cell infiltrates, oedema, and variable numbers of round to elongated protozoal schizonts observed in sections of lung. Development of disease may be influenced by host species and infective dose. **Transmission** – Become infected from ingesting oocysts in contaminated soil, plant or invertebrate matter. Opossums are the definitive host in North America shedding oocysts in faecal material and contaminating the surrounding environment, which are then ingested by the intermediate avian host. The Australian definitive host(s) remains unknown.	1	L	L	**M**	**L**

		Consequence –Likely to be exposed through ingestion of oocysts as low number of possible definitive carnivore hosts present at Phillip Island (Millowl). LPs only capable of transmitting this parasite by being eaten, therefore the risk to humans and non-carnivorous animals is negligible. Overall low consequences of disease for LP population.					
Toxoplasmosis	*Toxoplasma gondii*.	**Host range** – Global ubiquitous organism with a broad mammalian and avian host range (Greiner and Ritchie 1994). Commonly infects Australian wildlife species and causes fatal infections in wild birds (Dubey et al. 1991). Retrospectively reported in 12 wild LPs on necropsy exam from the Penguin Island colony WA (Campbell et al. 2019). A recent survey of Tasmanian LPs found a low seroprevalence to infection of <3% (Wells, M. pers. comm.). **Impact** – Infection often subclinical. When signs do occur, they may include lethargy, fluffed appearance, reduced appetite, diarrhoea, disorientation and seizures. Sudden death without prior signs of illness is a common presentation. Development of disease may be influenced by host species and infective dose. In LPs on necropsy exam birds were in good body condition with reported hepatomegaly and splenomegaly, multifocal hepatic and splenic necrosis and numerous, 1–2 µm diameter protozoan parasites within the necrotic foci (Campbell et al. 2019). Similar to the reported necropsy findings in LPs by Mason et al. (1991) **Transmission** – Become infected from ingesting oocysts in contaminated soil, plant or invertebrate matter. Cats are the definitive host shedding oocysts in faecal material and contaminating the surrounding environment, which are then ingested by the intermediate host. Infection is not uncommon and may be asymptomatic, few develop disease. *Toxoplasma* infection is not uncommon in many marine mammals, implying that the disease can be transmitted by contamination of waterways with cat faeces (Jones and Dubey 2010). Filter-feeding fish may also take up *Toxoplasma* oocysts from contaminated water, and mice can become infected when feeding on these fish (Mason et al. 1991). The pilchard can take up *Toxoplasma* cysts and transmit the disease (Mason et al. 1991), and this is an important prey species for the LPs of Penguin Island, WA. Alternatively it has been shown (Campbell et al. 2019) that *Toxoplasma* can aggregate on extracellular polymeric substances in marine environments, enhancing the efficiency with which the organism could enter the marine food chain in coastal ecosystems (Shapiro et al. 2014). **Consequence** – Moderate likelihood of exposure, low number of cats present on Phillip Island (Millowl), however, other transmission pathways may exist as reported in Campbell et al. (2019). LPs only capable of transmitting this parasite by being eaten, therefore the risk to humans and non-carnivorous animals is negligible. Moderate consequences of disease for LP population. eWHIS reports toxoplasmosis in jus one wild, older LP from Edithvale, Vic.	1	M	M	**M**	**M**

NEMATODES (roundworms)							
Disease	**Parasite**	**Hazard description**		L	C	**U**	**O**
Capillariasis	Capillaria	**Host range –** Broad avian host range, including most pet and aviary birds (Greiner and Ritchie 1994). Reported in LPs in New Zealand on necropsy exam (Rensburg 2010, Bennett et al. 2021) and in an Emperor penguin from the Weddell Sea, Antarctica, from eggs in faeces (Kleinertz et al. 2014) **Impact –** Clinical signs of infection in captive birds are primarily gastrointestinal and include loss of appetite, weight loss, vomiting, and diarrhoea, which may contain blood (Greiner and Ritchie 1994). Capillaria sp. infections in wild birds are primarily an incidental finding during post-mortem examination (Hall and Rose 2021). **Transmission –** Most *Capillaria* spp have a direct life cycle, with eggs shed in faeces and transmission via the faecal-oral route. Some species have an indirect life cycle, utilising invertebrates such as earthworms as intermediate hosts (Greiner and Ritchie 1994). But unknown intermediate host in this species. **Consequence –** Potential disease risk for LPs which may commensally carry these parasites becoming immunosuppressed and developing disease. Low likelihood of carriage and transmission to other birds as unlikely to be in close contact, other than shearwaters as LPs habit a different ecological niche. Infection and disease mostly of low consequence although stressors may increase numbers of parasites and cause clinical disease but typically of low population consequence. Effectively managed by hygiene practices, antiparasitic agents and low host density.	1	L	L	**L**	**L**
Contracaecum - helminths	*Contracaecum eudyptulae*,	**Host range –** Important parasite of fish-eating mammals and birds (Norman 2005). Reported in multiple species; rockhopper, gentoo, macaroni, yellow-eyed, little, emperor, and Magellanic penguins as well as pelicans, cormorants and shags, gannets, boobies, darters, grebes, albatrosses, mollymawks, and shearwaters (Stidworthy and Dent 2018). Often reported on post-mortem exam in emaciated LP submissions (PINP UM 270404, DPI 220808) presumably in cases of starvation. Reported in Australian and New Zealand LP populations. *Contracaecum* spp are also of zoonotic importance and there has been a recent report of human infection with *Contracaecum* larvae (Shamsi 2017). **Impact –** Obendorf and McColl (1980) reported 75% nematode (*Contracaecum*) prevalence with heavy burdens and severe, chronic and acute gastric ulceration in association with poor body condition of wild LP. Similar findings were recorded by Norman et al. (1992) and Harrigan (1992), with even small numbers of nematodes causing small to medium sized gastric ulceration. *Contracaecum*, has been implicated as the cause of severe parasitic disease and mortality (Obendorf and McColl 1980) and appears to be exacerbated when combined with other forms of stress such as starvation or inclement weather (Obendorf and McColl 1980, Norman 1992). **Transmission –** Likely ingestion of parasites of the genus *Contracaecum* Railliet et Henry, 1912 (Nematoda: Anisakidae) have several species of fish as intermediate or paratenic hosts, and piscivorous birds and mammals as definitive hosts (Anderson 2000). Unfledged LP chicks likely infected with *C. eudyptulae* when fed by regurgitation from parents (Norman 1998).	1	M	M	**L**	**M**
			3	VL	L	**L**	**L**

		Consequence –LPs likely to commensally carry these parasites, however, may become immunocompromised by multiple agents, infectious and non-infectious, and develop disease. Low likelihood of carriage and transmission to other sea- and shorebirds as infection acquired through ingestion of intermediate host. Infection and disease mostly of low consequence although stressors may increase numbers of parasites and cause clinical disease but typically of low population consequence. Effectively managed in humans by hygiene practices.					
TREMATODES (flukes)							
Disease	**Parasite**	**Hazard description**		L	C	**U**	**O**
	Cardiocephaloides physalis	**Host range** - *Cardiocephaloides physalis* has caused mortality in the African penguin (Randall and Bray 1983) and Magellanic, and Humboldt species in Africa and South America (Clarke and Knowles 1993). Not reported in LPs. **Impact –** Caused death in penguin chicks with severe small intestinal infection (Clarke and Knowles 1993). Clinical signs of weakness and ascending paralysis in Jackass penguin chicks leading to death (Randall and Bray 1983). **Transmission –** Unknown suspected ingestion of intermediate host, likely from the marine habit and piscivory. **Consequences –** Low likelihood of exposure, has not been reported in LPs may lack the intermediate host in Phillip Island (Millowl) colony. Has caused sporadic deaths only in chicks. Therefore overall, of low population consequence.	1	L	L	**M**	**L**
	Galactosomum angelae	**Host range –** Reported in LPs from Kangaroo Island SA (Norman 2005) and New Zealand (Rensburg 2010) and in gulls, terns, pelicans and boobies (Pearson 1973, Clarke and Knowles 1993). **Impact –** Infection reported in intestine and liver with varied pathology. **Transmission –** Unknown suspected ingestion of intermediate host, likely from the marine habit and piscivory. **Consequence –** Low likelihood of exposure, has not been reported in LPs may lack the intermediate host in Phillip Island (Millowl) colony. Has caused sporadic deaths only therefore overall, of low population consequence.	1	L	L	**M**	**L**
	Mawsonotrema eudyptulae	**Host range –** Known to contribute to mortalities in Australian LPs in South Australia, Victoria and Tasmania (Norman 2005) **Impact –** Reported in Australian LP colonies in very high numbers and associated with significant liver enlargement, severe lesions and haemorrhage (Norman 1992). In post-fledglings, the liver fluke (trematode) appears to be pathogenic. The fluke, which lives in the bile ducts, sometimes kills large numbers of juvenile penguins, which are found as 'wrecks' of carcasses on beaches, known as 'beach-wrecks' (Obendorf and McColl 1980) **Transmission –** Unknown suspected ingestion of intermediate host, likely from the marine habit and piscivory. **Consequence –** Potential disease risk for LPs which may commensally carry these parasites becoming immunosuppressed and developing disease. Low likelihood of carriage and	1	M	M	**M**	**M**

		transmission to other sea and shorebirds as unlikely to be in close contact. Infection and disease mostly of low consequence, although stressors may increase numbers of parasites and cause clinical disease of moderate population consequence. Trematodiasis is common in LPs, with eWHIS reports in over 10 records.					
Renal fluke	*Renicola*	**Host range –** Reported in Australian and NZ LPs (Clarke and Knowles 1993, Rensburg 2010), pelicans, cormorants, boobies, loons, northern fulmars, shearwaters (Stidworthy and Dent 2018) and Magellanic penguins (Jerdy et al. 2016) in Brazil. **Impact –** Renal fluke infestations are often severe in starving LP and cause degenerative and necrotic changes in the kidneys of the host (Obendorf and McColl 1980). However, moderate renal fluke burdens have also been reported in apparently healthy birds suggesting the parasite may be commensal (Crockett and Kearns 1975). Reported to cause renal lesions in wild Magellanic penguins (Jerdy et al. 2016) on necropsy exam. **Transmission –** Unknown suspected ingestion of intermediate host, likely from the marine habit and piscivory. **Consequence -** Potential disease risk for LPs which may commensally carry these parasites becoming immunosuppressed and developing disease. Low likelihood of carriage and transmission to other sea and shorebirds as unlikely to be in close contact. Infection and disease mostly of low consequence although stressors may increase numbers of parasites and cause clinical disease of low to moderate population consequence.	1	L	L	**M**	**L**

ECTOPARASITES		Typically, seasonal infestations, especially during spring and summer. May cause disease in their own right, or may act as a vector for other diseases such as arboviruses and blood parasites.					
Disease	**Parasite**	**Hazard description**	**Species Group***	**L**	**C**	**U**	**O**
Fleas	*Parapsyllus* sp	**Host range & impact** – *Parapsyllus* sp. are reported in many species of penguins (Ladds 2009). However, there are no fleas reported on Antarctic penguin species as the climatic conditions are too extreme for their survival. Fleas can cause pruritus, localised skin irritation and feather damage and even anaemia in large numbers (Greiner and Ritchie 1994). Short-tailed shearwaters in southern Australia compete for occupancy of the same nesting burrows (Reilly and Balmford, 1975). A recent study by Wells et al. (manuscript in review) found greater flea abundance in natural burrows compared to artificial nests, as well as finding higher flea prevalence in more urbanised sites, during moulting, in spring and while in better body condition. **Consequence-** Potential disease risk for LPs being infected in the wild and transmitting fleas to susceptible sea and shorebirds spp. Overall of low population consequence to LPs. The study by Wells et al. found no association between the presence of flea and any health impacts.	1	L	L	**L**	**L**
			2	L	L	**L**	**L**

Lice	*Austrogonoides waterstoni*	**Host range & impact** – Biting lice have been identified in subantarctic and Antarctic penguin species other than the Gentoo and Chinstrap species (Clarke and Kerry 1993). *Austrogonoides waterstoni* is reported to infect LPs (Obendorf and McColl 1980). Most species are host specific and die when they depart from the host (Greiner and Ritchie 1994). They don't suck blood but rather chew on feathers and debris but can be associated with annoyance and irritation (Ladds 2009). **Consequence –** Potential disease risk for LPs being infected in the wild and transmitting fleas to susceptible sea and shorebirds spp. However, biting lice often host specific therefore unlikely to infect other hosts. Overall, of low population consequence to LPs.	1	L	L	**L**	**L**
Mites	*Ingrassia eudyptula,* & *Veigaia* sp.) and others	**Host range & impact** – Two feather mite species reported in LPs (*Ingrassia eudyptula,* and *Veigaia* sp.) in Australian LPs. Possibly co-evolved with *I dubinini* known to infect the short-tailed shearwater possibly transferred relatively recently from a shearwater or petrel species, especially given LPs and short-tailed shearwaters in southern Australia compete for occupancy of the same nesting burrows (Reilly and Balmford, 1975). Feather mites do not occur on any other penguin species (Mironov and Proctor 2008) likely owing to their strongly modified plumage with dense, stiff coverts and aquatic lifestyle. *Ingrassia* sp. reported primarily in charadriiforme (shorebird) hosts. No reports of associated pathology exist, so impact is unknown. Domrow (1987) reported the presence of skin dwelling, blood-sucking mites. *Gymnolaelaps annectans, Haemolaelaps fahrenholzi, H. flagellatus, H. pachyptilae,* and *Mesolaelaps australiensis* in LP nests. In addition, Domrow (1992) reported: heavy infestations of *Ornithonyssus bursa* (in LPs near Kembla NSW). **Consequence** – Potential disease risk for LPs being infected in the wild but low likelihood of transmitting mites to susceptible species as unlikely to be in close contact, with sp. other than shearwaters as habit a different ecological niche.	1	L	L	**M**	**L**
Ticks	*Ixodes eudyptidis* *Ixodes uriae*	**Host range and impact –***Ixodes eudyptidis* is found in southern Australia and New Zealand (Heath 2006) in seabirds including LPs. *Ixodes uriae* infection reported in LP and other sub-antarctic penguins in the subantarctic islands and Antarctic Peninsula (Mobendorf and McColl 1980). Gauthier-Clerc et al. (1998) reported cases of death in adult King Penguins hyperinfested by the tick *Ixodes uriae*. The preferred feeding sites on the host were the head and the neck for adult and nymph ticks, and the lower parts for larvae. King Penguins carried up to 263 adults, 548 nymphs, or 3,500 larvae. This species of tick is also known to be the vector of a large number of arboviruses (Chastel 1980). *Borrelia burgdorferi* was recently isolated from *I. uriae* on Possession Island (Gauthier-Clerc et al. 1998). *Ornithodorus* sp. species are found in the tropical and temperate regions in Humboldt, Galapagos, African and LPs (Clarke and Kerry 1993). *Ixodes kohsli* was associated with eosinophilia, *Babesia*-like organisms and a severe regenerative anaemia (Spielman and Cunningham 1993) in LPs. In a molecular screening of LP ticks (*Ixodes kohlsi)* from PINP, evidence of apicomplexa however genus and species unknown (Ghafar et al. 2023). Chicks are most frequently infected. The sites of attachment typically coincide with sparsely feathered regions such at the base of the bill, inside the external ears, crown of the head, neck and chin and may have associated irritation and oedema (Ladds 2009). Wells et al. (manuscript in review)	1	L	M	**M**	**M**
			2	L	L	**M**	**L**

		found higher prevalence of ticks in natural nests of LPs in Tasmania, during moulting and the breeding season, and with lower burrow densities. **Consequence** – Likelihood of exposure with low population consequence. Ticks may cause local irritation but may also be associated with tick borne disease such as *Babesia* infection. Tick parasitism may be an important negative factor for the health of adult King Penguins and, probably, of chicks, during their periods on land. Parasitism may also affect those foraging at sea to obtain food for their chicks (Kooyman et al. 1992). Wells et al. (manuscript in review) also found a positive association between heterophil/lymphocyte ratio and tick burdens.					

NON-INFECTIOUS						
Hazard	**Hazard description**	**Species Group***	**L**	**C**	**U**	**O**
CONGENITAL /DEVELOPMENTAL						
Beak malformation, carpal rotation Ventricular septal defects Splay leg	**Host range & impact** –Congenital defects are uncommonly recorded in free-ranging or captive penguins (Stidworthy and Dent 2018). A ventricular septal defect was reported in a 45- day old female Humboldt penguin, a juvenile rockhopper penguin, a captive macaroni penguin and a 27-day-old Adélie penguin (Laughlin et al. 2016). An outbreak of craniofacial deformity occurred in free-ranging yellow-eyed penguin chicks in New Zealand (Buckle et al. 2014). Malformed beaks and lack of flippers have been observed in LPs in Victoria (Reilly and Balmford 1975) raising suspicion of teratogenic exposures in breeding colonies (Stidworthy and Dent 2018). Splay leg presents in growing chicks – causing rotation and deformity to limbs. May result in permanent deformity if not treated. Unsure of incidence in wild LP populations. **Consequence** – It is likely that congenital and genetic abnormalities in free-ranging populations of all bird species are overlooked, since long-term survival to allow identification of affected birds is unlikely. While consequences may be high for the individual overall suspected low consequence to population as typically only sporadic in occurrence.	1	L	M	**M**	**L**
DEGENERATIVE						
Cataracts	**Host range & impact** - Cataracts are the most commonly reported age related ocular pathology in penguins, (Bliss et al. 2015) and recent evidence suggests that cataracts and vitreal degeneration may be under-recognized in zoo-captive adult pelicans (OConnell et al. 2017). **Consequence –** Captive individuals can be closely monitored to gauge their ability to cope with visual deterioration and appropriate enclosure modifications made as long as the eye is non-painful. In the wild suspected that most aged birds probably die at sea rendering them unavailable for examination and diagnosis. Likely within normal population limits but when advanced likely poor survivability as more prone to predation, drowning, age related demise. A cataract was reported from a dead, wild LP from Mount Martha, Vic, and a wild LP from Werribee, Vic.	1	L	L	**L**	**L**
Degenerative joint disease	**Host range & impact –**Multiple case presentations in aged, rehabilitated LPs in captive institutions at Perth Zoo and Penguin Island WA. Decreased joint rotation, and mobility often in aged individuals may limit survivability in the wild and increase likelihood of predation. Bilateral	1	L	L	**M**	**L**

	degenerative joint disease was diagnosed in a free-ranging, juvenile, yellow-eyed penguin with abnormal stance and decreased mobility (Buckle and Alley 2011). Necropsy identified bilaterally distended, thickened coxofemoral joints with increased laxity, and small, roughened and angular femoral heads. **Consequence –** Often requires euthanasia in wild individuals if advanced and associated debilitation. In aged, captive LPs managed with quality of life at the forefront, owing to impact on movement and concomitant generalised debilitation. Likely to be sporadic and more likely to be present in aged individuals therefore of low population consequence.					
INTOXICATION						
Algal toxins	**Host range & impact** – Algal blooms (a fast-growing dense population of algae) can result from an increase in nutrients (e.g. from storm water), and a combination of favourable environmental conditions (DOC 2022). Less than 2% of these blooms can release toxins (LAWA 2022). Aquatic birds feeding predominantly on fish and invertebrates are susceptible to algal biotoxins generated during harmful algal blooms (Landsberg et al. 2007). Only circumstantial evidence for algal blooms and red tides in Australian and NZ LPs (Fortescue 1995). An event in Bowen Island coincided with an algal bloom in Jervis Bay, reducing water visibility but no other pathology reported in LPs (Norman 2005). In yellow eyed penguins a brevetoxin has been isolated causing neurotoxic and haemolytic effects across multiple species including Brandt's cormorants, double-crested cormorants, pelagic cormorants, frigatebirds, and pelicans (Stidworthy and Dent 2018). In New Zealand in 2010, a red algae bloom was linked to climatic fluctuations caused by El Nino and resulted in deaths in LPs along the west coast of the north island. In the Falkland Islands (Malvinas) in November 2002, an algal bloom caused paralytic shellfish poisoning and the subsequent death of a large number of Southern Rockhopper Penguins and other seabirds (Uhart et al. 2004). Potential risk for LPs if suitable environmental conditions. Given such events can kill large numbers of seabirds, they will probably become a greater problem for penguins and other seabirds in the future if the frequency of harmful algal blooms increases as a result of regional warming and altered ecosystem properties (Tratham et al. 2014). Humans can also be impacted through inhalation and ingestion as can dogs coming into contact with contaminated fish. **Consequence –** Sporadic in occurrence, low likelihood of algal bloom, requires suitable environmental conditions. However, may become of higher likelihood with increased nutrient loads as a result of regional warming and altered ecosystem properties. Of moderate consequences to population if it were to occur, overall moderate consequence to population. There are no confirmed cases of algal toxicity in eWHIS.	1	L	M	**L**	**M**
		2	L	M	**L**	**M**
		3	L	M	**L**	**M**
		4	L	M	**L**	**M**
Heavy metal intoxication	**Host range & impact** – Heavy metals (mercury, cadmium, lead, selenium) and chemical contaminants (TBT, DBT) have been found in little penguins from the Shoalwater islands group, including fledglings in WA (Cannell et al. 2016). In addition, heavy metals were also found in prey LPs in Tasmania (Wells, M. pers. comm.), in three prey fish species of LPs near St Kilda (Finger et al. 2017) and in LPs from three Victorian colonies including Phillip Island (Finger et al. 2015) and Free-ranging pelicans, cormorants, and loons are susceptible to lead intoxication, mainly by	1	L	L	**L**	**L**

	ingestion of fishing tackle and ammunition. Fishing related sinkers and coins may also be ingested by penguins (Stidworthy and Dent 2018). Disease and/or pathology are frequently absent but may contribute to hepatopathy and renal toxicity in penguins (Stidworthy and Dent 2018). High levels of mercury and cadmium have been associated with proximal renal tubular epithelial necrosis in Manx shearwaters and northern fulmars (Stidworthy and Dent 2018). As meso-predators in marine ecosystems, penguins are at an elevated risk of bioaccumulation and biomagnification of mercury (Hg). In seabirds, exposure to Hg has been linked to increased oxidative stress, altered gene expression, and decreases in reproductive fitness, immunocompetence, and neurotransmitter functioning (Ackerman et al. 2016). Clinical signs if ingested may include green urates and inability to swim. Necropsy lesions can be absent or include pectoral atrophy, fat depletion, oesophageal/proventricular impaction, ventricular erosions, gallbladder distension, and lead objects in gastric content. **Consequence –**. Exposure primarily through ingestion of metal containing products (e.g. fishing products. However, likelihood of exposure low in protected populations with controlled access and restrictions on land use. Likely to be few cases in wild LPs but may build up over time, overall low population consequence. Based on eWHIS, heavy metal toxicity (elevated zinc, mercury and selenium) was confirmed in 3 x LPs found dead at Kwinana Beach, WA; it was considered a possible differential in an LP at Fairhaven, Vic.					
Industrial chemical intoxication - oiling	**Host range & impact** – Oil pollution through shipwrecks and oil spills is possibly the major anthropogenic-induced cause of death among penguins worldwide (García-Borboroglu et al. 2008). Oil pollution contributes to deaths of LPs at sea (Dann 1992). In January 2000, an oil spill occurred in the Phillip Island (Millowl) surrounds. Most LPs released within 4-6 weeks. However, a few failed to subsequently moult and were kept in care. These individuals developed secondary complications including pododermatitis and then acute nephrosis (PINP W 140700). In 1995, The Iron Barron was responsible for a 325 ton oil spill in northern Tasmania, causing the death of an estimated 10,000-20,000 LPs (Goldsworthy et al. 2000). Oiling impacts penguins causing direct mortality of adults, juveniles and chicks and long-term physiological damage. Oiled feathers lose their waterproofing and insulating properties, causing the birds to lose heat rapidly (Ropert-Coudert et al. 2019). Oil may also cause chemical burns to the skin and eyes, and when ingested (e.g. during preening) it can cause gastrointestinal ulcers and bleeding (Crawford et al. 2000). Commonly reported clinical signs of intoxication include loss of waterproofing and insulation, gastroenteritis, teratogenicity/embryotoxicity, immunosuppression, endocrine, renal, hepatic and haematological abnormalities, impaired osmoregulation (Stidworthy and Dent 2018). On necropsy exam multifocal hepatic necrosis, embolic bacterial myositis, proventricular erosion and ulceration, ulcerative enteritis, adrenal cortical necrosis and lymphoid depletion in the Bursa of Fabricius were reported in LPs (Norman 2005). Acute asphyxiation secondary to oiling also reported (PINP MZ 210791). An oil spill off the coast of Phillip Island (Millowl) could significantly impact the population. At certain times of year, the entire colony comes ashore at night and could pass through a slick in doing so. Inshore and beached slicks will also impact those coming ashore (Dann 1996). Potential risk for exposure in the wild. Likelihood low in protected populations with controlled access and restrictions on land use.	1	L	H	**L**	**H**

	Consequence Likely to be sporadic in occurrence however high population consequence with both acute and chronic disease presentations. Could lead to significant morbidity and mortality of the population.					
Persistent organic pollutants (POPs) and other emerging contaminants	**Host range & impact** – . Persistent Organic Pollutants (POPs) are chemicals that are toxic, persist in the environment and animals, bioaccumulate through the food chain, and pose a risk of causing adverse effects to animal and human health and the environment even at low concentrations. POPs have been linked to adverse effects on human health such as cancer, damage to the nervous system, reproductive disorders and disruption of the immune system (Lallas EP 2001, UNEP 2009). Due to their potential for long range movement (circulating via the atmosphere, and other pathways), concerns regarding persistence, bioaccumulation and toxicity. Lewis et al. (2020) reported baseline information on POPs in migratory short-tailed shearwaters from Fisher Island, Tasmania, and LPs from Phillip Island (Millowl), Victoria. Levels of polychlorinated biphenyls (PCBs), organochlorine pesticides (OCPs) and brominated flame retardants (BFRs) were determined from blood samples, with total contamination ranging 7.6-47.7 ng/g ww for short-tailed shearwaters and 0.12-46.9 ng/g ww for LPs. In both species contamination followed the same pattern where PCBs>OCPs>BFRs. BFR levels included the presence of the novel flame retardant hexabromobenzene (HBB). Penguins have a high lipid content and slow metabolism, therefore most species have a very slow process of pollutant detoxification (Jara-Carrasco et al. 2017). Despite small fractions eliminated by guano (Falkowska and Reindl, 2015), POPs can persist and accumulate in penguins, with enhanced circulation of pollutants when animals are fasting and mobilize fat reserves. Differing levels of body fat influence tissue levels of lipophilic contaminants found in Sydney and Phillip Island (Millowl) LPs (Norman 2005). Dichlorodiphenyltrichloroethylene (DDE), polychlorinated biphenyl (PCB), hexachlorobenzene (HCB) and heptachlorepoxide redistribute to liver and brain as body fat is depleted. Concentrations at PI and Sydney interestingly were higher than those reported in subantarctic and antarctic species. Redistribution of organochlorines due to starvation noted in reports of other seabirds and Adelie penguins (Gibbs 1995) **Consequence –** Likely to be a few cases in LPs owing to proximity to Port Phillip Bay, however overall low population consequence but may be additive over time and increasing effect with food shortages. Surrounding agricultural land is likely to result in historical and continuing runoff of agricultural chemicals as well as heavy metals and trace elements from soil work, quarries and mining. Port Phillip Bay and Corio Bay with associated port, industry and recreational uses are assumed to be contributing to pollutants.	1	M	L	**L**	**M**
Per- and Polyfluoroalkyl substances (PFAS)	**Host range & impact** – Per- and Polyfluoroalkyl substances (PFAS) are increasingly detected in wildlife and present unknown health risks. PFAS are of potential concern owing to their environmental persistence and ability to disperse over great distances. There is a growing body of evidence describing PFAS in seabird species, however knowledge from temperate Southern Hemisphere regions is lacking other than in Tasmanian LPs where 14 PFAS in serum at 8 of 9 sites tested, and in scat-contaminated nesting soil at 16 out of 17 sites tested (Wells et al. 2024). Perfluorooctanesulfonic acid (PFOS) and perfluorohexanesulfonic acid (PFHxS) were most commonly detected (Wells et al. 2024). Recently published PFAS concentrations in Australian fur seals and sea lions, breeding and foraging in close proximity to LPs detected concentrations	1	L	L	**M**	**M**

	comparable to northern hemisphere counterparts (Taylor et al. 2021), and concentrations detected in stranded dolphins from south-east Australia were the highest globally reported (Foord et al. 2024). PFAS were widely detected in little penguins around Tasmania. Urban intensity represented by total road length (km) within 1 km of each site, was positively associated with PFAS concentrations in soils and plasma of little penguins (Wells et al. 2024). PFOS in female LPs negatively associated with haematocrit and plasma proteins, while PFOS in male LPs positively associated with erythrocyte nuclear abnormalities (Wells et al. 2024). Biological consequences from PFAS exposure in seabirds include disruption in thyroid function, increased oxidative stress, effects on telomere length, reduction in body condition and demographic responses which are typically initial symptoms in response to a stressor (Eckbo et al. 2019, Sebastiano et al. 2020). **Consequence –** LPs on Phillip Island (Millowl) likely to have been exposed given findings from Tasmanian study and levels detected in closely habiting sea lions however, further research required to understand trophic bioaccumulation pathways, impact on reproduction and to understand sublethal consequences from exposure. Wells et al. (2024) found evidence of PFAS eggshells (100% of shells tested). Likely to become of increasing importance in the future.					
ENVIRONMENTAL						
Antimicrobial resistance genes	**Host range & impact** – AMR is globally distributed, and a significant, emerging health issue in humans and animals. It has been detected in many wildlife species. Lundbäck et al. (2021) described the presence of class 1 Integrons in the microbiome of LPs from captive (over 45%) and wild (3%) LPs in Australia. This included four (out of 228 tested) positive penguins from Phillips Island (Millowl). While this is likely to have little impact on LPs populations themselves, LPs are likely to play a very small role in contributing to the spread of AMR in the environment, but may act as a sentinel. **Consequence** - The impacts of AMR on the treatment of infections in humans and livestock are substantial (World Health Organisation 2021), with estimates of up 700,000 human deaths per year due to AMR in a report from 2016 (O'Neill 2016).	3,4	VL	H	**M**	**L**
Botulism - Botulinum toxin from *Clostridium botulinum*	**Host range & impact** – With a global distribution, avian botulism can occur in any bird species, but is most frequently seen in waterbirds (ducks, geese, swans, ibis, egrets and pelicans) (WHA 2019). It is commonly reported in wild birds around wetlands in Australia and the Summerland Peninsula on Phillip Island (Millowl) is in close proximity to a Ramsar wetland. It is not a significant problem in seabirds in Australia (Vogelnest 2004) and has not been reported in penguins worldwide. Piscivores (common loons, red-throated loons, and horned grebes) are more typically affected by type E botulism where ingestion of fish is implicated, rather than the carcass-maggot cycle associated with type C botulism in waterfowl (Stidworthy and Dent 2018). However, this has only been associated with outbreaks in the USA and France (Thomas et al. 2007). Optimal environmental conditions for spore germination & bacterial growth, suitable material or substrates to provide energy for bacterial replication, and a means of toxin transfer to birds is required to set up the cycle of infection (Friend and Franson 1999). The toxin will then bioaccumulate in invertebrates & decaying carcasses which may then be ingested by other species. Botulinum toxins inhibit neurotransmission by blocking secretion of acetylcholine from peripheral cholinergic nerve terminals in the motor and autonomic nervous systems. Diagnosis is	1	VL	L	**L**	**L**

	based on history, clinical signs, absence of other causes of paresis/paralysis on gross, and histologic examination (e.g., trauma, inflammation), and laboratory confirmation of botulinum toxin in blood or tissue. Most of the reported cases occur from November to April, with fewer cases in the cooler months (Grillo et al. 2013). Often the first sign of an outbreak is a sudden increase in the number of dead birds on the edge of a water body. Typical clinical signs include an ascending progressive flaccid paralysis including paralysis of their legs, as the case progresses birds will no longer be able to hold up their head, hence the name, 'limberneck,' and the third eyelid protrudes across the eye. The majority of birds that develop botulism will die if not treated (WHA 2019). **Consequence –**disease not reported in LPs if infected likely to be sporadic therefore of low consequence to population.					
Environmental stressors due to climate change - sea level rise - decreased rainfall and humidity - increased fire risk - extreme climate events - ocean acidification - vector-borne disease	**Host range & impact –** Penguins appear to respond to changing environmental conditions in the short term through modifications in breeding parameters and in the long term by altering their distribution and abundance (Forcada and Trathan 2009). Changing environmental conditions place additional stressors on species and likely affect immunocompetence (Ropert-Coudert et al. 2019) and in doing so increase susceptibility to disease. Like many other species, penguin life cycles are affected by climate change directly through El Nino, increased frequency of heat waves and storms leading to egg and chick loss (Boersma and Rebstock 2014), or indirectly through increased frequency of bushfires (Chambers et al. 2010), and climate-driven changes in prey abundance and distribution (Trathan et al. 2006, Vargas et al. 2006). Climate variability and change affects seabirds, both directly (e.g. heat-related mortality) and indirectly (e.g. through the impact of climate on food webs) (Ainley et al. 2010). A report on climate change modelled on data from LPs at Phillip Island (Millowl) (Dann and Chambers 2009) predicted little direct impact of decreased rainfall and humidity. However, fire risk may increase leading to adult mortality and habitat loss and the fire season may be extended. Extreme climate events may also slightly reduce adult and chick survival. Warmer oceans were likely reported to improve recruitment into the breeding population (longer breeding season and greater availability and quality of feed predicted) but the effect on adult survival were not clear. Based on a projected tripling of days with temperatures >35°C by 2070 (the mid-range estimate for coastal regions of Western Port), heat stress in the LP is likely to increase. At temperatures >27°C, the daily energy budgets of penguins is expected to increase in conjunction with increasing temperatures, as the penguins expend energy to maintain core temperatures (Baudinette et al. 1986). Overall, many aspects of LP biology are likely to be affected by climatic change but no net negative effect on population size was projected. Climate change may also alter ecosystem properties for example air temperatures, precipitation, humidity, winds and rainfall, allowing disease-carrying vectors to establish where historically the climate was unsuitable (Tratham et al. 2014). At Penguin Island in WA **i**ncreasing ocean temperature and strong winds have been linked to negative effects on adult foraging and chick survival (Cullen et al. 2009, Cannell et al. 2012, IUCN 2020). Under La Niña conditions, there is typically higher rainfall and more storm activity, while the opposite is true for El Niño conditions (Holbrook et al. 2009). The variability in rainfall across years will influence habitat quality for LPs. Higher rainfall and storm activity may increase erosion of pathways to nest sites, while less rainfall may reduce native vegetation cover ideal for nesting	1	M	M	**L**	**M**

	(Sutton 2022). Marine heatwaves are projected to become more frequent, intense, and persistent in Australia with climate change predictions (Commonwealth of Australia 2020). Following the marine heatwave of 2011 around Penguin Island WA, the spawning and migration patterns of many fish species were impacted for subsequent years (Caputi et al. 2014). Changes in the diet of LPs from Penguin Island were also noted. For the colony on Penguin Island, elevated SST prior to a breeding season has been linked to reduced courtship, delayed egg laying, and a higher occurrence of egg abandonment (Cannell et al. 2012). Following the marine heatwave in 2011, where elevated temperatures persisted above average for several years, overall chick production was low (Cannell 2017, 2018). **Consequence -** overall medium likelihood of occurrence with current climatic predictions and of moderate consequence to the population. Climate change modelling should be updated as new information becomes available to assist with management decision making. However, we should also note that the predictive power of relationships built on past observations (when not only the average climatic conditions are changing but also the frequency of extreme climatic anomalies) may not be a good predictor of a species' future response to climate change (Tratham et al. 2014).					
Habitat loss – terrestrial - Trampling by humans, livestock, introduced sp. - Invasive weeds - Housing developments - Cape Baron geese - Coastal erosion - Dune cliffing - Sea level rise	**Host range & impact –** In Australia significant problem at mainland sites or on island colonies that are settled. Trampling of burrows by humans and livestock with secondary erosion contributes to habitat loss and lack of availability of suitable sites for breeding (Dann 1996). Introduced species such as rabbits can also destroy surrounding vegetation leading to erosion and may make burrows more prone to collapse. At Phillip Island (Millowl) Cape Baron geese, coastal erosion and dune cliffing are currently having significant impact (L Renwick pers comm Nov 2023). Invasive plants and weeds can also significantly destroy suitable habitat; for example Kikuyu grasses, Coprosoma, buffalo grass and Cape Ivy reduce available areas for nesting (Fortescue 1995). In addition, invasive species (e.g. sea wheat grass, Marram grass) can change landing site accessibility for penguins by causing cliffing (D. Sutherland, pers. comm). Dann and Chambers (2013) conducted a systematic review on climate predictions for LPs at Phillip Island (Millowl), this included a small loss of penguin breeding habitat due to sea level rise. The sea level has risen at a rate of 1.4 mm/year across Australia's coastlines between 1966-2009 (CSIRO and Bureau of Meteorology 2022). This is of concern as rising sea levels can threaten LP burrowing and nesting sites through inundation or erosion of pathways to nests, however, the relative impact to the population was thought to be low. **Consequence** – Indirect threats such as habitat loss through trampling by humans, livestock, introduced sp, invasive weeds and housing developments impact the distribution and abundance of penguins. Ongoing threat with medium consequences to the population although is being actively managed by PINP	1	M	M	**L**	**M**
Habitat loss – marine - Primarily from land based activities - Pollutants - Toxins - Fishing	**Host range & impact –** LPs spend 80% of their lives at sea. The marine ecosystem is dynamic and multiple threats to habitat exist. Land-based activities modify the marine environment through runoff of sediments, nutrients, toxins, and pollutants, and even alter the flow of currents and tides (Tratham et al. 2014). Changes in offshore ecosystems including the extraction of mineral resources, pollution from vessel traffic, and the construction of infrastructure for oil development or offshore wind farms (Halpern et al. 2008) have been reported to impact African penguins off the Nigerian coast (Trathan et al. 2014). Coastal and inshore mining operation along Namibia's	1	M	M	**M**	**M**

- Ocean vessel traffic - Construction for oil development - ocean acidification - climate change	southern coast threaten foraging habitats of African penguins through the large-scale release of sediment into coastal waters. Water turbidity can reduce prey availability and is likely to affect foraging behaviour. Sediment movement also contributes to the formation of temporary land bridges to some islands, which allows access by land predators (Kemper 2006). Dann and Chambers (2013) noted that ocean acidification has the potential to be a highly significant negative influence on food availability with climate change predictions, but present assessments are speculative. Greenhouse gas emissions are causing the oceans to become acidic, which threatens the entire ocean food web from plankton to penguins. The oceans absorb a large portion of carbon dioxide produced by our industrial society. As ocean waters have absorbed this excess carbon dioxide, the acidity of the ocean has increased by 30% and carbonate ions have become less available. Carbonate ions are used by calcifying creatures such as plankton, corals, and clams to build their shells. As ocean acidification reduces carbonate ion availability, these creatures will become increasingly unable to build new shells and existing shells will begin to dissolve, which leaves these animals with no way to survive, and therefore, affects the penguins' food supply. **Consequence** – Indirect threats such as predicted ocean acidification may impact the distribution and abundance of penguins through altering available food sources. Multiple threats which LPs are currently exposed to, and which are likely to escalate owing to human population growth. Ongoing threat with medium consequences to the population					
Predation – terrestrial - cats - foxes - dogs - rats - raven predation on penguin eggs - Tasmanian devils	**Host range & impact -** Predation by feral foxes is thought to be a major factor in the decline of Sheniciformes, with their preference for spending most of the time on or near the ground, and nesting on the ground, making them particularly susceptible to predation. Feral cats may also impact on populations, though little penguins make up only a small proportion of their diet on Phillip Island (Millowl) (Rendall et al. 2022; Kirkwood et al. 2005). Foxes and dogs and possibly ferrets and stoats in New Zealand have been implicated in a number of colony declines (Dann 1996), and dog attack has been linked to an increased probability of colony decline or extinction in Tasmania (Blamey et al. 2023). Foxes are capable of killing 40-50 penguins in one night. In the past several hundred LPs were killed by foxes on Phillip Island (Millowl) (Dann 1996) and while they are no longer present on the island, they still remain an ongoing threat. Rats have been implicated in the demise of LPs on Granite Island SA (Bool et al. 2007). Egg predation by ravens (*Corvus mellori*) is an emerging threat to penguin breeding success on Phillip Island (Millowl) with over 60% of eggs being taken in some areas (Ekanayake et al. 2015). In addition, Tasmanian devils, translocated to Maria Island where they did not previously occur as part of a conservation program for Tasmanian Devil Facial Tumour Disease, were responsible for the decline of the LP colony on that island (Scoleri et al. 2020). **Consequence** – May sustain low level injury or immediate death; may survive the initial attack but later die from complications of bite wounds or systemic infection. Overall potential for high consequences to population. However, active management to control predators at PINP (have eradicated foxes from the island) and actively managing feral cats and dog access. However, the penguin colonies are now expanding to beaches on the island where dogs are allowed, creating potential future management needs.	1	H	H	**L**	**H**

	Trauma is a consistent finding in LPs submitted to eWHIS, especially from Tasmania; this includes 24 from a mass mortality event at Shoalwater, WA; 1 x Kingston Beach, Tas; 3 x Victor Harbor, SA; 17 x Burnie, Tas; 37 x Bicheno, Tas; 15 x Ulverstone, Tas; 64 x Low Head, Tas. These attacks have mostly been linked to dogs.					
Predation – marine - Australian sea lions in western Victoria (very low numbers) - Long-nosed fur seals (recovering in Victoria but population currently at low numbers)	**Host range & impact -** sea-lions have been implicated in the demise of LPs on Granite Island SA (Bool et al. 2007). Predation of seabirds occurs amongst many seal species (Antarctic fur seals (Visser et al. 2008, leopard seals, (Ainley et al. 2005); South American sea lions, (Rey et al. 2012); New Zealand sea lions, (Morrison et al. 2016), and long-nosed fur seals (Page et al. 2005). In South Australia, penguin remains have been detected in 0-40% of long-nosed fur seal scats (Page et al. 2005). Predation in Victoria by long-nosed fur seals varies but can be high and most commonly occurs where seal and seabird colonies are in close proximity (Reinhold et al. 2022). **Consequence** - May sustain low level injury or immediate death; may survive the initial attack but later die from complications of bite wounds or systemic infection. Overall potential for high consequences to population.					
Reduction in food supplies	**Host range & impact –** Reduction in food supply either directly through over-fishing, competition with fishing, declining water quality or other factors reducing fish stocks. Starvation has been identified as a major factor determining the survival of young and adult penguins (Norman et al. 1992) and both pilchards and anchovies are taken at Port Phillip Bay for commercial fishing which may impact the Phillip Island (Millowl) population (Dann 1996). Fishing interactions include both direct effects such as incidental bycatch in fishing gear, as well as indirect effects such as competition for prey resources, habitat modification by fishing gear, or interaction with fisheries discards (Ropert-Coudert et al. 2019). A large disease outbreak in pilchard in 1995-96 resulted in significant population impacts in LPs in Victoria (Dann et al. 2000). Overfishing has been implicated in the collapse of African penguin populations (Dann 1996). When prey abundance is low in foraging grounds surrounding Penguin Island, WA LPs need to travel greater distances and expend more energy to find prey, which can be detrimental for raising chicks. Little penguins can also experience delayed breeding cycles, delayed growth, and delayed breeding success when food resources are low (Cannell et al. 2016). **Consequence** –sporadic in likelihood however may become more likely with predicted alterations in climate and overfishing, of medium consequence to population as may make population more susceptible to other threats. There are processes that drive food availability that are essentially 'natural' and those that are anthropogenically-altered. There is little that can be done to manage natural cycles and processes governing fish die-offs and marine productivity in general, however if there is an anthropogenic element to the origin, frequency or extent of these events, then some management may be feasible (Dann 2016). PINP are involved in Marine Spatial Planning to reduce this risk. Emaciation is a key finding in the eWHIS records (almost 20 of the records, including multiple animals per record), but can reflect multiple aetiologies including food shortages and disease processes.	1	L	M	**L**	**M**
Starvation	**Host range & impact –** Significant issue in free-ranging penguins, particularly chicks and juveniles, and can result in mass mortality events in Australia and globally (Stidworthy and Dent 2018). The widespread and large-scale mortality of pilchards in Southern Australia in 1995 had a significant	1	L	M	**L**	**M**

	effect on the numbers of penguins on Phillip Island (Millowl) (Dann 1996). Starvation is typically multifactorial and reported in LP in Australia (PINP DPI 230707). LPs can experience delayed breeding cycles, delayed growth, delayed breeding success, but most importantly, death due to starvation when food resources are low (Cannell et al. 2016). Egg desertion on Penguin Island in WA was associated with hunger as birds were thin (Wienecke, Wooler and Klomp 1995). Starvation was found to be the second highest cause of mortality in 163 deceased LPs collected in the Perth metropolitan coastal areas between 2003-2012 (Cannell et al. 2016). Most starved individuals were found in spring and summer. The increased rate of deaths of LPs from starvation following the marine heatwave in 2011 and subsequent La Niña years was attributed to higher sea surface temperatures reducing the abundance of coastal baitfish species in the Perth metropolitan region (Cannell et al. 2019). **Consequence** - Most affected birds are emaciated juveniles with reduced bodyweight compared to birds surviving migration, pectoral muscle atrophy, an empty stomach, complete fat exhaustion with serous atrophy, and multi-organ atrophy. In sea birds, food shortages and extreme weather events often precede such large-scale die-offs (Dann et al. 2000; Frederiksen et al. 2008, Rensburg 2010). Individuals may sustain low level injury or immediate death; may survive but later die from complications or systemic infection. Overall potential for medium consequences to population likely presenting as 'wrecks' washed up on shoreline. Emaciation is a key finding in the eWHIS records (almost 20 of the records, including multiple animals per record), but can reflect multiple aetiologies including food shortages and disease processes.					
Storm damage	**Host range & impact –** Storms and waves can cause erosion of sand banks and dunes and impact the ability of LPs to reach their nests (Cannell 2001). Storms have been suggested as a cause of death for LPS and eggs may be lost due to heavy rain (Reilly and Cullen 1981). More extreme climate events are predicted with climate change in certain regions (Dann and Chambers 2013) and this may impact chick growth rate. Wienecke et al. (1995) reported a decline in growth rate after parents were prevented from accessing nests to feed chicks following a significant storm event at Penguin Island. In addition, storms have been shown to impact on LP feeding efficiency and success, for example due to disturbed water column thermoclines and prey dispersal (Barreau et al. 2021). **Consequence –** Severe storms are likely to only be sporadic however may increase in frequency with climate change predictions. Likely to be of moderate consequence to the population if storms are severe however overall low population consequence	1	L	L	**L**	**L**
Thermal stress – terrestrial	**Host range & impact -** LPs can experience thermal stress in temperatures ≥ 30°C and hyperthermia at ≥ 35°C, which has caused mortalities of LPs from the Shoalwater islands WA particularly during the moult stage (Cannell et al. 2016). During daylight hours, nest temperatures exceed ambient temperatures, causing thermal stress in LPs on warmer days. Artificial nest boxes have higher maximum temperatures during the day, exceed upper thermoneutral limits more often & have prolonged extreme temperatures more so than natural nests (Sutton 2022). Ambient temperatures can create potentially lethal temperatures inside artificial nest boxes, however, manipulation of artificial nest boxes on Penguin Island found improvements could be made to boxes to reduce this threat to LPs (Clitheroe 2021). Elevated	1	M	L	**L**	**M**

	temperatures during the day can also delay the onset of breeding for some LPs on Penguin Island WA including, pre-laying nest attendance and courtship (Wienecke 1993). There was a 2-11% mortality in some areas of PI in a 2019 heat event (L Renwick, pers. comm). Similarly, 5% mortality has been reported in Magellanic penguins during a heat event in Argentina (Holt and Boersma 2022). **Consequence –**Thermal stress is likely to only be sporadic however, may increase in frequency with climate change predictions. Likely to be of moderate consequence to the population if prolonged heat stress, however, overall of low population consequence. eWHIS reports hyperthermia in 39 LPs from Shoalwater, WA.					
Thermal stress – marine - warmer oceans - SST and marine heatwaves	**Host range & impact –**Ocean warming in south-eastern Australia is causing pole-ward shifts in species ranges, including penguins and their prey. The fish penguins feed on, stay in cooler, deeper waters as surface temperatures rise. Penguins and petrels respond to these changes by foraging further away and diving deeper to find food. Starvation is a risk for themselves and their chicks if food is in short supply (Boresma and Rebstock 2014). Warming ocean waters can also change the distribution of water currents and lead to changes in prey recruitment and growth, making finding food more difficult for many of the world's penguin species (Ropert-Coudert et al. 2019). Increasing SST due to strengthening of the east Australian current may be responsible for the collapse of the LP colony at Gabo Island (D Sutherland, pers. comm). A recent study showed declines of 80% in a population of LPs as a consequence of a marine heat wave in 2011 (Cannell et al. 2023). In addition, the Galápagos penguin has proven to be extremely vulnerable to starvation during El Niño events. Adults are forced to abandon their eggs and chicks to search for food, leaving their chicks to starve. Regional instances of episodic warming of the marine environment have also been shown to cause mass starvation of penguins in Australia (Cannell et al. 2012) These changes can also cause a shift in abundance towards species tolerant of warmer waters (Last et al. 2011). Additional effects of rising ocean water temperatures include the increased occurrence of harmful algal blooms. Algae grow rapidly and accumulate into dense patches near the ocean surface, promoting the absorption of solar radiation, exacerbating warming. At Penguin Island in WA increasing ocean temperature and strong winds have been linked to negative effects on adult foraging and chick survival (Cullen et al. 2009, Cannell et al. 2012, IUCN 2020). In 2011, temperature anomalies of 2-4°C above average (marine heatwaves) persisted for months along the WA coastline. Following the marine heatwave of 2011, the spawning and migration patterns of many fish species were impacted for subsequent years (Caputi et al. 2014). Changes in the diet of LPs from Penguin Island were noted following the marine heatwave. For the colony on Penguin Island, elevated SST prior to a breeding season has been linked to reduced courtship, delayed egg laying, and a higher occurrence of egg abandonment (Cannell et al. 2012). **Consequence -** overall medium likelihood of occurrence with current climatic predictions and of moderate consequence to the population. Climate change modelling should be updated as new information becomes available to assist with management decision making. Can have moderate to high consequences, affecting reproduction, diet and population size.	1	M	M	**L**	**M**
Wildfire	**Host range & impact –** Deliberately lit fires are thought to have contributed to demise in LP numbers in Tasmania between 1975-1977 (Dann 1996). Wildfires can potentially decimate	1	H	H	**L**	**H**

	breeding habitat of LP populations. The impacts of fire can also be indirect. A large fire at Marion Bay, Tasmania, resulted in significant increases in Marram grass and coastal wattle, leading to dune-cliff formations and thick vegetation, respectively, which is thought to have prevented LPs from re-colonising this area after it was lost to the fire (Stevenson and Woehler 2007). **Consequence** – Individuals may sustain low level burns or sudden death; or alternatively may survive the initial episode, but later die from complications. Likely will have increased exposure to predation following fire, and reduction in the habitat available for nesting. Overall potential for high consequences to LP population. PINP actively managing wildlife risk through strategic prescribed burns and other pre-suppression work to provide protection and reduce the risk of bushfire impact.					
OTHER						
Cardiac (heart) disease	**Host range & impact** – Atherosclerosis has been reported and prevalence varies within captive penguin collections. In one review a 2% incidence with no associated mortality was noted while in another, 37% of penguins had atherosclerosis, predominantly as an incidental finding. Adélie and emperor penguins were most commonly affected (Stidworthy and Dent 2018). **Consequence –** The prevalence may be influenced by the age of captive populations, species, dietary or climatic differences. Typically diagnosed on histopathology as discrete, sporadic cases, likely within normal population limits, therefore of low population consequence.	1	L	L	**L**	**L**
Entanglement	**Host range & impact -** free-living seabirds can be entrapped and drowned in marine debris and fishing gear, including drift nets, gill nets, used fishing line and baited hooks. Closer to shore, nets catch diving species including cormorants, loons, grebes, and penguins. Potential competition for resources between fisheries and natural predators, such as penguins, is becoming an increasing concern and estimated capture rates by fisheries may be underestimated (Pauly and Zeller 2016), as is bycatch (Crawford et al. 2017). Most drowning cases occur in the absence of underlying disease. Recorded on occasion in LPs (Cannell 2001). In contrast to birds stranding or affected by pollution, drowned birds have well-developed muscles, good nutritional condition (including subcutaneous fat), and their stomachs frequently contain recently ingested fish. The air sacs or distal trachea may contain clear watery fluid. The lungs are often congested and oedematous, exuding white frothy supernatant-rich fluid from cut surfaces (Stidworthy and Dent 2018) **Consequence -** Likely to be a few sporadic cases in wild LPs (though gill nets are still legal in Tasmania (Iutruwita). Currently of low population consequence, however, increasing public awareness and encouraging effective disposal of fishing equipment may be a solution. eWHIS shows a record of 30 LPs found drowned, suspected of entanglement in fishing net, at Altona, Vic.	1	L	L	**L**	**L**
Gastrointestinal foreign bodies	**Host range & impact** – Gastrointestinal foreign bodies occur frequently in captive and free-ranging penguins, and reportedly cause significant morbidity and even death. Ingested objects are often retained in the stomach of curious juveniles and nesting females. These may include nesting and plant material, such as sticks and stones or bristles from cleaning utensils, coins, fence clips, nails, lead pellets, moulted tail feather shafts, enrichment items, and items introduced into enclosures by members of the public. Multiple necropsies (n=50) of LPs performed by University of Melbourne veterinarians, found no macroscopic plastics, other than	1	L	L	**L**	**L**

	one penguin with fishing line and a hook embedded in its stomach. Microplastics have not been significantly investigated. However, plastic ingestion has seldom been recorded in most penguin species (Ryan 2016). This is plausible as they mostly take live prey and pay little attention to floating objects, either a fish carcass or plastic objects, quite unlike petrels and albatross many of which are scavengers (Ropert-Coudert et al. 2019). Clinical signs are dependent on the size of the object and if a partial or complete obstruction. Common clinical signs include dehydration, lethargy, weakness, anorexia, distension, weight loss, regurgitation, vomiting, ill thrift, diarrhoea, scant faeces, and extended moult. Foreign bodies are definitively diagnosed on necropsy. Associated pathology ranges from mild mucosal irritation and ulceration to transmural foreign body penetration/perforation with localized to diffuse coelomitis and sepsis (Stidworthy and Dent 2018) **Consequence -** Likely to be a few cases in wild LPs, however likely to increase with predictions for human plastic consumption. By 2050 we will be producing three times as much plastic as we do today (Jambeck et al. 2015). Currently of low population consequence, however, likely to increase in importance in future.					
Hepatic (liver) disease	**Host range & impact** – Hepatic pathology is seen in multiple avian disease processes. Multiple reports of liver related death in Sphenisciformes. Cases only included when other diagnosis not reached. Little penguin (PINP Gippsland 210421) hepatic abscess Typically diagnosed on histopathology often in conjunction with multiple other disease processes. **Consequence -** Likely to be a few sporadic cases in wild LPs, overall low population consequence.	1	L	L	**L**	**L**
Neoplasia	**Host range & impact** –A few case reports in Spheniciformes exist. A fibrosarcoma was reported on the webbing between toes (LP PINP EV 251193), and lymphoma in LP from Victoria (Reece 1992). Malignant melanomas are reported with the highest frequency, in macaroni, Humboldt, and rockhopper penguins. Predilection sites are the skin of the foot or hock, and subcutis and adjacent muscle near the beak and oral cavity (Stidworthy and Dent 2018). Other reported cases include Uropygial gland squamous cell carcinoma and gastric adenocarcinoma in a Humboldt penguin (Yonemaru et al. 2004). **Consequence** – Although high individual consequence low prevalence – suspect within the normal population limit therefore overall low consequence to population.	1	L	L	**L**	**L**
Pododermatitis (bumblefoot)	**Host range & impact** – Commonly reported in captive penguins, waterbirds, raptors and fowl globally. Predisposing factors in captive penguins include sedentary behaviours, decreased swimming and prolonged standing on abrasive, hard, moist, or faecally contaminated flooring. The primary lesion is ischaemic pressure necrosis of soft tissues of the foot, or an initial puncture wound that compromises perfusion of the dermis and leads to erosion and ulcer formation. A grading system of 1 (mild) - 5 (severe) exists (Oaks 1993). Clinical signs include abnormal gait, increased resting behaviour, and discomfort on foot palpation (Stidworthy and Dent 2018). Unilateral or bilateral swelling and increased redness of one or multiple areas of the footpads followed by epithelial thinning, ulceration, haemorrhage, and crusting or granulation tissue formation can occur (Reiderson et al.1999). Secondary opportunistic bacterial colonization with *Escherichia coli, Proteus mirabilis* and *Staphylococcus, Enterococcus*, *Pseudomonas*, and *Clostridium* species, or invading fungi is common (Stidworthy and Dent 2018). Prevention is paramount, focusing on environmental enrichment, encouragement of swimming, and avoidance of hard, rough, wet, or contaminated surfaces.	1	L	L	**L**	**L**

	Consequence – Primarily a disease of captivity hasn't been reported in free-living penguins. Low likelihood of occurrence in wild population of variable consequence but overall low consequence to wild population					
Tourism	**Host range & impact –** At several breeding colonies throughout their range in southern Australia and New Zealand, LPs attract large numbers of tourists, which are of significant importance to local economies (Dann 1992**).** However, disturbance caused by tourism can adversely affect penguin breeding success (Carpenter et al. 2004). For example, by direct habitat destruction, or indirectly through causing adults to prematurely desert their nests (Bolduc and Guillemette 2003), and by causing chicks to receive fewer meals (Wilson et al. 1991). Visitor guidelines are often based on overt behavioural responses. However, human disturbance can also disrupt vital behaviours. Even without any behavioural reaction, human presence can increase energy demands and compromise the immune system through physiological stress responses (Ellenberg et al. 2013). There is also the possibility of 'rogue traders' taking visitors on private viewings adjacent to the managed ecotourism facility at Phillip Island (Millowl) (L. Renwick pers comm Nov 23) given the LP is such a highly charismatic species and penguins are one of the most iconic taxonomic groups of wild animals (Ropert-Coudert et al. 2019) **Consequence –** remove	1	H	L	**L**	**M**
Trauma	**Host range & impact –** predatory attack is a significant cause of morbidity and mortality in free-ranging penguins and many of the colonial nesting seabirds, particularly ground-nesting or burrowing species are at high risk. Introduction of feral rodents and predators, such as cats or primates, can threaten extinction (Stidworthy and Dent 2018). Road trauma has also been identified as a threat at Phillip Island (Millowl) (Dann 1996). However, road kills have been managed and eliminated with the completion of the buyback of the Summerland estate and the design of carparks to minimise access to vehicles in penguin areas. LPs are killed by cars when crossing coastal roads at night in Portland Victoria, Bruny Island and Lillico beach Tasmania and Oamaru and Wellington in New Zealand (Dann 1996). Boat strike (propeller strike from motorised vessels) has also been reported as a pressure affecting LP populations at Penguin Island WA (Sutton 2022). Little penguins forage in shallow waters and need to surface to breath, making them susceptible to interactions with vessels (Cannell et al. 2020). Disturbance to resting penguins by approaching vessels or vessel noise may also impact upon LPs energy and oxygen reserves and, in turn, affect foraging success and chick survival (Cannell et al. 2020). There have also been instances of sporadic animal cruelty by humans (Mel Wells, pers. comm). **Consequence** - May sustain low level injury or sudden death; may survive the initial episode, but later die from complications. Overall potential for low consequences to LP population as often sporadic and low numbers affected. Managed by PINP protocols to reduce and eliminate exposure to introduced predators.	1	M	L	**L**	**L**

4.2 Hazard exclusion

4.2.1 Commensal agents which have not been reported to cause clinical disease, for example:

- Infection with *Campylobacter jejuni* in penguins tends to be subclinical (Stidworthy and Dent 2018)
- Commensal microflora, studies that provide descriptive data on microbial fauna and parasites but lack data on pathogenicity of these organisms.

4.2.2 Hazards no longer present, for example:

- Penguins used to be taken for crayfish bait during the 1950s and 1960s, decreasing the population on De Witt Island, Tasmania (White 1980)

4.2.3 Hazards which would only be accessible in captivity, for example nutritional disease and drug toxicities, for example:

- Nutritional deficiencies in penguins are not common in captive settings and are usually associated with inadequate husbandry or dietary composition. An example is nutritional metabolic bone disease, which can be a result of poor parental care or incorrect mineral supplementation (Stidworthy and Dent 2018).
- Captive piscivores may suffer from thiamine deficiency if their diet is not supplemented, leading to neurological signs (Ladds 2009).
- Penguins can be susceptible to Vitamin A deficiency in captivity, affecting their periocular glands (Ladds 2009).
- Seabirds fed frozen-thawed fish without appropriate supplementation may suffer from hypovitaminosis E; this has been reported from pelicans, cormorants, and rarely penguins (Ladds 2009).
- Antifungal treatment can result in drug toxicities in penguins, including dose dependent voriconazole toxicity (Hyatt et al. 2015).
- Fenbendazole toxicity has been reported from penguins and American white pelicans. In African penguins, such a toxicity event occurred after feeding them with fish accidentally loaded with 66–77 mg/kg of fenbendazole (Hyatt 2015).

4.3 Detailed risk assessment

4.3.1 Introduction

The process of risk assessment for the highest priority hazards based on stakeholder input is described below. The wording is quoted and adapted from Jakob-Hoff et al. (2014a). Detailed risk assessments with management recommendations were undertaken for hazards rated moderate or high, except for those where extensive management plans for the PINP colony already exist (e.g. predation/trauma).

Table 13. Summary risk assessment table for infectious hazards for the LP DRA

<table>
<tr><td colspan="3">Justification of hazard</td></tr>
<tr><td colspan="3">• ‘What can cause disease in the population of concern?’, ‘How can this happen?’ and ‘What are the potential consequences?’</td></tr>
<tr><td colspan="3">Risk assessment
‘What is the likelihood and consequences of a specified hazard occurring within an identified pathway or event?’</td></tr>
<tr><td>Entry assessment</td><td>Exposure assessment</td><td>Consequence assessment</td></tr>
<tr><td>• (or release assessment) estimates the likelihood of the defined species introducing the hazard into an area.</td><td>• Estimates the likelihood of susceptible animals being exposed to the hazard, becoming infected (infectious agent hazards) and disseminating the hazard</td><td>• Estimates the likely magnitude of potential biological, environmental and economic consequences associated with the entry, establishment or spread of the hazard and the likelihood of their occurrence. Includes consequences for resident population of same and other species and for the wider ecosystem</td></tr>
<tr><td colspan="3">Risk evaluation</td></tr>
<tr><td colspan="3">• Risk estimation – summarises the entry, exposure and consequence assessments to provide an overall measure of risk.</td></tr>
<tr><td colspan="3">Risk management options</td></tr>
<tr><td colspan="3">• ‘What can be done to minimise the likelihood of a hazardous event?’ and ‘What can be done to minimise the consequences once a hazardous event has happened?’</td></tr>
</table>

Table 14. Summary risk assessment table for non-infectious hazards for the LP DRA

<table>
<tr><td colspan="2">Justification of hazard</td></tr>
<tr><td colspan="2"></td></tr>
<tr><td colspan="2">• 'What can cause disease in the population of concern?', 'How can this happen?' and 'What are the potential consequences?'</td></tr>
<tr><td colspan="2">Risk assessment
'What is the likelihood and consequences of a specified hazard occurring within an identified pathway or event?'</td></tr>
<tr><td>Entry and exposure assessment combined</td><td>Consequence assessment</td></tr>
<tr><td>• estimates the likelihood of the defined species introducing the hazard into an area.
• Estimates the likelihood of susceptible animals being exposed to the hazard, becoming infected (infectious agent hazards) and disseminating the hazard</td><td>• Estimates the likely magnitude of potential biological, environmental and economic consequences associated with the entry, establishment or spread of the hazard and the likelihood of their occurrence. Includes consequences for resident population of same and other species and for the wider ecosystem</td></tr>
<tr><td colspan="2">Risk evaluation</td></tr>
<tr><td colspan="2">• Risk estimation – summarises the entry, exposure and consequence assessments to provide an overall measure of risk.</td></tr>
<tr><td colspan="2">Risk management options</td></tr>
<tr><td colspan="2">• 'What can be done to minimise the likelihood of a hazardous event?' and 'What can be done to minimise the consequences once a hazardous event has happened?'</td></tr>
</table>

Noting that for non-infectious hazards for example pollutants entry and exposure assessment is combined as the hazard is often environmentally acquired.

4.4 Detailed risk assessments for moderate and high risk hazards

Please note that in these assessments, some text (especially, but not exclusively, in the 'Justification of hazard' section) has been quoted and/or adapted directly from the sources cited, and information for Justification, Release, Exposure and Consequence, has been taken from Table 12.

4.4.1 Disease risk assessment for High pathogenicity avian Influenza (HPAI).

Justification of hazard -
Disease due to avian flu (AIV) is a result of infection with influenza A viruses. Influenza A viruses are members of the Orthomyxoviridae family. Influenza A viruses are described by variations in their hemagglutinin (HA) protein and neuraminidase (NA) (Webster et al. 1992). They are subsequently divided into low pathogenicity avian influenza viruses (LPAIV), or high pathogenicity avian influenza viruses (HPAI) (Abad et al. 2013). The high pathogenicity avian influenza (HPAI) viruses, which have previously only included H5 and H7 viruses, generally cause significant morbidity and mortality in poultry (Alexander 2000a), but impact variably on other species, ranging from no clinical signs, to high case fatality rates. It is thought that HPAI strains emerge in poultry after introduction of LPAIV precursors into flocks; they may subsequently spill back into wild bird populations (Capua and Alexander 2006). So far, HPAIs have not been detected in Australian wild birds (WHA 2023a); they are a notifiable infection. In Australia, the National Avian Influenza Wild Bird Surveillance Program has been going since 2006 (WHA 2023a). The natural reservoir for all influenza A viruses are the Anseriformes (waterfowl: ducks, swans, geese) and Charadriiformes (gulls, terns and shorebirds) (Olsen et al. 2006). However, the host range for Influenza A viruses (including HPAI H5N1) is vast, including wild and domestic birds and mammals. Infection, illness (and sometimes death) have been reported in species such as humans, pigs, horses, cats, lions, leopards, tigers, dogs, mink, marten, weasels, ferrets, badger, foxes, otters, lynx, racoons, skunks, bears, opossum, coyote, rats, mice and marine mammals (WOAH 2023a, Webster et al. 1992). In a study that looked at six species of penguins, evidence of exposure to avian influenza virus was found in sub-Antarctic rockhopper and macaroni penguins (Abad et al. 2013). Since 2021, the frequency and geographic range of outbreaks overseas has increased. In late 2022, HPAI reached Central and South America, spreading through nine countries within four months (WOAH 2023a). HPAI has now been detected in the brown skua population in South Georgia, the first known cases in the Antarctic region (Oct 23 2023, WOAH 2023b). Worryingly, HPAI viruses in Clade 2.3.4.4b H5 should now be seen as a potential conservation threats, as they have killed thousands of wild birds on nearly all continents other than Australia. It is estimated that at least a thousand endangered African penguins (*Spheniscus demersus*) died as a result of HPAI infection in southern Africa (Roberts et al. 2023). More recently (Jan 2024), 35 Gentoo penguins found dead in the Falkland Islands in the South Atlantic tested positive for H5N1; a few days later, 200 chicks were found dead alongside a small number of adults (Spring and Dickie 2024). HPAI H5N1 was also confirmed in King Penguins on South Georgia in March 2024. Mass mortality events associated with HPAI H5N1 virus strains (in particular Clade 2.3.4.4b viruses) have been reported from a variety of wild bird species overseas (e.g. swans, migratory geese, wood ducks, African penguins and other seabirds) (CMS FAO Co-convened Scientific Task Force on Avian Influenza and Wild Birds 2023, Molini 2019, WHA 2023a). In some wild bird species, HPAI can result in sudden death. After experimental infection, wild bird species have shown similar clinical signs as those observed in poultry, including watery diarrhoea, depression, inappetence, neurological and respiratory signs and death (Stallknecht et al. 2007). In affected African penguins, clinical signs included mucoid ocular discharge, cloudy eyes, apparent blindness, lethargy and an inability to stand, open-mouthed breathing, and neurological signs such as head or whole-body tremors, head tilt, dorsal neck flexion, bilateral nystagmus, and seizures (Roberts et al. 2023). Necropsy of birds that have died of AIV infection may reveal necrosis of the pancreas and liver, pulmonary congestion and oedema, subepicardial haemorrhage, and myocarditis (Kim et al. 2015). HPAI H5N1 is also a potentially zoonotic infection, with some subtypes having caused disease in humans, ranging from mild illness to severe respiratory disease, but also death (WHO 2016).

The highest risk period for an incursion of HPAI viruses into Australia is most likely spring (September to November), due to the return of migratory birds from the northern hemisphere to Australia (WHA 2023a). In relation to LPs, the most likely route of infection was from migrating shearwaters returning from wintering in the northern hemisphere. Faecal-oral transmission is thought to be the predominant means of AIV spread in wild bird populations (Wille et al. 2022; WHA et al. 2023). Airborne transmission may be important in some species, when in close contact (CIDRP 2013). Arnal et al (2015) proposed that the respiratory tract may be a more important route of infection in gulls. Given the presence of infections in marine mammals, however, and the arrival of the virus in the sub-antarctic, incursion through other species or routes should perhaps not be fully excluded as a possibility.

Concerns remain about the potential for any avian influenza viruses providing the precursor for a human pandemic strain of influenza and the extreme social and economic consequences that can cause.

Risk assessment

Release assessment	Exposure assessment	Consequence assessment
Known to infect African, King and Gentoo penguins, not reported in LPs but not currently present in Australia or New Zealand, so exposure unlikely to have occurred. The risk of introduction of HPAI virus to Australia was previously assessed as low (Wille et al 2019, WHA 2023a), however, recent assessment indicates that with the emergence of the new strains of HPAI virus, the likelihood of introduction to Australia via migratory birds such as short-tailed shearwaters has increased. The short-tailed shearwater is a trans-hemispheric migrant to the northern Pacific particularly the Sea of Japan and Bering Sea (Scheelings 2022). LPs are in close proximity when they assemble in groups on land and offshore upon departing and returning from a foraging trip and are in direct and indirect contact with shearwaters and other local sympatric species at nesting sites. Overall, there is a low likelihood of HPAI being present in LPs at Phillip Island (Millowl) as there has been no evidence on necropsy exam of prior records, or reports of disease through a	LPs forage in water, mate, and rear offspring on land; if infected, they could be exposed to HPAI through inhalation, ingestion, direct inoculation into the eye and nares and vertical transmission. The likelihood of exposure to other avian spp. is probably medium at nesting sites. Other reported sympatric avian species include the short-tailed shearwater, pacific gull, kelp gull, silver gull, crested tern, Cape Baron goose, Purple swamphen, Sooty oystercatcher, little raven and the Hooded plover. If exposed, it is likely that the individual will disseminate HPAI through faeco-oral transmission or aerosols with transmission likely to be accelerated where birds aggregate. Given the wide host range, all avian spp. may be susceptible; likewise, seals which live in close proximity, may also become infected, potentially leading to worse consequences (e.g. morbidity and mortality in seal populations with and without spread to other species)	The consequences are hard to predict, but based on overseas experience, could be devastating for LPs, wild birds, as well as poultry, and potentially also for some marine mammals. Currently low likelihood of exposure exists from contact between LPs and infected migratory seabirds and shorebirds at Phillip Island (Millowl) as there is no current evidence of infection in Australia. There is a medium likelihood of carriage and transmission given species susceptibility and not in close proximity to poultry production facilities (except for one free-range facility!). HPAI is not present in Australia, therefore there is currently very low disease risk for humans contracting the zoonotic disease from LPs. The disease causes heavy losses for small scale poultry keepers as well as the poultry industry. Disease control operations for poultry involve slaughter and eradication of susceptible birds as well as infected individuals. The disease has great impacts on local and national economies both in terms of costs of disease control operations but also lost revenue from trade restrictions. Most illness and deaths associated with AIV infection in humans occurred after close contact with infected poultry or with objects contaminated by their faeces (WHA 2018) which will not occur if strict biosecurity is undertaken, and access of domestic fowl to penguins is minimised should disease incursion with HPAI occur in Victoria. There is a medium likelihood of exposure through females and males raising offspring, and through grouped movements from the ocean to land. If exposed, infection could result in mild to severe disease including death. Stressors and co-infections may also play a role in promoting overt disease.

national surveillance programme commencing in 2006. However, when present it could be acquired through exposure to migratory seabirds and shorebirds eg shearwaters at nesting sites. If infection ensues there is a medium likelihood of developing disease.		

Risk evaluation

Preventative measures should be employed to reduce the risk of HPAI virus introduction and further spread.

Risk management options (largely cited from Roberts et al. 2023 and Roberts et al. 2024)

Management of HPAI outbreaks in poultry is subject to international and national government regulations and includes quarantining of infected farms, culling of poultry, and vaccination in some countries (Roberts et al. 2024). In parts of Asia, vaccination of birds against HPAI is routine, with countries such as Hong Kong requiring all poultry farmers to vaccinate their birds (Mills 2023). Options available to manage the disease are limited. Historically, vaccination of poultry against HPAI has been restricted to a few countries, and vaccination of wild species, including Spheniscus spp., has been performed only in zoos, and one trial in captive African penguins Roberts et al. (2024). Roberts et al. (2023) provides an excellent review of the descriptive epidemiology of and response to the HPAI (H5N8) epidemic in South African coastal seabirds, in 2018. In this case, actions were limited to removing carcasses and sick birds as sources of the virus, where possible, and limiting the additional mechanical spread of the virus and disturbance caused by human activities. The public was requested via media releases to avoid handling dead bird carcasses, especially if they had contact with domestic birds. At the seabird colonies, conservation authorities distributed information to staff on the disease, with instructions for biosecurity and managing sick birds and carcasses and for record-keeping. Protective clothing, including gloves as a minimum, rubber boots, disposable aprons, and face masks for added protection, was to be worn in the colonies, especially when handling sick birds and carcasses. Disinfectant was distributed for application to equipment, clothing, footwear, and vehicles. At the two mainland penguin colonies, additional measures were required to manage visitors, including footbaths and confining access to the raised boardwalks. Notices were also displayed to inform guests of the situation and potential risk to domestic birds. Over the peak outbreak period, the release of African penguins and other seabirds from rehabilitation centres was discontinued at the Simon's Town African penguin colony.

In Australia, management of this notifiable disease is tightly controlled owing to the potential economic, political, animal welfare, and zoonotic impact. It is strongly recommended that Wildlife Health Australia's Risk mitigation Toolbox for wildlife managers is closely referred to plan appropriate responses https://wildlifehealthaustralia.com.au/Portals/0/Incidents/WHA_HPAI_Risk_mitigation_toolbox.pdf (WHA 2023d)

The PINP formulated an Avian influenza Response Plan in 2023 to manage and mitigate this hazard (Renwick 2023). This is an excellent document detailing planned biosecurity responses to risk scenarios based on lack of presence in Australia (low risk), presence in Australia (medium risk) or presence in Victoria (high risk). The document outlines appropriate training in biosecurity for PINP staff, monitoring for clinical signs and mortality and logging these incidences, appropriate PPE, access restrictions to wildlife for PINP staff and public with increasing risk, public communication of disease risks, carcass removal and establishment of isolation facilities.

Diagnosis

If there is a high index of clinical suspicion, contact the relevant health authority eg WHA state co-ordinator or Emergency Animal Disease Hotline on 1800 675 888 and await further instruction from govt authorities in regards to sampling (WHA 2023b, 2023c)

Note information such as the date/time/location, HPAI signs detected, approximate number and species of birds affected, and photographs and videos as possible. Report this information as soon as possible to the Manager of PINP

Do not handle dead or sick birds unless you are specifically permitted to do so.

If approval is given to handle suspect birds (dead or alive) appropriate PPE consisting of disposable gloves, overalls, goggles and an N95 facemask should be worn.

All boots, exterior clothing, and equipment should be cleaned and then disinfected with a broad spectrum disinfectant such as 70% ethanol, Virkon S, F10, soap + 10% bleach solution, or 0.1% iodine solution (please note different suitability for different disinfectants for various materials, e.g. ethanol and iodine are not suitable for clothing).

HPAI should be considered as a differential diagnosis in the following scenarios for wild birds (WHA 2023d):

- Small groups or clusters (5 or more; temporal or spatial clusters) of sick or dead wild birds of any species.
- Individual or sick or dead wild birds: (5 or more) seabirds, waterbirds, shorebirds or birds of prey or any other bird species with signs of avian influenza infection as outlined below.

Infected live birds may show a wide range of clinical signs, including:

- Neurological signs (ataxia, paralysis, seizures, tremors, abnormal posture)
- Respiratory signs (conjunctivitis, increased nasal secretions, oedema of the head, dyspnoea)
- Gastrointestinal signs (diarrhoea)
- Sudden death

Some species may be asymptomatic or show only very mild clinical signs. In some cases, birds may die suddenly without displaying any clinical signs (WHA 2023c)

A primary diagnosis of avian influenza is usually made via qPCR testing of oropharyngeal and cloacal swabs. Plain sterile swabs are used to collect samples individually from the cloaca and oropharynx and are then placed in tubes containing viral transport media. In carcasses a post-mortem examination should only be performed if requisite PPE is present, alternatively submit the whole carcass to the laboratory in at least a double layer of plastic bags or sealed, waterproof box. On necropsy, brain swabs from dead birds are also useful for PCR testing (Roberts et al. 2023), especially if neurological signs were observed; otherwise, also include at least a lung swab and spleen, liver and kidney if possible. All samples and carcasses must be stored at 4°C prior to submission (WHA 2023c). Enzyme-linked immunosorbent assays (ELISA) or haemagglutination inhibition (HI) and neuraminidase inhibition (NI) assays (Abad et al. 2013) may also be undertaken for evidence of antibodies and previous exposure but are not useful in clinical diagnosis.

Potentially HPAI-virus positive sample (including cloacal and pharyngeal swabs) tubes should be individually labelled. The outside of the sample container should be disinfected, clearly marked as samples for HPAI analysis, and stored separately from other samples. Samples should never be stored in areas containing food.

Ensure proper labelling in adherence to the testing laboratory requirements and/or federal regulation if shipping. Labelling should identify sample contents, date collected, and responsible contact person; if possible, add sample or bird ID linked to additional records, which should at least refer to the location where the bird was first found, the date

and species.

PPE and other material used in the collection of samples should be combined in a sealable bag and disposed of in sanitary waste. Any PPE used in the collection of presumed HPAI-positive samples should be combined in a sealable bag, 20ml of broad spectrum disinfectant should be added before disposal in hazardous waste. Hands should be washed with soap or sanitized with alcohol-based hand sanitizer after any bird-handling

Strict biosecurity protocols are required when undertaking post-mortem examination and sampling LPs to minimise disease transmission. Cleaning and disinfection, including personal hygiene, (e.g., hand washing, cleansing/ disinfection of footwear) are also important in preventing transmission on fomites.

Brain swabs and a pooled tracheal and cloacal sample have been used as diagnostic samples since the HPAI outbreaks in poultry in South Africa in 2017 (Roberts et al. 2023). The brain swab is advantageous as it does not require the carcass to be opened. The sample can be taken quite cleanly, and it saves opening the whole carcass, especially where a full postmortem examination is not possible or desired, and the only aim is to determine the presence or absence of the HPAI virus.

Treatment

Clinically diseased birds pose a risk to other wildlife, livestock and humans for disease transmission and therefore must be managed with strict biosecurity and isolation practices at all times. Treatment was attempted in an endangered African penguin with mild neurological signs with supportive therapy and antivirals comprising 35 mg oseltamivir twice a day, in isolation, for 3 weeks. A PCR test after two weeks of treatment indicated that virus shedding may have ceased, however, the bird was euthanised due to a deteriorating neurological state (Roberts et al. 2023). In the same 2018 event affecting African penguins, the mortality rate of symptomatic birds was high (no birds with neurological disease survived) and the few that survived all had permanent neurological deficits, limiting release (Roberts et al. 2023). Euthanasia was advised for swift terns with suspicious clinical signs, given that treatment had already proven unsuccessful. Private veterinarians, without frequent bird patients, assisted with this. Penguins with mild or moderate signs were accepted for assessment at the rehabilitation centres, given their endangered status.

Birds undergoing treatment, or awaiting the results of disease testing, should be placed in an isolation facility until completion of treatment and follow up testing indicates they are no longer shedding. The incubation period of HPAI is typically 2-8 days, however, it can be up to 14 days. Therefore, while awaiting results all birds should enter a strict 14-day quarantine period. Euthanasia of sick wild birds may take place based on considerations of individual animal welfare, consistent with the relevant animal welfare legislation (WHA 2023d). However, Australia's policy as per the AUSVETPLAN Disease Strategy for Avian Influenza is that no destruction or culling of healthy wild birds will occur because it is not practical or environmentally sound and may be counterproductive in stopping spread of the disease (WHA 2023d). The isolation facility should have strict biosecurity protocols to limit disease transmission and ideally have separate staff, which do not have backyard birds or chickens.

Management of carcasses

In the 2018 HPAI event in African penguins minimising colony disturbance was prioritised, so although carcass removal was perceived as ideal, it was ultimately not considered essential (Roberts et al. 2023). People managing and handling sick individuals and dead carcasses must adhere to strict biosecurity recommendations to limit further transmission of infection. Indirect effects of people trampling over beaches and through habitat and burrows and potential disease transmission must be counter balanced with removal of carcasses to minimise scavenging and secondary infection. Removal should be assessed on a site-by-site basis, taking into account accessibility, and ability to collect and dispose of carcasses in line with the Australian Environmental Protection Authority guidelines.

The Wildlife Health Australia high pathogenicity avian influenza risk mitigation toolbox for wildlife managers further asks to consider:

- Staff awareness and training
- Documenting current Australian policy as part of the site HPAI risk mitigation plan (e.g. wild birds would not be culled as a result of infection detected)
- Document features of the site and wild animal populations that may be useful when making decisions around removal of infected carcasses.
- Could people and vehicles easily access sites if carcasses were to be removed?
- What scavengers are present in the area that are likely to scavenge on carcasses?
- Do members of the public have access to the site? If so, can their access be restricted?
- What would the risk to the population be of significant disturbance of the site, such as accessing the site with people, vehicles and interacting closely with the population and habitats to remove carcasses?

Preventative management and vaccination

While the above practices entail best practice disease risk management, what is also needed is consideration of preventative health and control strategies to promote health and longevity in regards to anthropogenic hazard mitigation. Vaccination should be floated as realistically one of the few ways to minimise the consequences of exposure to infection. Again, this must be counterbalanced with the logistics and feasibility of a large scale catch up for initial vaccination and individual identification. The WOAH (2023c) 'Considerations for emergency vaccination of wild birds against high pathogenicity avian influenza in specific situations' is an excellent discussion paper providing guidance on considerations for emergency vaccination of wild birds against HPAI in immediate response to an outbreak or increased risk of introduction of HPAI. This recent paper provides high level international guidance, however, still requires national interpretation within governmental jurisdictions.

Vaccination against H5N1, or any strain of AI, comes with challenges, including the logistics of giving a vaccine to a large group of birds and difficulties in differentiating between infected and vaccinated birds, meaning there may be trade implications (Mills 2023). Vaccination, as a method of preventing infection, virus shedding and (or) disease, was stated as impractical for wild populations by Roberts et al. (2024) in regards to the 2018 HPAI disease event. However, available vaccines and vaccine technology should be explored to assess possible situations where vaccination may become feasible. At the LP DRA workshop held Nov 30 at Werribee in Victoria, Roberts advocated for vaccination as a method to control disease and recommended further consideration of vaccination. The only reported avian wildlife species to be vaccinated to date for HPAI is the critically endangered Californian condor (Mills 2023). This is the first time the US has approved vaccination of any bird against the disease and may pave the way for further discussions. The European Food Safety Authority (2007) reported on vaccination of zoo birds against HPAI in 13 EU Member States. The report stated that bio-security measures should be implemented in zoos as a first line of protection of zoo birds against the introduction of AI viruses. It also indicated that vaccination against avian influenza of the H5 and H7 subtypes with currently authorised vaccines developed for use in poultry is safe, and in most taxonomic orders of zoo birds, effective in terms of inducing HI serum antibody titres. Roberts et al. (2024) conducted a vaccine trial on 24 captive African penguins with either a conventional inactivated clade 2.3.4.4b H5N8 HPAI whole virus or a tobacco leaf-produced H5 haemagglutinin-based virus-like particle (VLP). Six birds received a second dose of the inactivated vaccine. Antibody responses were assessed and compared by employing haemagglutination inhibition tests. A second dose of inactivated vaccine was required to induce antibody titres above the level required to suppress virus shedding and antibody levels had dropped below protective levels by day 175, while a single dose of VLP vaccine produced suppressive levels by day 14, and one bird still had antibodies on day 430. Further trials are obviously necessary, but this is an example of where newer vaccine technology, combined with innovative vaccine administration systems, could provide more feasible options for wild bird vaccination.

It was discussed at the LP DRA workshop in November 2023 that it may be beneficial to conduct a clinical vaccine trial on captive LPs at Australian zoo's post seeking approval from the Australian govt for this purpose. Based on prior experiences and presumed susceptibility, it would seem this avenue should be further explored. However, this would

require Australian government approval (decisions around implementing vaccine programs are made by the Consultative Committee on Emergency Animal Disease (CCEAD) which according to the current Wildlife Health Australia risk mitigation toolbox for wildlife managers (2023) may be challenging to obtain see section 5.9 below https://wildlifehealthaustralia.com.au/Portals/0/Incidents/WHA_HPAI_Risk_mitigation_toolbox.pdf (WHA 2023d):

Wildlife Health Australia guidelines current state that "Following an outbreak of HPAI, Australia's preferred policy is to control the disease without the use of vaccination. The use of vaccination in wild birds is not considered to be feasible. Ensure that communications and procedures clearly state that environmental modification, environmental disinfection, wildlife dispersal or wildlife containment are not considered effective or appropriate for HPAI control, irrespective of the species.

However, at the same time we note that vaccination may be considered in poultry if the outbreak has become widespread, or to protect rare, endangered and valuable captive birds (see AUSVETPLAN Avian Influenza and AUSVETPLAN Guidance Document-Risk-based assessment of disease control options for rare and valuable animals) and that decisions around implementing vaccine programs will be made by the Consultative Committee on Emergency Animal Disease (CCEAD)' (WHA 2023d)

The current global HPAI situation has provoked interest in the use of vaccination in wild birds. The advice from the joint CMS and FAO's Scientific Task Force on Avian Influenza and Wild Birds is that vaccination could be considered for key localised populations where it is practically and financially feasible (WHOA 2023c). However, there are a number of constraints to the use of vaccination as a risk mitigation tool for HPAI in free-ranging wildlife and to date its application in this context has been limited (for example vaccination of California Condors in the California Condor Recovery Program).

The Phillip Island (Millowl) LP population could be considered a key localised population of significance owing to the importance of the species in regards to ecotourism and the Phillip Island (Millowl) economy as well as being a highly charismatic species (high public interest value), useful bioindicator species (Dann et al. 2005) and logistically, owing to its inability to fly would be reasonably robust to manually restrain for vaccination and subsequent monitoring. However, It is recommended that all of the suggested steps in the HPAI Risk mitigation toolbox are fulfilled prior to making a case to the CCEAD for vaccination of PI LPs.

Summary of potential risk management strategies:

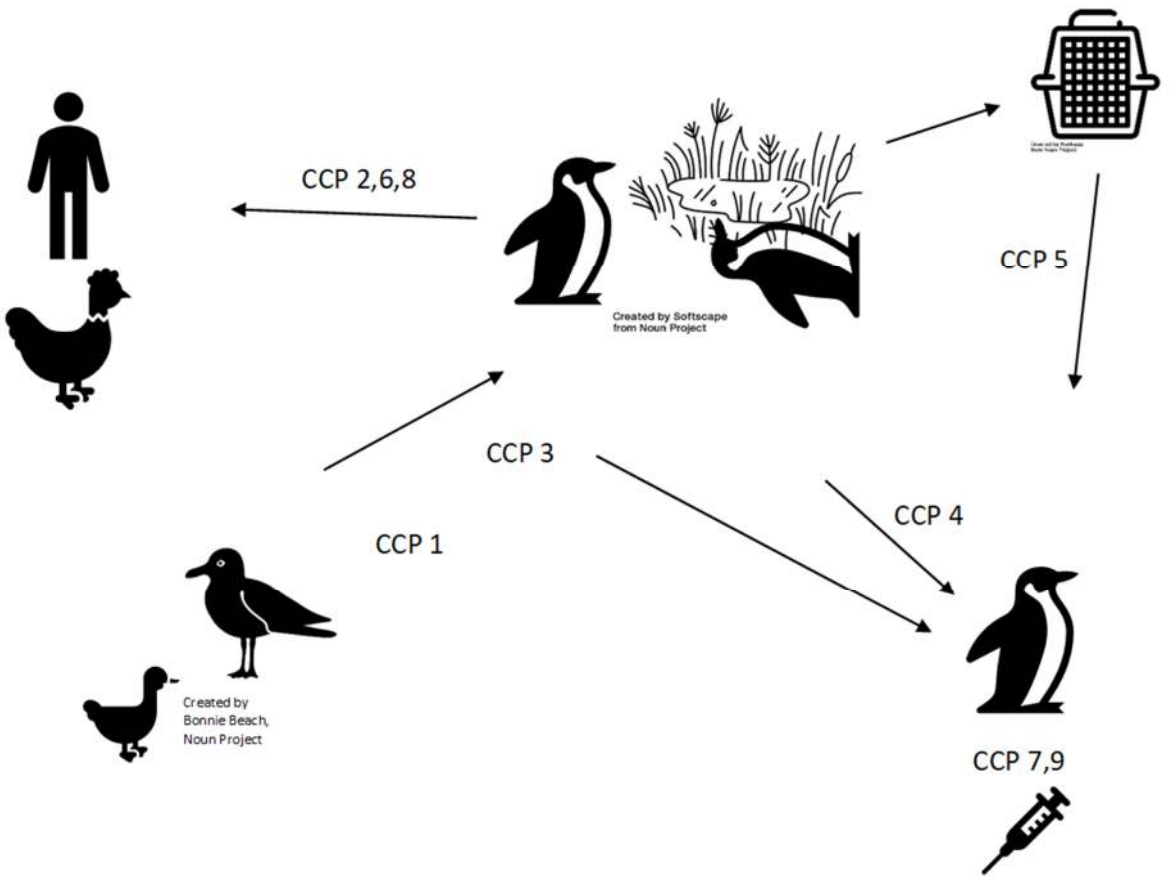


Figure 4. Critical control points for the hazard avian influenza (HPAI)

Information about avian influenza in humans should be distributed to conservation authorities, with instructions to visit the nearest clinic if flu-like symptoms were experienced.

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	High	High	Passive surveillance of relevant populations on the peninsula can lead to preventative actions such as removing affected carcasses to minimise access to penguins and other seabirds. Staff that are trained to observe carefully and recognise signs of HPAI will improve effectiveness of passive surveillance.	Y
2	Communication with public	High	High	Educating and warning public of disease risks and importance of avoiding direct contact with penguins in outbreak scenario, for example flyers to close residents, posters at community areas. This is particularly important also for the free-range poultry facility on the island. It is important that they are aware of the clinical signs associated with HPAI, and how to respond. The public can also assist with reporting suspected cases.	Y
3	Active surveillance of live birds	Low	Low	Disease can be transmitted through direct contact between conspecifics. Therefore, early detection in live birds could be used as an early warning system. However, this is likely to be not cost effective, with most surveyed birds negative currently. Passive surveillance of dead or sick penguins (and potentially other seabirds) with prompt laboratory testing is a far more effective strategy.	N

4	Passive surveillance and removal of any dead carcasses	High	High	It is important to catch outbreaks early, because dead birds create an infection source for the rest of the population. Dead birds can be removed in an attempt to reduce outbreak severity.	Y
5	Avian Biosecurity	High	High	In case of an outbreak, HPAI can be carried on fomites (cages, car tyres, clothing etc); high biosecurity and appropriate decontamination is therefore essential. PINP staff with chickens at home must shower before work and wear clean clothing not used to service backyard chickens or fowl, or ideally, have NO contact with chickens during the outbreak.	Y
6	Human biosecurity	High	High	Humans are relatively resistant to AI viruses but high standards of personal hygiene should be used when dealing with poultry or handling wild birds including hand washing and taking care to avoid rubbing eyes and touching the mouth, eating, drinking or smoking until hands are clean. Appropriate personal protective clothing should be worn, gloves, facial and ocular protection; birds should be held as far as possible from the face during handling.	Y
7	Treatment	Moderate	Low	Theoretically, supportive care only however, many birds die peracutely, and treatment of large numbers of affected birds would be difficult. They would need to be housed in strict isolation during treatment and PPE	N

7	Routine sample collection / clinical examinations should cease at times of high risk of infection	Moderate	High	At times of higher risk, e.g. when infection has been found within country or region, and/or during long periods of extreme weather conditions, stressors to wild bird populations (e.g. routine monitoring) should be minimised.	Y
8	If disease has been confirmed in a region:	Moderate	High	Extra care should be taken regarding potential for introducing infection on fomites such as footwear or vehicle tyres, using disinfection procedures, as appropriate. Site access should be restricted during these times. Disturbing activities, should be suspended. Public education to raise awareness of HPAI H5N1, the risks it poses, and some simple precautions and response actions, should be given, including suspension of feeding of wild birds.	Y
9	Vaccination	High	Moderate	To reduce associated morbidity and mortality associated with disease and control outbreak scenario. Emergency vaccination of wildlife now being considered by WOAH (2023c).	consider

4.4.2 Disease risk assessment for Saumarez reef virus

Justification of hazard		
The flavivirus Saumarez Reef Virus is an arbovirus (arthropod borne virus) that was initially isolated from seabird ticks collected from four localities (St George et al. 1977). Two strains were isolated from ticks of the species *Ornithodoros capensis* Neumann 1901 collected from the nests of Sooty Terns, *Sterna fuscata* Linnaeus 1766 on coral cays off the east coast of Queensland, Australia. The other three strains were isolated from ticks of the species *Ixodes eudyptidis* Maskell 1885 taken from two dead Silver Gulls (*Larus novaehollandiae)* Stephens 1826 in northern Tasmania. Saumarez Reef virus is believed to have been responsible for febrile illness in meteorological workers on the Saumarez and Frederick reefs in Australia, (St George et al. 1977). Yet subsequent serological studies of Heron Island Qld human inhabitants showed no evidence of exposure (Humphrey-Smith et al. 1991). Furthermore *I. eudyptidis* does not attach to and feed on humans (Dehhaghi et al. 2019). However *Ornithodoros capensis*, that primarily feeds on seabirds, can bite humans if the opportunity is provided. Off-shore islands are the most likely place that this tick bites humans because they provide nesting grounds for seabirds; therefore, campers, explorers, and those who participate in recreational and professional fishing and seabird researchers are at higher risk (Dehhaghi et al. 2019). An experimental transmission study was reported in Phillip Island (Millowl) LPs (Morgan et al. 1985). Saumarez reef virus, and an unnamed virus isolated from ticks on Macquarie Island, Southern Ocean were reportedly pathogenic causing disease and mortality in Phillip Island (Millowl) LPs inoculated with the viruses (Morgan et al. 1985). The Saumarez reef virus caused death 9-13 days post inoculation in LPs. Lesions included gastrointestinal haemorrhage, hepatic periacinar necrosis and severe necrotising enteritis (Ladds 2009). Transmission in this case was via direct inoculation (for a research study) and naturally would be via a presumed tick bite. Ticks were considered the most likely vector of infection as flavivirus strains have been isolated from Ixodid ticks (*Ixodes uriae).* Natural infection with this virus in LPs in Australia has however not been reported.		
Risk assessment		
Entry assessment	Exposure assessment	Consequence assessment
LPs are susceptible to virus experimentally. Direct inoculation caused resultant morbidity and mortality however unknown if ticks present at Phillip Island (Millowl) carry this virus although seems unlikely based on the fulminant results of the transmission trial and lack of subsequent morbidity and mortality. Currently would seem to be a low likelihood that LPs could introduce the hazard to PINP with the appropriate vector and infection present in the vector	If the tick (vector) is present and infected with Saumarez Reef Virus and LPs are infected there is a medium likelihood of dissemination to other sympatric avian species at LP nesting sites including the short-tailed shearwater, pacific gull, kelp gull, silver gull, crested tern, Cape Baron goose, Purple swamphen, Sooty oystercatcher and the Hooded plover. Especially those that are gregarious such as gulls which tend to congregate with high host density.	Has caused death in Phillip Island (Millowl) LP population when directly and experimentally inoculated. However, low likelihood of exposure and transmission to other penguins and sympatric species unless competent vector present. Potential disease risk for LPs being exposed in the wild if vector species present and of moderate consequences to population and other avian species present at nesting sites. Potential for human infection but not definitively proven
Risk evaluation		
Preventative measures should be employed to reduce the disease risks.		

Risk management options

Diagnosis

Serological tests for antibodies (haemagglutination inhibition, serum neutralisation, immunofluorescence and enzyme immunoassays) are typically used to indicate exposure to flaviviruses, in humans, and other hosts. PCR to detect RNA can be undertaken on whole blood (EDTA) serum, or cerebrospinal fluid (CSF) although this would require general anaesthesia of birds and is not feasible in the field. Virus isolation of blood, serum or other tissues may also be used. Confirmation of a suspected infection requires either the isolation of the virus itself, detection of RNA, a fourfold rise in the IgG titre between acute and convalescent serum samples, or detection of IgM in serum or CSF. Recommended to contact Victorian Infectious Disease state laboratory regarding testing given Saumarez Reef Virus not currently listed on routine flavivrus testing within Victoria.

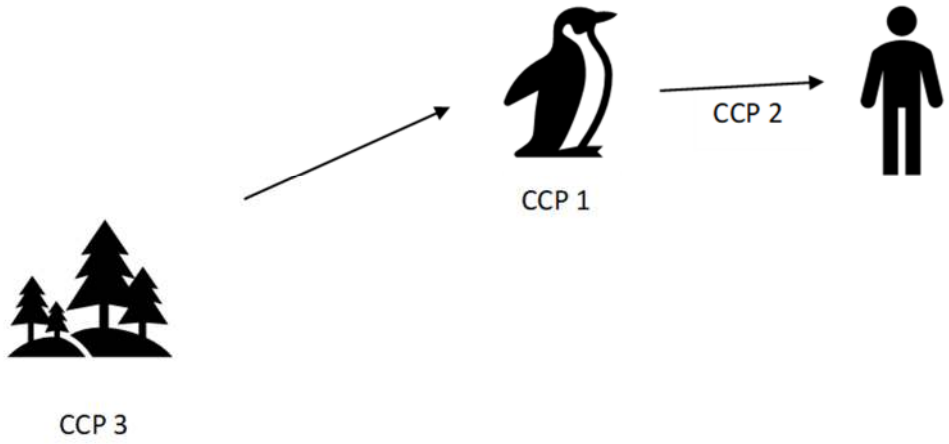


Figure 5. Critical control points for the hazard Saumarez Reef virus

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Infection prevalence unknown. Direct inoculation resulted in mortality, so useful to screen LPs and sympatric species for seroconversion. It would be beneficial to conduct flavivirus passive surveillance via PCR and serology and include Saumarez reef virus testing, to detect sudden increases in prevalence or severity. As passive surveillance is recommended for other diseases, it makes sense to also document infection with this and other flaviviruses present in the colony.	Y
2	PPE	High	High	Personal precautions for humans can include avoiding tick infested areas, using repellents, wearing light coloured clothing and regularly checking for ticks.	Y
3	Environmental strategies	Low	Medium	Modification of the habitat to decrease humidity through clearing of vegetation and chemical control of ticks in the environment may also be used.	N

4.4.3 Disease risk assessment for Avian paramyxovirus 1 or Avian orthoavulavirus 1

Justification of hazard
Avian orthoavulavirus 1 (AOAV-1), belongs to the genus Orthoavulavirus of the family Paramyxoviridae. Strains of AOAV-1 vary greatly in their virulence and tissue tropism, and in susceptible birds infection can result in wide ranging clinical signs and pathological lesions (Brown and Bevins 2017). Due to multiple name changes in recent years, the terms APMV-1 and Newcastle disease virus (NDV) still appear in the majority of scientific literature. Newcastle disease (ND) is a commonly used term for virulent APMV-1, when it is found in poultry. However, there are strict definitions of when an APMV-1 infection is termed ND (WHA 2016). Furthermore AOAV-1 infection in other avian species is often referred to as ND in the scientific literature, although this does not meet the WHOA definition. AOAV-1 strains are further classified into 5 pathotypes based on clinical signs seen in **infected chickens**: 1) viscerotropic velogenic: highly pathogenic/virulent, haemorrhagic intestinal lesions are frequently seen 2) neurotropic velogenic: highly pathogenic/virulent, high mortality usually following respiratory and nervous signs 3) mesogenic: moderately pathogenic, respiratory signs, occasional nervous signs, but low mortality 4) lentogenic or respiratory: lowly pathogenic, mild or subclinical respiratory infection 5) asymptomatic: usually subclinical enteric infection (AHA 2014; OIE 2019). NDV can have devastating effects on the poultry industry due to the high morbidity and mortality associated with the virulent strain. Clinical signs of Newcastle disease in chickens include a drop in egg production, respiratory distress, listlessness, weakness, and central nervous system signs and mortality (Hines and Miller 2012). Epizootics continue to occur on a regular basis in Central and South America, Africa, and Asia, while sporadic epizootics occur in Europe (Hines and Miller 2012). AOAV-1 is endemic in Indonesia, East Timor and South-East Asia, with West Papua being the closest infected area to Australia (AHA 2014). 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 (WHA 2016). 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). Wild gulls, waterfowl and shorebirds may be reservoir hosts for lentogentic pathotypes, which can become virulent following mutation in domestic poultry (OIE 2013). Outbreaks of ND from 1998 to 2002 in NSW and Victoria were a result of mutations in one or more of these strains. Australia is currently free from ND, with vaccination, in accordance with the WHOA definition of an ND-free country (OIE 2018). Pigeon paramyxovirus (PPMV-1) is present in domestic and wild pigeons in Australia and is notifiable in Victoria, New South Wales and Western Australia (Agriculture Victoria 2018). The Department of Agriculture, Water and the Environment monitors for the disease in birds across northern Australia through the Northern Australia Quarantine Strategy (NAQS) surveillance program (Department of Agriculture, Water and the Environment 2020). It is compulsory in Victoria for owners or managers of commercial poultry flocks to vaccinate chickens against ND in line with nationally agreed National ND Management Plan. This is required by the Disease Management Plan and regulation 63 of the Livestock Disease Control Regulations 2017. A commercial poultry flock is any managed group of more than 1000 domestic chickens including meat chickens, laying hens and chickens used for breeding purposes (Agriculture Victoria 2023) Paramyxovirus seropositivity is widespread in free-ranging penguins but disease is rare (Stidworthy and Denk 2018). Penguins seropositive for APMV-1 have been reported in the Antarctic (Morgan and Westbury 1981), Argentina (Karesh et al. 1999), Macquarie Island (Morgan et al. 1981) and South Shetland Islands (Thomazelli et al. 2010). Thomazelli et al. (2010) reported that the strains detected in penguins at the South Shetlands Islands had low pathogenicity. APMV-1 infection has also been demonstrated in captive penguins in the United States (Pierson and Pfow 1975), where a velogenic neurotropic strain was identified in birds being shipped from Scott Base in the Ross sea to the USA resulting in multiple deaths. Infection and disease was also reported in a captive king penguin (Krauss 1963). It is clear that penguins are susceptible to this virus and that some strains, presumably those with low pathogenicity, circulate in wild penguin populations.

Recurrent, seasonal high mortality events in double-crested cormorants, attributable to virulent Newcastle Disease Virus (NDV); APMV-1, have occurred in breeding colonies in Canada and USA since 1992 (White et al. 2015) with deaths of 20,000 or more and up to 90% mortality. APMV-1 infection has not been reported in cormorants in Australia. Wild birds usually do not demonstrate classic clinical signs of virulent paramyxovirus infection however birds in a weakened state (e.g. during annual migrations), co-infections or adverse environmental conditions may develop clinical signs and die.

The virus can infect non-avian hosts, including primates (humans, monkeys), rabbits, and pigs. Human infection may result in transient conjunctivitis and/or flu-like signs (AHA 2014; OIE 2019) and more recently a fatal case of neurological infection caused by PPMV-1 has been reported in a child (Hurley et al. 2023).
APMV-1 is highly contagious and spread via aerosolisation and ingestion (faecal-oral route). Rodents, insects, humans and other animals can act as mechanical vectors (Australian Department of Agriculture, Water and the Environment 2020). Environmental transmission (e.g. via nesting hollows or nesting material) is also suspected, due to the long term persistence of the virus (Eastwood et al. 2019). The incubation period varies from days to weeks and clinical signs can be severe, although subclinical infections may occur. Sub-clinical carrier birds shed virus intermittently. Treatment of birds with ND is ineffective, however, vaccination can reduce clinical signs of disease (AHA 2014).

Risk assessment

Entry assessment	Exposure assessment	Consequence assessment
Known to infect LPs and low pathogenicity strains may already be circulating at PINP colony. Virulent strains are highly contagious and thought to be transmitted between individuals through inhalation and, ingestion. Overall there is a low likelihood of virulent APMV1 presence in LPs at Phillip Island (Millowl) as there has been no evidence on necropsy exam of prior records, however if and when present it could be acquired through exposure at nesting sites and would likely rapidly disseminate through the colony. Penguins may acquire infection from exposure to sympatric species such as kelp gulls which frequent poultry farms especially free-range farms, where wild birds (waterbirds, raptors,) visit and then return to Phillip Island (Millowl) (Singh et al. 2014). Wild gulls, waterfowl and shorebirds may be reservoir hosts for lentogentic pathotypes, which can become virulent following mutation in domestic poultry (OIE 2013). Kelp gulls, are present all year round within the penguin colony at Phillip Island (Millowl) (Dann	LPs will forage in the ocean, walk up the beach in groups to nesting sites then mate and rear offspring and if infected could excrete virulent APMV1 via aerosolization and ingestion. The virus is highly contagious. Sympatric avian species could also become infected via close contact at nesting sites, contaminated water or food of infected birds, contact with people, equipment, vehicles, shoes or clothing contaminated by infected birds. Given the wide host range all avian sp. may be susceptible and high likelihood of exposure and dissemination. Poultry and other avian species are susceptible to infection with AOAV-1 from waterbirds. Mild human infection is possible. Overall likelihood of susceptible species in exposure groups being exposed to virulent APMV-1 associated with LPs was estimated to be medium.	Once exposure of susceptible species to virulent APMV-1 has occurred, a number of possible outbreak scenarios could occur, ranging from no spread to widespread establishment. If virulent APMV--1 establishes in directly exposed populations (wild birds, and/or low biosecurity poultry), it may then spread to other populations of wild birds and/or low biosecurity poultry and becomes endemic in Australian wild birds. This is based on APMV-1 being highly contagious, that it can persist in the environment and be spread by mechanical vectors and on fomites, and because waterbirds can be a subclinical reservoir of APMV-1 infection and shed virus (Department of Agriculture, Water and the Environment 2020). The effect on international trade would depend on the species infected. Infection of poultry with virulent APMV-1 is notifiable and would likely result in trade restrictions. There would be cessation of exports in the short term, with recommencement after a period of review by, and negotiations with, trading partners. Some markets may not return to normal trade for some time following ND eradication in poultry. Virulent APMV-1

2007). The greatest risk of incursion is likely to come from kelp gulls that travel from the island to mainland sites, where it is possible for them to have contact with domestic chickens in both commercial and private settings. LPs are typically grouped in close contact when exiting the water to walk up the shore towards their nesting sites. If infection ensues there is a medium likelihood of developing disease which may cause respiratory and neurological signs compromising ability to forage and feed and likely mass morbidity and mortality.		detected in wild birds only should not result in trade restrictions in poultry. An outbreak in commercial poultry would impact communities in affected areas, depending on the scale of the outbreak. A large outbreak may impact supplies of poultry and poultry products. Movement restrictions and suspension of community activities due to control/eradication measures (if the outbreak occurs in an area close to commercial poultry farms) could result in significant levels of community concern. Other consequences of an outbreak in wild birds may include a reduction in biodiversity if high mortalities are encountered. Humans may also become infected, those that handle wild birds and poultry workers are most at risk, however clinical signs are typically mild. Based on these considerations it was estimated that the likelihood of establishment and spread of virulent APMV-1 through populations of penguins, wild birds and/or poultry was moderate if penguins were exposed to virulent APMV-1 at PINP.
Risk evaluation		
Preventative measures should be employed to reduce the disease risks.		
Risk management options		
Diagnosis – Initial laboratory testing is conducted by quantitative RT-PCR and documented wild bird mortality. Separate cloacal and tracheal swabs (not pharyngeal) should be submitted in viral transport media (PBGS) and with clear labels to identify bird and tissue sampled. ○ Use sterile swabs. ○ Paediatric swabs can be used for small birds. ○ Submit chilled. Dead birds should be submitted securely packed (i.e. double bagged and in a rigid container that is clearly labelled as suspect APMV1) and can be swabbed at the laboratory. The carcass will be put on hold for further testing pending results.		

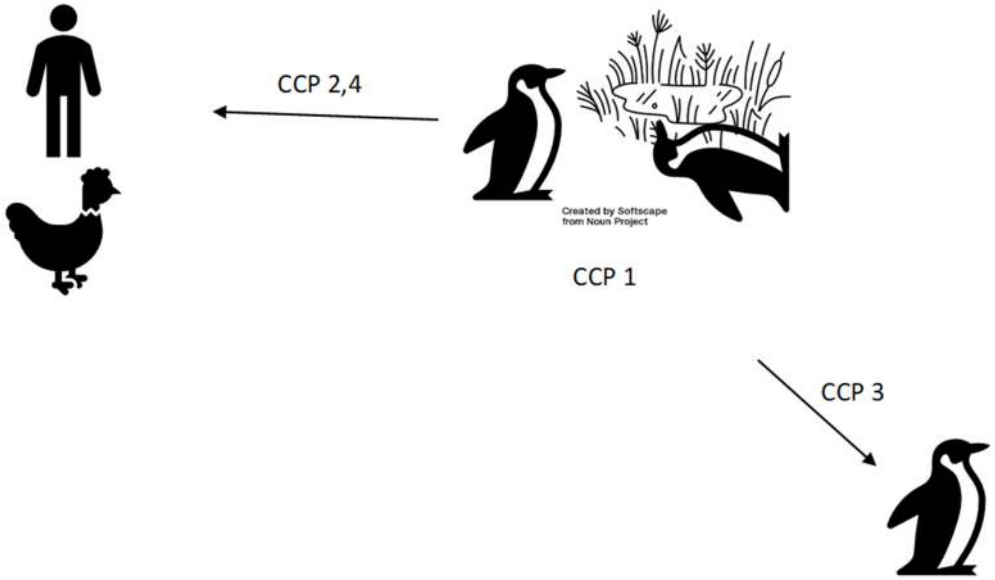


Figure 6. Critical control points for the hazard avian paramyxovirus 1

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance, Research/active surveillance, treatment	Moderate	High	Passive surveillance can be used to investigate/research prevalence and potential impacts. Active surveillance for the presence of the virus could be used to inform on current prevalence of infection. Treatment of infected individuals is not feasible. There are no known measures to reduce risk of infection in free ranging wildlife. However,	Y

				given infection can be subclinical, reducing stressors during planned catch ups and monitoring is recommended.	
2	Biosafety and education	High	High	Zoonotic infection is known to occur but is mild unless immunocompromised. General PPE and biosafety considerations should be used to reduce likelihood of infection. Education of people in contact with penguins should be used to raise awareness of possibility of infection. Cleaning and disinfection, including personal hygiene, (e.g., hand washing, cleansing/ disinfection of footwear) are also important in preventing transmission on fomites.	Y
3	Removal of any dead carcasses	High	High	Dead birds create an infection source for the rest of the population. Dead birds can be removed to reduce outbreak severity.	Y
4	Vaccination	High	High	Both commercial producers and owners of backyard poultry may have to vaccinate as part of the response policy or may choose to vaccinate if they do not already, at an additional expense. Poultry may be vaccinated for AOAV-1 with live or inactivated vaccines. Vaccine-induced immunity lasts 10–12 weeks and repeat vaccinations are required to maintain adequate protection (AHA 2014). However, any vaccination of birds may mask clinical disease and hamper viral detection techniques.	N (for LP)

4.4.4 Disease risk assessment for Avian chlamydiosis

Justification of hazard -
Globally distributed, *Chlamydia psittaci* are gram-negative intracellular bacteria. All bird species are susceptible to *C. psittaci* infection however associated disease, avian chlamydiosis is rarely reported in wild bird populations (Amery-Gale et al. 2020, WHA 2017b). Strain differences exist with host-adapted strains of *C. psittaci* tending to produce only a mild to moderate disease in the natural host, although may result in prolonged or persistent infection (Andersen and Franson 2007) and increased susceptibility to disease in other species. *C. psittaci* is a zoonotic pathogen commonly referred to as psittacosis, ornithosis, and parrot fever. Avian chlamydia increasing in frequency in LPs and Yellow-eyed penguins in New Zealand (B. Gartrell pers comm Nov 2023). Zoonotic pathogen. An outbreak of *C psittaci* occurred in an outdoor colony of 63 Magellanic penguins at the San Francisco Zoo (Jencek et al. 2012). Serological surveys (complement fixation test) show that wild populations of various penguin species have antibodies to *C. psittaci* (Gallo-Vaulet et al. 2022). However unknown if results reflect exposure to *C. psittaci* or to other antigenically similar organisms. Cameron (1968) reported on the isolation of "a member of the Psittacosis-Lymphogranuloma Venereum group of viruses" from the lungs of an emperor penguin chick found dead in Antarctica, but it is unknown whether this organism was *Chlamydia* sp. or another *Chlamydiales*-like organism. Recent attempts to detect DNA from *C. psittaci* in faecal samples from wild penguins with real-time PCR tests failed, but Isaksson et al. (2015) detected DNA from *Chlamydiales* sp. in cloacal swabs from 7/105 chinstrap penguins sampled in the Antarctic Peninsula. Gallo Vaulet et al. (2022) reported the existence of a group of Chlamydiaceae-like bacteria that can be detected in the faeces and cloacal swabs of aquatic birds such as penguins, gulls, and flamingos. This group of bacteria is comprised of the recently proposed genus Chlamydiifrater as well as other strains that have yet to be formally described, including the strain detected in cloacal swabs from wild Magellanic penguins. The nature of disease in infected birds will depend on the host and strain of bacteria. Stress and immune function can also influence susceptibility to infection and disease. Clinical signs of disease include lethargy, weakness, reduced appetite, ruffled feathers, weight loss, ocular and nasal discharge, diarrhoea and green coloured urates. More severe cases may be fatal (WHA 2017b). However, there may be no clinical signs and many chronically infected birds show no signs of infection until stressed. Younger birds are considered more susceptible to infection and may develop a higher level of morbidity and mortality, though transfer of maternal antibody via the egg may provide protection to young nestlings (Andersen and Franson 2007). Psittacine birds often shed Chlamydia and develop clinical signs after transportation and introduction to new environments (WHA 2017b). In the outbreak of *C psittaci* in 63 Magellanic penguins at the San Francisco Zoo affected penguins presented with inappetence, lethargy, and light green urates. Haematologic and serum biochemical findings were consistent with chronic inflammation. Penguins did not respond to initial supportive and antimicrobial therapy, and three died. Necropsy results reported hepatomegaly and splenomegaly, and histologic lesions included necrotizing hepatitis, splenitis, and vasculitis. *C. psittaci* was confirmed by Gimenez staining, immunohistochemistry, and tissue PCR (Jencek et al. 2012). Diagnosis can be challenging, as *Chlamydia* are often shed intermittently (Stokes *et al.* 2020, Gerlach 1994) particularly in chronically infected birds, which can result in false negative test results. In humans, clinical signs can vary from mild to potentially fatal systemic disease with severe pneumonia (CDC 2010). Commonly reported clinical signs include conjunctivitis, non-productive cough, fever, chills, headache, malaise, and myalgia (WHA 2017b). Infection more severe in the immunocompromised. Transmission mostly occurs via ingestion or inhalation, with infectious material shed in faeces, nasal secretions, and eye secretions. Exposure to infection may arise from close contact with an infected bird or via contamination of the environment with infectious material, enhanced by prolonged or persistent shedding of highly resistant elementary bodies from infected birds. Aerosolisation may be an important route for dissemination of infection in a flock setting, especially around communal feeding areas. Levels of vertical transmission are considered to be low, though transmission from parent to offspring may occur through regurgitated crop content (Andersen and Franson 2007). Potential disease risk for LPs being exposed in the wild and developing disease, may also reactivate and shed during times of stress or immune-compromise e.g. Wide

host range makes other avian sp. susceptible.		
Risk assessment		
Release assessment	Exposure assessment	Consequence assessment
Known to infect LPs. Thought to be transmitted between individuals through inhalation, ingestion, direct inoculation into the eye, or venereal transmission (CFSPH 2017). LPs come into contact with other LPs when exiting the water to come ashore and find their nestboxes and nesting sites. Overall, there is a low likelihood of Chlamydia being present in LPs at Phillip Island (Millowl) as there has been no evidence on necropsy exam of prior records, however if and when present it could be acquired through exposure at nesting sites and nest boxes, or at mating. LPs can be in close contact when exiting the water to walk up the shore towards their nesting sites. If infection ensues there is a medium likelihood of developing disease which may cause respiratory infection and compromise sight, and therefore ability to forage and feed.	LPs will forage for resources, mate and rear offspring and if infected could excrete *Chlamydia* sp. through inhalation, ingestion, direct inoculation into the eye and nares and vertical transmission. The likelihood of exposure to other avian sp. is probably low other than to sympatric avian species at nest sites including the short-tailed shearwater, pacific gull, kelp gull, silver gull, crested tern, Cape Baron goose, purple swamphen, sooty oystercatcher and the hooded plover. If exposed it is likely that the individual will disseminate *C. psittaci* through inhalation, ingestion, direct inoculation into the eye and nares and possibly vertically to other susceptible avian hosts. Given the wide host range, all avian sp. may be susceptible and may be exposed to a novel strain of *C. psittaci* which may lead to higher consequences.	There is a medium likelihood of exposure through females and males raising offspring, and through foraging behavior. If exposed, infection could result in mild to severe respiratory disease including death. Stressors and co-infections may also play a role in promoting overt disease. However, populations that have existed with chlamydia for a significant period of time appear to achieve a balance with moderate to high infection rates but low rates of clinical disease. Yet, when combined with other pressures, there is potential for the organism to cause significant declines, particularly in naïve destination populations. The likelihood of humans contracting Chlamydiales infection from LPs is low but should be considered in the immunocompromised. Therefore the overall risk of *Chlamydia* sp. Infection is MEDIUM. However, it is also important to understand that changing environmental factors and the presence of external stressor factors in the future could lead to changes in the clinical expression of this disease. Disease may therefore become more or less apparent relative to environmental change and this needs to be considered when managing the species into the future.

Risk evaluation		
Preventative measures should be employed to reduce the disease risks.		

Risk management options

Diagnosis of AC is based around detection of the organism and/or demonstration of an increasing antibody titre, alongside presence of clinical signs or pathologic lesions. Molecular techniques, particularly PCR, are thought to be the gold standard for detection of the organism. However false positive results can arise through contamination of sample material, particularly when large numbers of birds are being sampled. The sites typically sampled for diagnostic PCR include the cloaca, choanae and conjunctiva.
Birds with chlamydiosis often show anaemia (haematocrit 30,000) and absolute heterophilia and monocytosis (WHA 2017b).

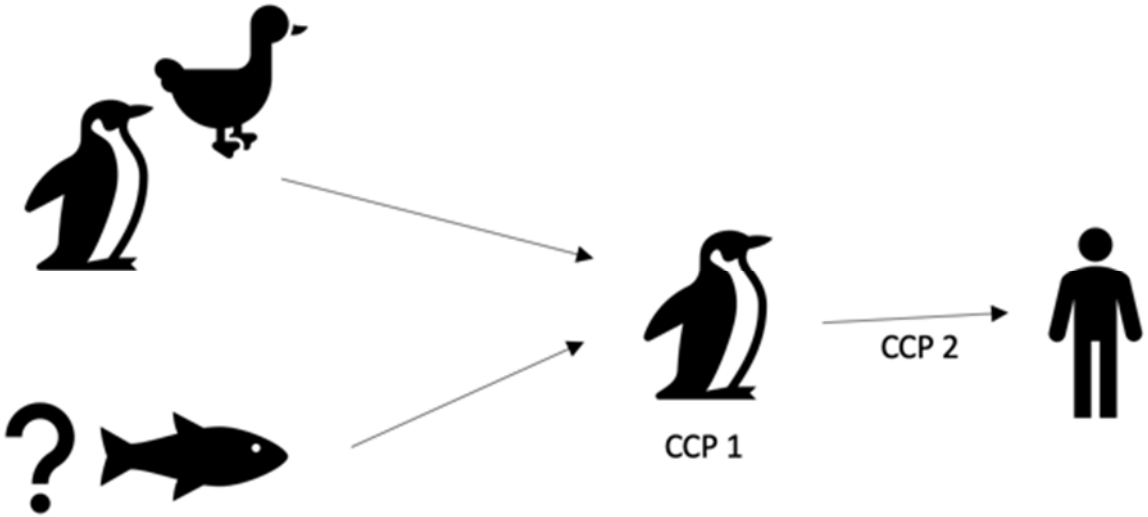


Figure 7. Critical control points for the hazard avian chlamydia

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance, Research/active surveillance, treatment	Moderate	High	The consequences and probability of chlamydial infection in little penguins are poorly understood. Passive surveillance can be used	Y

				to investigate/research prevalence and potential impacts. Active surveillance for the presence of the bacteria could be used to inform on current prevalence of infection. Treatment of infected individuals is possible but prolonged. Drugs effective against *C. psittaci* include doxycycline and azithromycin (Andersen and Franson 2007). The choice of administration e.g. intramuscular doxycycline every 5 -7 days and retest at 45 days compared to in water medication, or mixing doxycycline into fish will be dependent on the number of birds affected, stress associated with capture and presumed compliance (WHA2017b). Birds undergoing treatment for AC should be placed in isolation until completion of treatment and follow up testing indicates they are no longer shedding. There are no known measures to reduce risk of infection in free ranging wildlife. However, given infection can be subclinical, reducing stressors during planned catch ups and monitoring is recommended.	
2	Biosafety and education	High	High	Zoonotic infection with chlamydial bacteria is known to occur, though probability of infection in contact with penguins is unknown. General PPE and biosafety considerations should be used to reduce likelihood of infection. Education of people in contact with penguins should be used to raise awareness of possibility of infection. Cleaning and disinfection, including personal hygiene, (e.g., hand washing, cleansing/ disinfection of footwear) are also important in preventing transmission on fomites.	Y
Further research is necessary to isolate and characterise these Chlamydiaceae-like bacteria, and to investigate their epidemiology, pathogenicity, and zoonotic potential (Gallo Vaulet et al. 2022).					

4.4.5 Disease risk assessment for Avian cholera

Justification of hazard		
Pasteurella multocida is the highly infectious bacterial agent of avian cholera. It is found worldwide and has multiple names, specifically pasteurellosis, avian cholera and fowl cholera (Allen et al. 2024). It is reported to infect domesticated and wild fowl causing significant and explosive mortality. Over 100 species of wild birds are susceptible to disease with North American wild fowl the most severely impacted (Botzler 1991). Outbreaks of avian cholera leading to significant mortality events in free-ranging birds, are reported in southern rockhopper (Jaegar et al. 2018), Adélie (Leottie et al. 2006), King and macaroni (Cooper et al. 2009) penguins, brown and great white pelicans, double-crested and Cape cormorants, common loons, pie-billed, horned, eared, and Western grebes (Stidworthy and Dent 2018). Infected birds can remain carriers for life. There have been no reported mass mortality events of avian cholera in wild Australian birds (including penguins), but it has routinely been implicated as the cause of serious outbreaks in domestic fowl in Australia since the 1930s. *Pasteurella multocida*, was isolated from two deceased wild waterbirds in Victoria, Australia, in 2013 (Allen et al. 2024) and has been described in farmed chickens from Queensland, and more recently in feedlot cattle and in pigs across a broader area of the continent. More recently *P. multocida* has emerged as a disease of major economic concern in the Australian free-range layer industry (Singh et al. 2013). *Pasteurella multocida* is a cause of septicaemia, respiratory diseases, and soft tissue infections in a wide range of hosts, including birds, pigs, ruminants, rabbits, companion animals, and humans (Allen et al. 2024). Infection by a virulent strain usually leads to acute septicaemic disease with high morbidity and mortality. Birds may die within a few hours of the onset of clinical signs (Friend 1999a). Clinical signs typically are neurological or respiratory in origin. However, the disease may also occur in a chronic form. Birds with chronic avian cholera constitute a major reservoir of disease in a population (Songer and Post 2005). Gross and histological findings include haemorrhage, intestinal hyperemia, hepato- and splenomegaly, and necrotizing hepatitis and splenitis containing intralesional Gram-negative coccobacilli (Stidworthy and Dent 2018). Transmission is primarily through Inhalation and ingestion of contaminated water and food sources (Samuel et al. 2007). Diseased birds contaminate their environment, food and water mainly via respiratory excretions. Dead birds and fomites including equipment used by humans may also serve as sources of infection. Carcasses from birds that have died of avian cholera that remain in the environment may play an important role in maintaining outbreaks, as some epizootics have ceased once affected bodies have been removed (Allen et al. 2024). Predator attacks: Non-fatal predator attacks from wild or domestic animals (dogs, cats, are known to be carriers of high amounts of the bacterium in their oral cavities and underneath their nails). Any chicken or bird that has been in a predator's mouth or scratched by a predator should be treated immediately with appropriate antibiotics. Transmission may also occur through the inhalation of airborne water droplets when birds take flight and possibly through mechanical transfer by biting arthropods that feed on birds after having fed upon contaminated carcases or contaminated environments. Bacteria are released into the environment by dead and dying birds, by live birds carrying the disease or from contaminated objects (e.g. cages, equipment and clothing).		
Risk assessment		
Entry assessment	Exposure assessment	Consequence assessment
P multocida is intermittently present in poultry farms (Agriculture Victoria, 2020), especially free-range farms, where wild birds (waterbirds, raptors,) visit and then return to Phillip Island (Millowl) (Singh et al. 2014). PINP LPs likely to be	Factors that may precipitate epizootics include high host density, habitat degradation, drought, presence of shallow or stagnant water, and inclement weather (Samuel et al. 2007). Any other stressful event may result in carrier birds to start shedding *P. multocida*	Based on overseas experience In Southern Rockhopper, Adelie, King and Macaroni penguins, disease could be devastating for LPs, wild birds, as well as poultry. Causes significant mass mortality of poultry and can affect future viability of poultry flocks. Not considered a high risk

susceptible given other penguin species susceptibility and reported epidemic mortality. Kelp gulls, which are present all year round within the penguin colony at Phillip Island (Millowl) (Dann 2007), have been shown to be important vectors for cholera outbreaks in other species of penguins (Leotta et al.2006). The greatest risk of cholera incursion is likely to come from kelp gulls that travel from the island to mainland sites, where it is possible for them to have contact with domestic chickens in both commercial and private settings (Scheelings 2022).	organisms into the environment (Wobeser 1997). LPs may be infected but non-clinical and become clinical and commence shedding if significant stressors prevail. LPs forage in water, congregate in groups (high host density) when exiting the ocean and walking up the beach to nesting sites, mate and rear offspring and if infected could excrete *P. multocida* if stressed through inhalation, and ingestion. *P. multocida* is highly infectious and spreads easily from sick and dead birds and continues to contaminate the ecosystem. The bacterium can be highly pathogenic leading to large numbers of dead birds and birds shedding large numbers of organisms into the environment. The organism may also persist for long periods (up to 7 weeks) in the environment therefore birds may be persistently exposed (Samuel et al. 2007). The likelihood of exposure to other avian sp. is probably medium at nesting sites. Other reported sympatric avian species include the short tailed shearwater, pacific gull, kelp gull, silver gull, crested tern, Cape Baron goose, Purple swamphen, Sooty oystercatcher and the Hooded plover. These species are all likely susceptible to exposure and have a medium to high likelihood of developing disease. Humans can also be exposed from an animal bite or scratch, mainly from domestic dogs and cats. Infections can also arise through inhalation of bacteria which is most likely to happen in confined areas of air movement where a large amount of infected material is present (e.g. during disease control operations).	disease for humans although infections are not uncommon. Potential for significant economic impacts on the poultry industry through mass mortality of birds. However, there are no reports of avian cholera in any populations of Australia penguins, and its prevalence in these species is currently unknown (Scheelings 2022). Kelp gulls, which are present all year round within the penguin colony at Phillip Island (Millowl) (Dann 2007), have been shown to be important vectors for cholera outbreaks in other species of penguins (Leotta et al.2006) and co-habit LP nesting sites. Stressors and co-infections may also play a role in promoting overt disease. Therefore the overall risk of *P. multocida* infection is MEDIUM.
Risk evaluation		
Preventative measures should be employed to reduce the disease risks.		
Risk management options		

Diagnosis: Diagnosis is based on a combination of history (mass mortality of wild birds), pathological lesions, and isolation and identification of the organism. On necropsy *P. multocida* can be cultured from heart blood and most organs. Tissue samples, especially liver, intestine and heart must be collected both fresh and fixed. Oropharyngeal swabs should be collected from live birds (Samuel et al. 2007). Polymerase chain reaction (PCR), and multi-locus sequence typing (MLST) are commonly used to further characterise the nature of *P. multocida* infections (Jaeger et al. 2018). Strain typing is essential to understanding the sources and modes of transmission of *P. multocida* in animal populations (Allen et al. 2024).

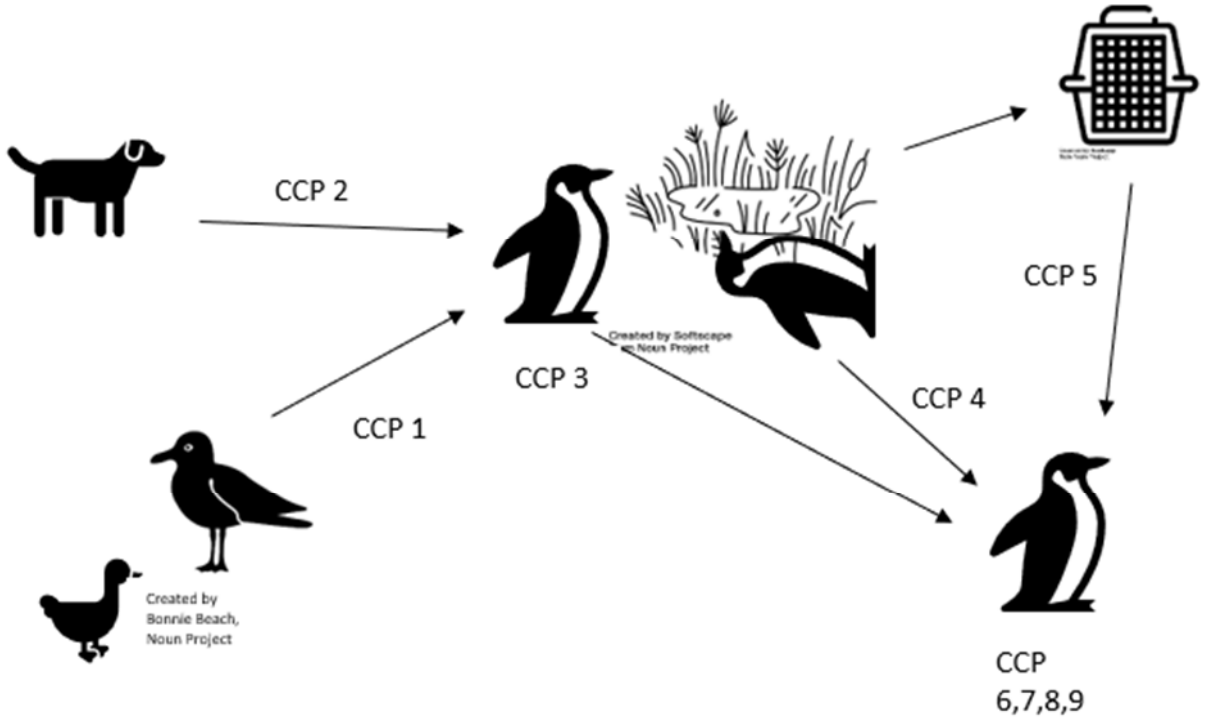


Figure 8. Critical control points for the hazard *Pasteurella multocida*

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	High	High	Gulls, short-tailed shearwater and waterfowl are highest risk group for carrying, shedding *Pasteurella* and infecting penguins. Passive surveillance of relevant populations on the peninsula can lead to preventative actions such as removing affected carcasses to minimise access to penguins and other seabirds.	Y
2	Minimise predation risk	Low	High	Predators carry the bacteria in their oral flora and can infect individual birds (which could lead to outbreak). Penguin colony is already quite well protected from most important predators (foxes, feral dogs). However, not thought to be a major route of infection in wild birds (USGS Field Manual).	Y
3	Active surveillance of live birds	Low	Low	Disease can be transmitted through direct contact between conspecifics. Therefore, early detection in live birds could be used as an early warning system. However, this is likely to be not cost effective, with most surveyed birds negative. Passive surveillance of dead penguins is a far more effective strategy.	N

4	Passive surveillance and removal of any dead carcasses	High	High	It is important to catch outbreaks early, because dead birds create an infection source for the rest of the population. -Remove carcasses before there is a major arrival of scavengers which may spread the disease further. -Take care to ensure these measures do not cause the dispersal of infected birds out of the area. -Take care to avoid contaminating new areas whilst carcases are being transported to the laboratory and disposal site. - Disinfect field equipment used in infected areas. -Scavengers and predators can be attracted away from infected areas to other feeding sites using other food sources such as road killed carcases. -These actions need careful evaluation of bird movement patterns and of the disease cycle to assess whether they are suitable. - Moving infected or potentially infected birds from one geographical location to another is not advised (USGS 2015)	Y
5	Biosecurity	High	High	In case of an outbreak, Pasteurella can be carried on fomites (cages, car tyres, clothing etc); high biosecurity and appropriate decontamination is therefore essential; potential spread through tourists will need to be considered (USGS 2015)	Y

6	Biosecurity & minimising contact with poultry	High	High	Strict biosecurity & PPE to be worn by any staff which have poultry at home. The disease in livestock may be avoided by employing good sanitation and animal management practices. -Prevent the introduction of infection through movement controls, testing and quarantine. -Detect any infected animals in the population as early as possible through surveillance and thoroughly investigate all suspect cases. -Vaccination with an approved vaccine can be effective.	Y
7	Human biosecurity	High	High	Wear gloves and thoroughly wash exposed skin surfaces after any contact with contaminated birds. -Process infected birds outdoors or in a well-ventilated area.	Y
8	Treatment	Moderate	Low	Theoretically, affected birds can be treated with antibiotics, however, many birds die peracutely, and treatment of large numbers of affected birds may be challenging (though perhaps not impossible). They would need to be housed during treatment.	N
9	Vaccination	High	Moderate	Vaccination of albatross chicks on Amsterdam Island (Bourret et al. 2017) was undertaken to reduce the epidemic mortality reported in chicks. A killed vaccine was used on chicks 2 weeks post hatching caused 100% seroconversion and reduced the death risk by a factor exceeding 2.5, raising fledging probability	consider

				from 14% to 46%. These results suggest that using a specifically tailored vaccine could be a key tool to effectively protect endangered seabirds from disease outbreaks threatening them with extinction.	

4.4.6 Disease risk assessment for *Babesia*

Justification of hazard		
Babesia spp. are tick-borne protozoan parasites, and 16 avian-infecting species have been described, including one species (*Babesia peircei*) that infects penguins (Vanstreels et al. 2018). van Rensburg (2010) observed round intracytoplasmatic inclusions compatible with *Babesia* sp. in the blood smears of LPs at Tiritiri Matangi Island (9/79 = 11.4%) (Hauraki Gulf, NZ). *Babesia* sp. was identified in seven wild LPs, with positive individuals recorded in New South Wales, Victoria and Tasmania with prevalence estimated between 3.4% and 4.5%. Gene sequencing confirmed the identity of the parasite and demonstrated close relatedness to *Babesia poelea* from boobies (*Sula* spp.) and *B. uriae* from murres (*Uria aalge*). (Vanstreels et al. 2015). A recent surveillance study of Tasmanian LPs suspected 70 out of 300 samples to be positive, though only 4 were confirmed through sequencing (Wells, M. pers. comm.). Sequencing revealed the same species as that recorded in Australian LPs by Vanstreels (2015), as well as being consistent with the species found in red-billed gulls, white-fronted terns and Australasian gannets in New Zealand (Paparini et al. 2014). A review of Melbourne Zoo medical records showed that of the 148 little penguin admissions over the past 10 years, *Babesia* has been identified in three individuals, with a fourth individual had a concurrent *Plasmodium* and *Babesia* infection. This indicates that haemoparasitism with Babesia may not be a major factor for hospital admissions of little penguins in Victoria (Scheelings 2022) None of the Babesia-positive penguins in Vanstreels et al. (2015) study presented with signs of disease, confirming earlier suggestions that chronic infections by these parasites are not clinically significant to otherwise healthy LPs. However, Babesia infection may lead to mild anaemia in LPs (Sergent et al. 2004) and is associated with mild anaemia, leukocytosis and impairment of hepatic function in African penguins (Parsons et al. 2016). Overall, the pathogenicity of Babesia infection in birds is still unclear. Transmission is seasonal and dependent on the vector's population. It is generally thought that hard ticks (Ixodidae) are the most relevant invertebrate hosts of avian *Babesia* spp., but soft ticks (Argasidae) are thought to play a significant role for colonial ground-nesting birds (Peirce 2000). The hard tick *Ixodes kohlsi* may play a key role in the transmission to LPs at New South Wales, Australia (Cunningham et al. 1993) and both soft and hard ticks were observed on *Babesia*-infected little penguins in Tasmania, Australia (Vanstreels et al. 2015). In Australia vectors are typically active during spring and summer. Birds are infected by the parasites' sporozoites when they are bitten by the infected tick vector. The lifecycle of *Babesia* consists of asexual multiplication in the red blood cells of birds, and in gametogony followed by sporogony in the salivary glands of ticks (Ebani and Mancianti 2021).		
Risk assessment		
Entry assessment	Exposure assessment	Consequence assessment
Babesia has a high likelihood of already being present in the PINP colony. It may already be endemic.	If PINP LPs are already infected with *Babesia* and a competent tick vector exists there is a high likelihood of exposure to other sympatric bird species present at nesting sites such as the short tailed shearwater, pacific gull, kelp gull, silver gull, crested tern, Cape Baron goose, purple swamphen, sooty oystercatcher and the hooded plover. These species are all likely susceptible to exposure if a competent vector is present and have a medium to high likelihood of developing infection and disease although they may also be endemically infected.	Highly likely that *Babesia* is already present in the Phillip Island (Millowl) population. Consequences of infection to the population may range from subclinical to mild including anaemia and leukocytosis to more significant for example affecting hepatic function. However, it is important to note that the effects of infection may be heightened if birds become compromised for some other reason, i.e. concurrent illness or environmental stressors.
Risk evaluation		

Preventative measures should be employed to reduce the disease risks.

Risk management options

Diagnosis:

Diagnosis of haemoparasitism in is based on a combination of clinical signs, demonstration of parasites within erythrocytes, and utilisation of molecular techniques such as PCR to identify presence of parasite genetic material.

Vanstreels et al. (2015) reported using two nested PCR targeting the 18S rRNA gene post direct microscopy of Giemsa-stained blood smears (the traditional method of diagnosing avian Babesia infection)

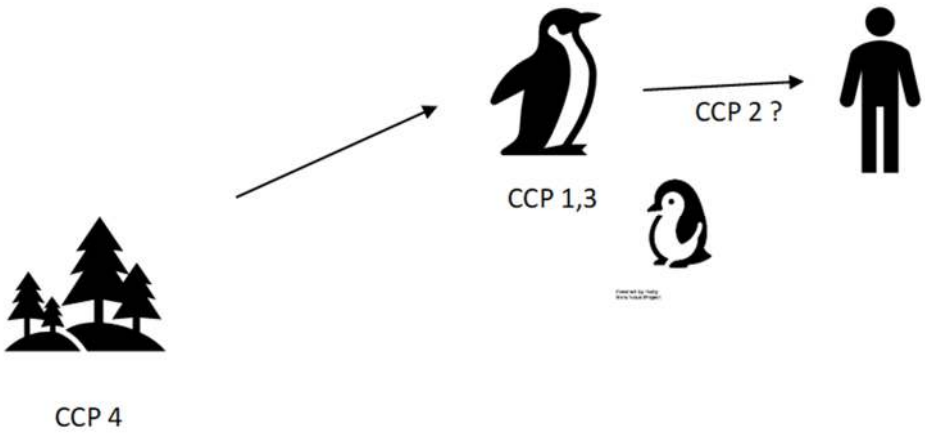


Figure 9. Critical control points for the hazard *Babesia* sp.

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Infection prevalence unknown. It would be beneficial to conduct *Babesia* passive surveillance via PCR, blood smear and serology. This will enable a current	Y

				prevalence estimate in the population which will inform the need for prioritising ongoing management actions.	
2	PPE	High	High	Personal precautions for humans can include avoiding tick infested areas, using repellents, wearing light coloured clothing and regularly checking for ticks although *Babesia* infecting penguins is not known to cause zoonotic disease.	Y
3	Stress reduction	Moderate	Moderate	Reducing stressors where feasible in infected birds as physiological stressors (moult, chick rearing, or poor husbandry) and environmental stressors can increase clinical effects.	Y
3	Environmental strategies	Low	Medium	Modification of the habitat to decrease humidity through clearing of vegetation and chemical control of ticks in the environment may also be used	N

4.4.7 Disease risk assessment for *Plasmodium* sp.

Justification of hazard		
Host range – *Plasmodium* sp. is an avian haemosporidian, protozoan parasite with a global distribution. It infects red blood cells and is commonly reported in many species of birds in Australia. This vector-borne parasite is spread by mosquitoes and has a variable significance depending on environmental, host, mosquito and parasite factors (Ings and Denk 2022). Infection can also occur in poultry species. Seven genera of haemoparasites have been reported to infect penguins (Scheelings 2022). Infection with *Plasmodium* sp, has been documented in 13 species of penguins in both wild and captive settings, including king, Humboldt, African, northern rockhopper, yellow-eyed, Snares, chinstrap, little, gentoo, Macaroni, Galapagos, and southern rockhopper (Vanstreels et al. 2016). In a molecular screening of LP ticks (*Ixodes kohlsi)* from PINP, evidence of apicomplexa was reported, however, the genus and species were unknown (Ghafar et al. 2023). *Plasmodium* spp. have not yet been reported from Tasmania (Iutruwita) (Wells, M. pers. comm.). In wild LPs in the islands in the Gulf of St Vincent, South Australia Burt et al. (2016) reported that *Haemoproteus* spp. was seen in 98% of individuals, and *Plasmodium* spp. detected in 82% of penguins. Interestingly, 80% of all penguins had multiple infections with both *Plasmodium* and *Haemoproteus.* There are increased reports of frequency of occurrence in New Zealand penguins (B Gartrell pers comm, Nov 2023). Infections with multiple species and genera of haemosporidia are common and infections may persist for years, although a percentage of infected birds will cure themselves of infection (van Rooyen et al. 2013). These parasites have historically been considered to be host-adapted and to cause little disease in the species that they infect (WHA 2013). Infection is endemic in large parts of the world and is typically subclinical in birds that are native to these regions. Several penguin species have evolved in non-endemic regions without the selective pressure that these parasites exert and are highly susceptible to infection when moved to endemic regions, for example, in the context of zoological collections or rehabilitation centers (Ings and Denk 2022). In penguins, avian malaria (*Plasmodium*) is one of the most significant parasitic diseases, with mortality rates as high as 50%–80% (Grilo et al. 2016). Disease is common in captive penguin colonies and rehabilitation centers and also occurs in the wild (Stidworthy and Denk 2018). Disease risk is highest in chicks, juveniles and naive adults, and in animals with outdoor access. Stressors (moult, chick rearing, or poor husbandry) increase mortality. Clinical signs range from acute death to lethargy, anorexia, depression, vomiting, dyspnoea, pale mucous membranes (anaemia), and behavioural separation. Severe forms induce neurological signs including motor incoordination, seizures, and paralysis. Antemortem diagnosis is difficult as animals frequently succumb rapidly without detectable blood parasitaemia (Stidworthy and Denk 2018). Fatal infection is most commonly associated with *Plasmodium relictum* or *P. elongatum*, but cases involving *P. cathemerium, P. juxtanucleare, P. tejerai, P. nucleophilum*, and *P. unalis* have been reported (Clarke and Kerry 1993). In birds that survive, periods of recrudescence during spring or breeding season have been reported (Atkinson 2008a). Transmission is seasonal and dependent on the population of vectors. In Australia vectors typically active during spring and summer. Birds are infected by the parasites' sporozoites when they are bitten by the infected insect vector the mosquito (Culicidae), particularly *Culex* spp., *Mansonia* spp., *Culiseta* spp. and *Aedeomyia* spp. (Vanstreels et al. 2016). The geographic distribution of *Culex* mosquitoes overlaps with the breeding habitat of penguins in Peru, Chile, Namibia and Australia, and it is therefore plausible that wild penguins in these countries may be infected by *Plasmodium* sp. (Vanstreels et al. 2016).		
Risk assessment		
Entry assessment	Exposure assessment	Consequence assessment
Plasmodium has a high likelihood of already being present in the PINP colony. It may already be endemic.	If PINP LPs are already infected with *Plasmodium* and a competent vector exists there is a high likelihood of exposure to other sympatric bird species present at nesting sites such as	Highly likely that *Plasmodium*, the agent of avian malaria, is already present in the Phillip Island (Millowl) population. However, the effects of infection may be heightened if

	the short-tailed shearwater, pacific gull, kelp gull, silver gull, crested tern, Cape Baron goose, purple swamphen, sooty oystercatcher and the hooded plover. These species are all likely susceptible to exposure if a competent vector is present and have a medium to high likelihood of developing disease although they may also be endemically infected.	birds become compromised for some other reason, i.e. concurrent illness or environmental stressors. Consequences of infection to the population may range from mild or subclinical to significant mortality. Factors such as climate change, expansion of the range of some species, and possibly the introduction of new haemosporidial species into Australia may have contributed to outbreaks, may predispose to an increased prevalence of infection and, additional outbreaks of disease in the future (WHA 2013). An understanding of current prevalence is required.
Risk evaluation		
Preventative measures should be employed to reduce the disease risks.		
Risk management options		
Diagnosis: Diagnosis is based on a combination of clinical signs, demonstration of parasites within erythrocytes, and utilisation of molecular techniques such as PCR to identify presence of parasite genetic material. Ante mortem diagnosis is challenging as sudden death is commonly reported. Direct microscopy of Giemsa-stained blood smears has been the traditional mean of diagnosing avian malarial infection—intraerythrocytic merogony is not seen in other Haemosporidia and is therefore pathognomonic. However, this method is often not diagnostic in penguins, which frequently fail to demonstrate parasitemia in the course of acute disease (Ings and Denk 2022). PCR techniques are not currently commercially available and involve nested PCR targeting the Cyt-B subunit to detect avian malaria and has a significantly improved sensitivity compared to direct microscopy. ELISA-based testing methods are valuable in assessing exposure at a population level, in observing infectious trends over population, time, and geography; and are sensitive in diagnosing chronic disease. However, the typically acute nature of fatal disease in penguins does not allow sufficient time for the bird to generate a humoral response and therefore renders ELISA testing less useful in the context of an acute disease outbreak (Sallaberry-Pincheira et al. 2015). Often diagnosed post-mortem where the most common findings include splenomegaly, hepatomegaly, pulmonary oedema, hydropericardium, pneumonia, and/or myocarditis and splenitis in animals in good body condition (Ings and Denk 2022). Impression smears from the liver, spleen, heart, and lungs may detect extraerythrocytic meronts and histopathology may detect a mononuclear hepatitis with intralesional protozoa (Stidworthy and Denk 2018).		

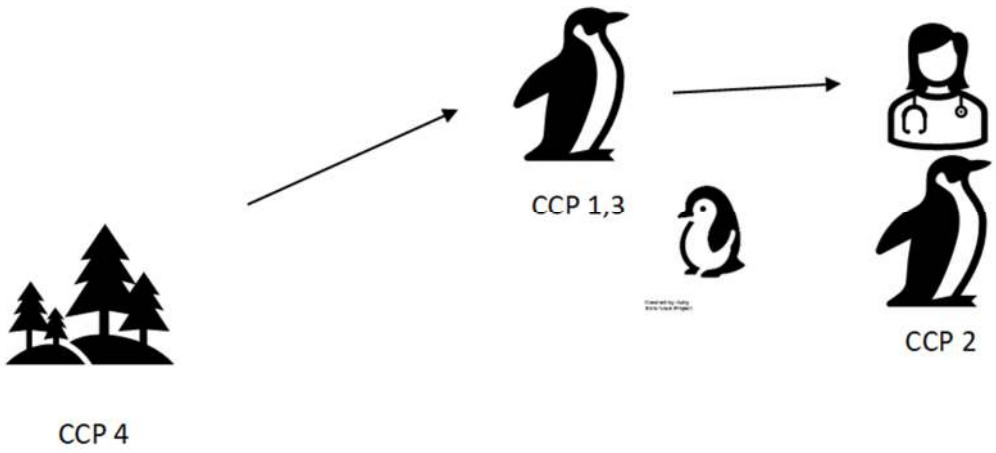


Figure 10. Critical control points for the hazard *Plasmodium* sp.

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Infection prevalence unknown. It would be beneficial to conduct *Plasmodium* passive surveillance via blood smears, PCR and serology. This will enable a current prevalence estimate in the population which will inform need for prioritising ongoing management actions.	Y
2	Treatment	Low	High	In wild birds, treatment may only be attempted in the rehabilitation setting. Therapy that has been used includes the use of chloroquine and primaquine to suppress erythrocytic stages. Posology in treatment protocols is based on empirical	Y

				evidence since studies on the pharmacokinetics and pharmacodynamics of anti-malarial drugs in penguins are lacking. Atovaquone and proguanil (Malarone junior®) and primaquine have been recommended (Meister et al (2021) and a clinical trial in Yellow eyed penguins has commenced, contact details below arlene.mcdowell@otago.ac.nz	
3	Stress reduction	Moderate	Moderate	Reducing stressors in infected birds where feasible as physiological stressors (moult, chick rearing, or poor husbandry) and environmental stressors can increase mortality.	Y
4	Environmental strategies to reduce insect vectors	To be determined	To be determined	Rising global temperatures are anticipated to increase the threat that avian malaria infection poses to penguins through increases in vector abundance. Mosquito control measures have been suggested, such as the introduction of infertile or malaria-resistant mosquitoes, and the alteration of the environment to reduce the possibility of larval development (Birdlife International 2020). Landscape-level mosquito control through the Wolbachia incompatible insect technique, which is a form of mosquito birth control has commenced to manage endangered Hawaiian honeycreepers. However, it would need to be implemented continuously to maintain its effect on mosquito populations (Hawaii Department of Land and Natural Resources, Division of Forestry and Wildlife 2023).	N

4.4.8 Disease risk assessment for *Toxoplasma gondii*

Justification of hazard		
The protozoan parasite *Toxoplasma gondii* is a global ubiquitous organism with a broad mammalian and avian host range (Greiner and Ritchie 1994) including humans. *T. gondii* commonly infects Australian wildlife species and can cause fatal infections in wild birds (Dubey et al. 1991). Toxoplasmosis can also be clinically severe in pigeons and canaries (Dubey 2002). Episodes of blindness and encephalitis were reported in several birds in a flock of 40 canaries in Victoria, Australia (Lindsay et al 1995). Mason et al. (1991) reported an acutely fatal infection resulting in marked hepatitis and splenitis, in a LP from Tasmania. Toxoplasmosis has also been reported to cause fatal infections in juvenile African penguins (Ploeg et al 2011). Ten cases of toxoplasmosis among 1,300 submissions in 12 years to the Taronga Zoo Pathology Collection suggests this protozoan disease is of minor significance in Australian native birds. However more recently disease was retrospectively reported in 12 wild LPs on necropsy exam from the Penguin Island colony WA with concurrent *Haemoproteus* infection (Campbell et al. 2019). A recent survey of Tasmanian LPs found a low seroprevalence to infection of <3% (Wells, M. pers. comm.). Infection is often subclinical. When signs do occur, they may include lethargy, fluffed appearance, reduced appetite, diarrhoea, disorientation and seizures. Sudden death without prior signs of illness is a common presentation. Development of disease may be influenced by host species and infective dose. In LPs on necropsy exam birds were in good body condition with reported hepatomegaly and splenomegaly, multifocal hepatic and splenic necrosis and numerous, 1–2 µm diameter protozoan parasites within the necrotic foci (Campbell et al. 2019). Similar to the reported necropsy findings in LPs by Mason et al. (1991). Transmission typically occurs from ingesting oocysts in contaminated soil, plant or invertebrate matter. Cats are the definitive host shedding oocysts in faecal material and contaminating the surrounding environment, which is then ingested by the intermediate host. 90% of feral cats on Phillip Island (Millowl) carry the parasite toxoplasmosis (Adriaanse 2018). Despite this, a low level of environmental contamination with *T. gondii* oocysts on Phillip Island (Millowl) was found Adriaanse (2018). Recently a 24 hour cat curfew was introduced on Phillip Island (Millowl) in July 2023 to limit the movement of cats and subsequent contamination with Toxoplasma oocysts on the island. *T. gondii* infection is common in sea birds, and may be asymptomatic, few develop disease. *Toxoplasma gondii* oocysts from feline faeces can be washed into sewage and freshwater run-off and contaminate marine waters (Dubey et al. 2021). A single cat can excrete millions of oocysts that can remain viable in the environment for months under natural conditions (Dubey 2010). *Toxoplasma* infection is also not uncommon in marine mammals for similiar reasons, disease can be transmitted by contamination of waterways with cat faeces (Jones and Dubey 2010). Filter-feeding fish may also take up *Toxoplasma* oocysts from contaminated water, and mice can become infected when feeding on these fish (Mason et al. 1991). Pilchards can also take up *Toxoplasma* cysts and transmit disease (Mason et al. 1991), and this is an important prey species for the LPs of Penguin Island, WA and a likely source of infection reported by Campbell et al (2019). Alternatively it has been shown (Campbell et al. 2019) that *Toxoplasma* can aggregate on extracellular polymeric substances in marine environments, enhancing the efficiency with which the organism could enter the marine food chain in coastal ecosystems (Shapiro et al. 2014). In humans, exposure to *T.gondii* is frequent, with estimated exposure rates of between 30 and 35% in the general population (Robert-Ganganeux et al. 2012).		
Risk assessment		
Entry assessment	Exposure assessment	Consequence assessment
There is a medium likelihood that PINP LPs are already subclinically infected with *T.gondii* owing to the presence of cats, and the environmental resistance of oocysts. LPs may ingest oocysts through the food chain (for example pilchards),	Other avian sympatric species can only become infected by ingesting the infected intermediate host (the LP). This is unlikely in non-carnivorous bird species.	Moderate likelihood of exposure, low number of cats present on Phillip Island (Millowl), however, other transmission pathways for example ingestion of filter feeding fish may exist as reported in Campbell et al. (2019). LPs only capable of transmitting this parasite by

or through contaminated water in the marine environment.		being eaten, therefore the risk to humans and non-carnivorous animals is negligible. Moderate consequences of disease for LP population. Of low consequence to other identified populations at risk.

Risk evaluation

Preventative measures should be employed to reduce the disease risks.

Risk management options

Diagnosis:

Serology is useful to detect exposure and presence of a rising titre if clinical evidence of infection. The MAT is most useful because it is sensitive, specific, does not require special equipment, and works well with all species of birds tested. A titer of >1:25 is considered positive for *T. gondii* infection.

T. gondii DNA can be detected by using *T. gondii*-specific primers with PCR. However, in most cases, the diagnosis will be made by histologic examination of birds submitted for necropsy examination and in most cases the tissues have already been fixed in buffered neutral 10% formalin. A preliminary diagnosis can be made by examining Giemsa-stained impression smears of affected tissues. T. gondii tachyzoites in smears are crescentic to globular, depending on the stage of division. However, in histologic sections tachyzoites are globular to oval and about half of the size of those in smears (Dubey 2001). Immunohistochemical staining with *T. gondii* specific antibodies can also aid diagnosis.

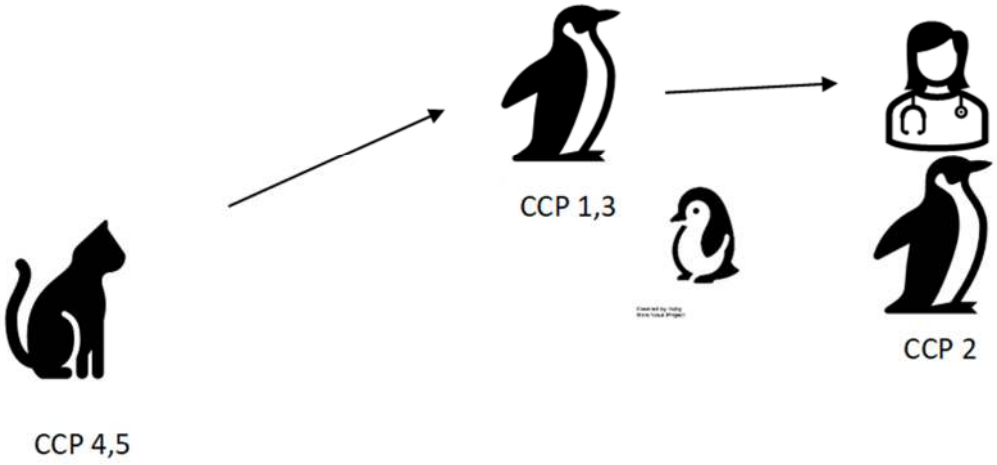


Figure 11. Critical control points for the hazard *Toxoplasma gondii*

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Infection prevalence unknown. It would be beneficial to conduct *T.gondii* passive surveillance via PCR and MAT serology. This would enable a current prevalence estimate in the population which will inform need for prioritising ongoing management actions.	Y
2	Treatment	Low	High	In wild birds treatment may only be attempted in the rehabilitation setting. There are no pharmacokinetic studies conducted in penguins for anti-protozoals and no case reports describing treatment regimes in seabirds. Diclazuril (1 mg/ml) (Janssen Pharmaceutica, Beerse, Belgium) has been used in 'Alala (*Corvus hawaiiensis).* The dosage regimen consisted of a loading dose of 10 mg/ kg orally once a day for three days (days 44– 46) followed by a single dose orally (10 mg/kg) every other day (days 48, 50, 52, 54, 56) (Work et al. 2000). In water treatment with trimethoprim and sulfadiazine for two weeks was prescribed to canaries (Williams et al. 2001). However in wild birds once detected clinically will likely be severely compromised and euthanasia should be considered from a quality of life perspective.	N

3	Stress reduction	Moderate	Moderate	Reducing stressors in infected birds where feasible as physiological stressors (moult, chick rearing, or poor husbandry) and environmental stressors can increase mortality.	Y
4	Eradication of feral cats through culling.	High	Low	Will eliminate risk of exposure to infective oocysts. Likely to be difficult and labour intensive	Y if can secure funding
5	Reduce cat density through culling to reduce environmental contamination	Moderate	Moderate	Reduce risk of exposure to infective oocysts	Y

4.4.9 Disease risk assessment for *Contracaecum* sp. infection

<table>
<tr><th colspan="3">Justification of hazard</th></tr>
<tr><td colspan="3">Important parasite of fish-eating mammals and birds (Norman 2005). Reported in multiple species; rockhopper, gentoo, macaroni, yellow-eyed, little, emperor, and Magellanic penguins as well as pelicans, cormorants and shags, gannets, boobies, darters, grebes, albatrosses, mollymawks, and shearwaters (Stidworthy and Dent 2018). Often reported on post-mortem exam in emaciated LP submissions (PINP UM 270404, DPI 220808) presumably in cases of starvation. Reported in Australian and New Zealand LP populations. Contracaecum spp are also of zoonotic importance, however there has only been one report in Australia of human infection with Contracaecum larvae (Shamsi and Butcher 2011). Infection with this parasite likely varies based on dietary preferences.

Obendorf and McColl (1980) reported 75% nematode (Contracaecum) prevalence with heavy burdens and severe, chronic and acute gastric ulceration in association with poor body condition of wild LP. Similar findings were recorded by Norman et al. (1992) and Harrigan (1992), with even small numbers of nematodes causing small to medium sized gastric ulceration. Contracaecum, has been implicated as the cause of severe parasitic disease and mortality (Obendorf and McColl 1980) and appears to be exacerbated when combined with other forms of stress such as starvation or inclement weather (Obendorf and McColl 1980, Norman 1992). Norman (1998) necropsied LPs dying on Phillip Island (Millowl) between 1992 and 1995 and found the prevalence of infection amongst unfledged penguins was 98% (mean intensity 56.5; range: 1–244; S.D. 55.1; n=112) and the prevalence of lesions was 77%. The prevalence of infection amongst fledged penguins was 52% (mean intensity 35.8; range: 1–374; S.D. 69.9; n=114) and the prevalence of lesions was 61%. Therefore, Contracaecum is an infection of particular significance to unfledged young likely acquiring infection from ingestion of infected regurgitated material when fed from parents. Widespread LP wrecks involve large numbers of juvenile birds which died either directly from parasitic infection (e.g. gastric ulceration) or indirectly from parasite associated effects such as starvation (due to obstruction of the gut). Juvenile LP appear to be more susceptible to parasite infestation and associated pathogenic effects (Harrigan 1992). Although parasites have been shown to be an annual factor in the mortality of young LP, the severity varies from year to year (Norman 1992). Environmental conditions such as adverse weather may be an important stressor causing sudden high mortalities in juveniles affected by parasitic disease (Harrigan 1988). Alternatively, environmental fluctuations may alter the suite of endoparasites through differential effects on prey species (van Rensburg 2010).
Adults are likely infected by ingestion of parasites of the genus Contracaecum Railliet et Henry, 1912 (Nematoda: Anisakidae) which have several species of fish as intermediate or paratenic hosts, with piscivorous birds and mammals as definitive hosts (Anderson 2000).</td></tr>
<tr><th colspan="3">Risk assessment</th></tr>
<tr><td>Entry assessment</td><td>Exposure assessment</td><td>Consequence assessment</td></tr>
<tr><td>Adult LPs infected by ingestion of parasites of the genus Contracaecum which have several species of fish as intermediate or paratenic hosts. Juvenile young likely acquire infection from ingestion of infected regurgitated material when fed from parents. Moderate likelihood of exposure given parasite presence in fish in the waters surrounding Phillip Island (Millowl).</td><td>All piscivorous birds and mammals that forage in the water surrounding Phillip Island (Millowl) would be susceptible to ingestion and infection. The LP is a definitive host and would only pass on infection if scavenged and this would likely be by an unsuitable host.</td><td>LPs likely to commensally carry these parasites, however, may become immunocompromised by multiple agents, infectious and non-infectious, and develop disease. Low likelihood of carriage and transmission to other sea- and shorebirds as infection acquired through ingestion of intermediate host. Infection and disease mostly of low consequence although stressors may increase numbers of parasites and cause clinical disease of moderate population consequence exacerbated by starvation and / or inclement weather. Effectively managed in humans by hygiene practices.</td></tr>
</table>

Risk evaluation

Preventative measures should be employed to reduce the disease risks.

Risk management options

Diagnosis

Typically, on necropsy and histopathological exam through gross examination, light microscopy and electron microscopy. Gastric ulcerative processes of the oesophagus and stomach caused by *Contracaecum* sp. have been described in LPs (Obendorf and McColl 1980). Typically diagnosed as a fulminant cause of death only if associated perforation leading to coelomitis. The smallest lesions were superficial foci of epithelial erosion or coagulative necrosis associated with a single nematode attached by its mouthparts to the gastric mucosa, or occasionally the oesophageal mucosa. Nematodes penetrated the compound tubular glands of the proventriculus and initiated proventricular gland abscesses. Proventricular or ventricular ulceration resulted from nematodes invading the lamina propria and submucosa. Chronic infection resulted in obliteration of compound tubular proventricular glands, with fibrosis and granulomas at the sites where nematodes were embedded in the tissue. Rarely, nematodes reached the muscularis or serosa. Healed lesions with scarring and distortion of the mucosa were sometimes observed in mature penguins (Norman 1998). Diagnosis via faecal parasitological exam, consisting of a faecal flotation and looking for evidence of eggs ante-mortem indicated in penguins undergoing rehabilitation. Freezing of fish being fed out to penguins undergoing rehabilitation should kill any larval stages.

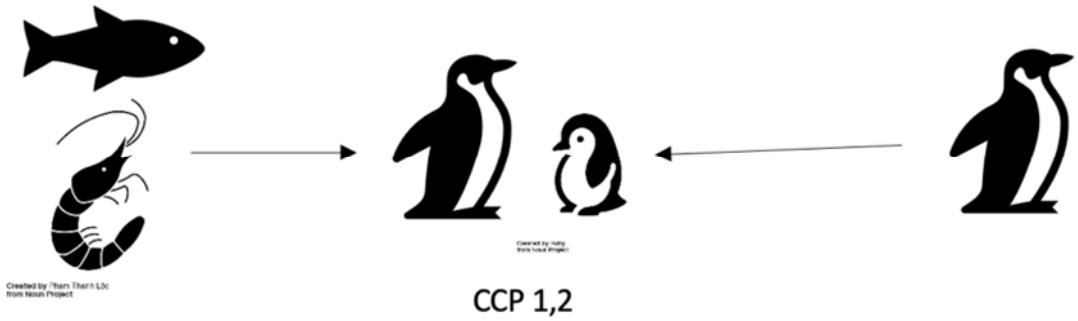


Figure 12. Critical control points for the hazard *Contracaecum*.

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Infection with *Contracaecum* is extremely prevalent in little penguins (between 50-98%) (Norman 1998). However, this prevalence data is now 25 yrs old and requires updating. Impacts on individuals can be significant, but in most cases probably don't lead to significant morbidity or mortality. It is highly likely that infection and lesions will be detected during passive surveillance, and there may be some value in that it can detect sudden increases in prevalence or severity. As passive surveillance is recommended for other diseases, it makes sense to also document infection with this parasite.	Y
2	Treatment	Moderate	High	It is theoretically possible to attempt treatment of penguins coming into care for infection with *Contracaecum* using conventional broadspectrum (e.g. albendazole, fenbendazole (Grimes et al. 1989) anthelminthics. It is thought that immune suppression/stress may exacerbate the impacts of infection, thus compromised penguins might benefit from treatment? However, more research would be needed to provide evidence that this is a useful strategy in LPs.	Y

4.4.10 Disease risk assessment for *Mawsonotrema eudyptulae* infection

<table>
<tr><td colspan="3">Justification of hazard</td></tr>
<tr><td colspan="3">Mawsonotrema eudyptulae is a trematode parasite, known to contribute to mortalities in Australian LPs in South Australia, Victoria and Tasmania (Norman 2005). Reported in Australian LP colonies in very high numbers and associated with significant liver enlargement, severe lesions and haemorrhage (Norman 1992). In post-fledglings, the liver fluke (trematode) appears to be pathogenic. The fluke, which lives in the bile ducts, sometimes kills large numbers of juvenile penguins, which are found as ‘wrecks’ of carcasses on beaches, known as ‘beach-wrecks’ (Obendorf and McColl 1980). Infection with this parasite likely varies based on dietary preferences. Unknown transmission but suspected to be through ingestion of intermediate host, likely from the marine habit and piscivory.</td></tr>
<tr><td colspan="3">Risk assessment
‘What is the likelihood and consequences of a specified hazard occurring within an identified pathway or event?’</td></tr>
<tr><td>Entry assessment</td><td>Exposure assessment</td><td>Consequence assessment</td></tr>
<tr><td>Adult LPs infected by ingestion of parasites of the genus Mawsonotrema eudyptulae which have several species of fish as intermediate or paratenic hosts and require obligatory participation of molluscs to complete their lifecycle (Galaktionov 2017). Moderate likelihood of exposure given parasite presence in fish in the waters surrounding Phillip Island (Millowl).</td><td>All piscivorous birds and mammals that forage in the water surrounding Phillip Island (Millowl) would potentially be susceptible to ingestion and infection. The LP is a definitive host and would only pass on infection if scavenged and this would likely be by an unsuitable host.</td><td>LPs likely to commensally carry these parasites, however, may become immunocompromised by multiple agents, infectious and non-infectious, and develop disease. Low likelihood of carriage and transmission to other sea- and shorebirds as infection acquired through ingestion of intermediate host. Infection and disease mostly of low consequence although stressors such as starvation and / or inclement weather may increase numbers of parasites and cause significant clinical disease but typically of moderate population consequence.</td></tr>
<tr><td colspan="3">Risk evaluation</td></tr>
<tr><td colspan="3">Preventative measures are required to reduce disease risk</td></tr>
<tr><td colspan="3">Risk management options</td></tr>
</table>

Diagnosis

Typically, on necropsy and histopathological exam through gross examination, light microscopy and electron microscopy. Angel (1973) describes typical microscopic findings required for definitive identification.

Diagnosis via faecal parasitological exam, consisting of a faecal flotation and looking for evidence of eggs ante-mortem indicated in penguins undergoing rehabilitation. Freezing of fish being fed out to penguins undergoing rehabilitation should kill any larval stages.

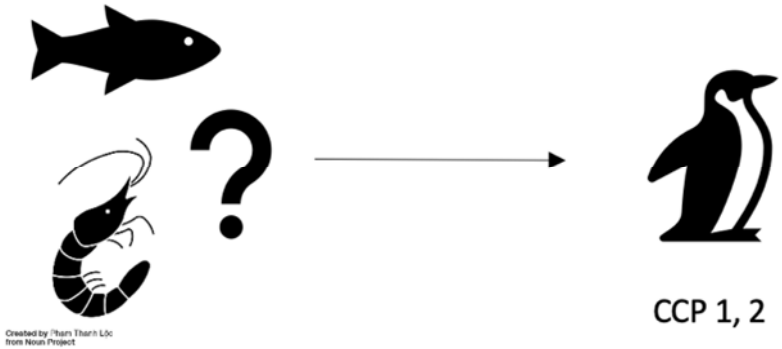


Figure 13. Critical control points for the hazard *Mawsonotrema eudyptulae*

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Infection with *Mawsonotrema* is highly prevalent in juvenile LPs. Impacts on individuals can be significant, but in most cases don't lead to mortality. It is highly likely that infection and lesions will be detected during passive surveillance, and there may be some value in that it can detect sudden increases in prevalence or severity. Noting prevalence data in not recent. As passive surveillance is recommended for other diseases, it makes	Y

				sense to also document infection with this parasite.	
2	Treatment	Moderate	Low	There is no published treatment for infection with Mawsonotrema. It is thought that immune depression/stress may exacerbate the impacts of infection. Therefore, compromised penguins for example in rehabilitation may benefit from treatment. However, more research would be needed to find treatment options and provide evidence that this is a useful strategy.	N?

4.4.11 Disease risk assessment for ticks

Justification of hazard		
Host range and impact –*Ixodes eudyptidis* is found in southern Australia and New Zealand in at least 17 seabirds including LPs and two land birds (Heath 2006). *Ixodes uriae* infection has been widely reported in both the northern and southern hemisphere in at least 52 seabirds including LPs and other sub-antarctic penguins in the subantarctic islands and Antarctic Peninsula (Mobendorf and McColl 1980). *I. uriae,* is regarded as a generalist tick affecting seabirds (Roberts 1970). Gauthier-Clerc et al. (1998) reported cases of death in adult King Penguins hyperinfested by the tick *Ixodes uriae*. The preferred feeding sites on the host were the head and the neck for adult and nymph ticks, and the lower parts for larvae. King Penguins carried up to 263 adults, 548 nymphs, or 3,500 larvae. This species of tick is also known to be the vector of a large number of arboviruses (Chastel 1980). *Borrelia burgdorferi* was isolated from *I. uriae* on Possession Island (Gauthier-Clerc et al. 1998). *Ornithodorus* sp. species are found in the tropical and temperate regions in Humboldt, Galapagos, African and LPs (Clarke and Kerry 1993). *Ixodes kohsli* was associated with eosinophilia, *Babesia*-like organisms and a severe regenerative anaemia (Spielman and Cunningham 1993) in LPs. Wells et al. (manuscript in review) found higher prevalence of ticks in natural nests of LPs in Tasmania, during moulting and the breeding season, and with lower burrow densitities. Wells et al. (manuscript in review) also found a positive association between heterophil/lymphocyte ratio and tick burdens. Little penguins on Phillip Island (Millowl) are heavily parasitised by Ixodes ticks (*I. eudyptidis* and *I. kohlsi*) when breeding. The penguins are also known to harbour *Babesia* spp. a protozoan parasite causing piroplasmosis in vertebrates, which is a common co-infection partner of *Borrelia burgdorferi* in North America (Moon et al. 2017). However large-scale sampling of the Phillip Island (Millowl) colony conducted by Moon et al (2017) strongly suggests that *Borrelia* is either absent or has an extremely low prevalence in little penguin ticks at this site. *Bothriocroton* ticks have also been reported in penguin burrows at Phillip Island (Millowl) Nature Park (Moon et al. 2017) suggesting echidnas and penguins on the island may share parasites and associated pathogens. Chicks are most frequently infected. The sites of attachment typically coincide with sparsely feathered regions such at the base of the bill, inside the external ears, crown of the head, neck and chin and may have associated irritation and oedema (Ladds 2009).		
Risk assessment		
Entry assessment	Exposure assessment	Consequence assessment
Ticks are already present in the PINP colony. High likelihood of exposure especially at nesting sites	There is a high likelihood of exposure to other sympatric bird species present at nesting sites such as the short-tailed shearwater, pacific gull, kelp gull, silver gull, crested tern, Cape Baron goose, purple swamphen, sooty oystercatcher and the hooded plover. These species are all likely susceptible to exposure and have a medium to high likelihood of acquiring ticks which may or may not harbour other disease causing agents.	Likelihood of exposure with low population consequence. Ticks may cause local irritation however importantly may also be associated with tick borne disease such as *Babesia* infection. Consequences of infection to the population may range from subclinical to mild including anaemia and leukocytosis to more significant for example affecting hepatic function. However, it is important to note that the effects of infection may be heightened if birds become compromised for some other reason, i.e. concurrent illness or environmental stressors. Tick parasitism may be an important negative factor for the health of adult King Penguins and, probably, of chicks, during their periods on

		land. Parasitism may also affect those foraging at sea to obtain food for their chicks (Kooyman et al. 1992).
Risk evaluation		
Preventative measures should be employed to reduce the disease risks.		
Risk management options		
Diagnosis: Ticks grossly evident on clinical examination. Definitive diagnosis can be conducted by light microscopy and assessment of morphology and parasite identification keys		

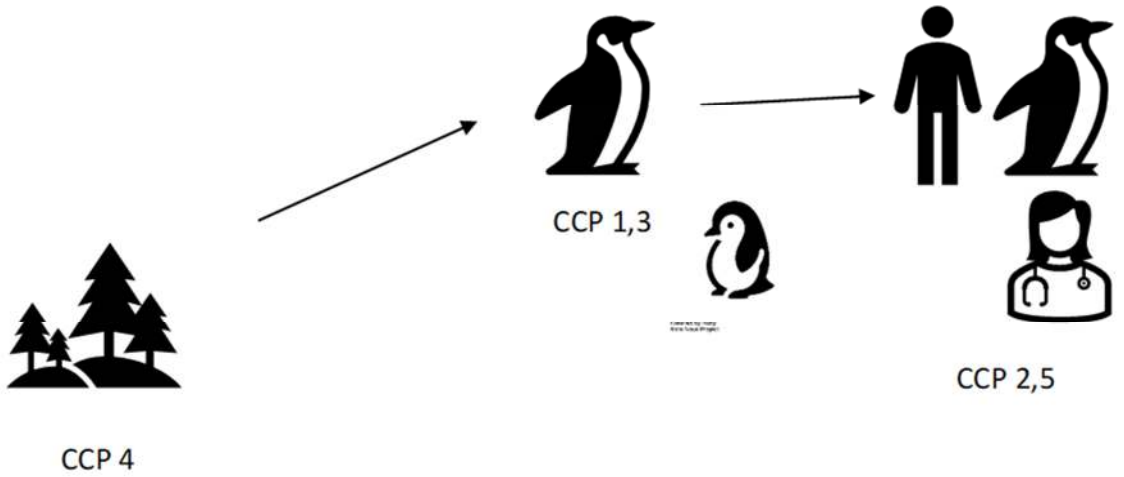


Figure 14. Critical control points for he hazard ticks

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	A full survey of ectoparasites including ticks would be beneficial to undertake including associated prevalence of infection with each tick species and severity of infection eg low, medium, high, and documenting sites of attachment	Y
2	PPE	High	High	Personal precautions for humans can include avoiding tick infested areas, using repellents, wearing light coloured clothing and regularly checking for ticks	Y
3	Stress reduction	Moderate	Moderate	Reducing stressors in infected birds where feasible as physiological stressors (moult, chick rearing, or poor husbandry) and environmental stressors can increase clinical effects.	Y
4	Environmental strategies	Low	Medium	Modification of the habitat to decrease humidity through clearing of vegetation and chemical control of ticks in the environment may also be used	N
5	Treatment	High	High	In a rehabilitation setting and if a high load causing clinical signs of disease ticks can be mechanically removed and/or treated with a topical acaricide	Y

4.4.12 Disease risk assessment for algal toxins

Justification of hazard
Marine biotoxins have been frequently implicated in morbidity and mortality events in multiple species of birds globally, with North America and Europe leading the reporting perhaps owing to increased monitoring in these regions (Ben-Gigirey et al 2021). However, overall the number of published studies confirming the presence of marine biotoxins from harmful algal blooms (HABs) in seabirds, remains low. Algal blooms (a fast-growing dense population of algae) typically result from an increase in nutrients (e.g. from storm water, run off from agriculture), and a combination of favourable environmental conditions (DOC 2022). The extent and degree to which HABs negatively affect marine organisms, such as seabirds, is related to the fate of algal derived secondary metabolites (toxins or bioactive compounds for example dinoflagellates, diatoms, raphidophytes, pelagophytes and haptophytes) in the ecosystem and the biological activity and bioavailability of those substances (Landsberg et al. 2014). Less than 2% of these blooms reportedly release toxins (LAWA 2023) however they can still cause direct or indirect mortalities by physical mechanisms e.g. clogging fish gills, covering beaches or depleting oxygen levels in the water as they naturally die off, sink to the seafloor and decompose, becoming detritus. HABs also disrupt the ecosystem (e.g. reducing light availability to benthic environment and reducing visibility for foraging animals) (LAWA 2023). Aquatic birds feeding predominantly on fish and invertebrates are susceptible to algal biotoxins generated during harmful algal blooms (Landsberg et al. 2007). Harmful algal toxins can be transmitted through the food web from zooplankton to different fish, marine invertebrates (gastropods, crustaceans, equinoderms, tunicates), seabirds, marine mammals, and humans (Ben-Gigirey et al. 2021). There are three main groups of marine biotoxins involved in seabird morbidity and mortality worldwide the brevetoxins (PbTXs), Paralytic shellfish poisoning (PSTs) and Amnesic shellfish poisoning (ASTs) (Ben-Gigirey et al. 2021). HABs of the brevetoxin-producing dinoflagellate *Karenia brevis*, also known as “Florida red tide”, are periodically reported in the Mexican Gulf and coastal waters of Ecuador where seabird mass mortality events have been associated with PbTXs. PSTs are mostly associated with marine dinoflagellates (genera *Alexandrium*, *Gymnodinium* and *Pyrodinium*) and freshwater cyanobacteria, which form extensive blooms around the world. ASTs (domoic acid (DA) and its isomers) are a group of marine biotoxins of which DA is the main compound. ASTs are produced only by diatoms (mainly the genus *Pseudo-nitzschia*, but also some *Nitzschia* and *Amphora* species) and certain rhodophytes. DA can bioaccumulate in the tissues of marine organisms, such as shellfish, anchovies and sardines that feed on the phytoplankton able to produce this toxin (Ben-Gigirey et al. 2011). Therefore, other marine animals, seabirds, or even humans could exhibit an acute intoxication via the consumption of contaminated foods primarily causing effects on both the gastrointestinal tract and nervous system. Only circumstantial evidence for algal blooms and red tides in Australian and NZ LPs exists (Fortescue 1995). An event in Bowen Island coincided with an algal bloom in Jervis Bay NSW, reducing water visibility but no other pathology was reported in LPs (Norman 2005). In yellow eyed penguins a brevetoxin was isolated causing neurotoxic and haemolytic effects across multiple species including Brandt’s cormorants, double-crested cormorants, pelagic cormorants, frigatebirds, and pelicans (Stidworthy and Dent 2018). In New Zealand in 2010, a red algae bloom was linked to climatic fluctuations caused by El Nino and resulted in deaths in LPs along the west coast of the north island. In the Falkland Islands (Malvinas) in November 2002, an algal bloom caused paralytic shellfish poisoning and the subsequent death of a large number of seabirds (Uhart et al. 2004). Affected species include gentoo, Southern rockhopper, and Magellanic penguins as well as albatrosses, petrels, and prions Estimates suggest that 100,000–200,000 seabirds may have died during this event. Clinical signs in affected birds were sudden death, loss of equilibrium, incoordination, convulsions, paralysis, vomiting, abnormal green-brown faeces, and congestion of organs including lung (Shunway et al. 2003, Uhart et al. 2004) Histopathology of affected birds showed minor microscopic changes.

As these toxins are neurotoxins both clinical and postmortem findings reflect this action. Routes of exposure vary based on the toxin and species affected. Exposure occurs either directly through respiratory exposure or indirectly via food-web transfer. Clinical presentation can vary from individual affected animals to mass mortalities. History and environmental assessments may include associated die-offs of birds or fish, and detection of pathogenic algal blooms within the area.

There are four main groups of toxins of concern within Australia that may accumulate in shellfish tissue and cause illness in humans.
1)A range of Paralytic Shellfish Toxins such as STX, C toxins and gonyautoxins are produced by several dinoflagellate species including *Alexandrium catenella, A. minutum, A. tamarense* and *Gymnodinium catenatum*. These toxins may be fatal to human consumers of contaminated shellfish through respiratory paralysis, although this is rare and there have been no fatal cases in Australia. PSP was detected in Port Phillip Bay mussels in 1993 and 1994 at the Clifton Springs and Grassy Point harvesting areas; the most likely source was *A. tamarense* (Arnott et al. 1999).
2)Amnesic shellfish poisoning is caused by domoic acid produced by several species of diatoms belonging to the genus *Pseudo-nitzschia*, such as *P. australis* and *P. multiseries.* ASP may cause symptoms from nausea, vomiting and abdominal cramps to dizziness, hallucinations, short-term memory loss and seizures. Although most species of *Pseudo-nitzschia* are non-toxic, they are very difficult to separate definitively using only light microscopy. Hence, all *Pseudonitzschia* are initially assumed to be toxic until definitive identification is made. There are no documented cases of amnesic shellfish poisoning in Australia. Domoic acid has not been detected in Victorian mussels but has been detected in scallops from Bass Strait (Arnott et al. 1999).
3)A range of DSP toxins such as OA, DTX 1 – 3 and PTX are produced by several species of dinoflagellate including *Dinophysis acuminata, D. acuta, D. fortii* and *Prorocentrum lima*. Diarrhetic shellfish poisons may cause gastrointestinal problems including diarrhoea, vomiting and abdominal pain; recovery occurs within three days irrespective of medical treatment (Hallegraeff 1997). There have been no reported cases of diarrhetic shellfish poisoning within the areas covered by the Victorian Marine Biotoxin Management Plan.
4)Neurotoxic shellfish poisoning is caused by brevetoxins produced by some dinoflagellates, particularly *Karenia brevis.* NSP symptoms vary from headaches, diarrhoea, muscle and joint pain, and vomiting in mild cases, to paraesthesia, altered perception of hot and cold and breathing and swallowing difficulties in extreme cases. Which species produce BTX (brevetoxins) at levels sufficient to cause human intoxication is confounded somewhat by a lack of knowledge of the taxonomy of this group. The only suspected NSP incident in Australia was reported in 1994 and resulted from the consumption of wildstock mussels from the Tamboon Inlet in Gippsland, Victoria. *K. cf brevis* was identified as the organism responsible (Arnott 1999).

HAB's are a potential risk for LPs if suitable environmental conditions occur. Given such events can kill large numbers of seabirds, they will probably become a greater problem for penguins and other seabirds in the future if the frequency of harmful algal blooms increases as a result of regional warming and altered ecosystem properties (Tratham et al. 2014). Domoic acid has been associated with extensive mortalities of California sea lions, dolphins, and southern sea otters on the California coast so PI seals are also of high likelihood of exposure and clinical disease (Guiland 2000). An increased frequency, duration, and magnitude of HABs has been attributed to anthropogenic inputs of nutrients interacting with natural cycles (Landsberg et al. 2007) and, in some cases, the global transport of species into areas where the lack of competition or predation may allow for extensive growth (Hallegraeff 1995). Changes in agriculture and aquaculture practices, over-fishing, and climate change may also be important factors in the global increase in HABs (Landsberg et al. 2007).

Risk assessment	
Entry and exposure assessment	Consequence assessment
Direct ingestion appears to be the primary route of exposure in previous events as contaminated fish were confirmed in the stomachs of several birds. Shorebirds and gulls and pet dogs could also be exposed via the scavenging of fish deposited on beaches during blooms. Humans may also be affected through the	Sporadic in occurrence, low likelihood of algal bloom, requires suitable environmental conditions. However may become of higher likelihood with increased nutrient loads as a result of

consumption of affected fish and direct dermal contact leading to multiple symptoms such as rash and irritation. Under certain environmental conditions, toxins generated from HABs may become airborne, and subsequent inhalation of the generated aerosols can induce adverse health effects (Lim et al. 2023).	regional warming and altered ecosystem properties. If a HAB occurs of moderate consequences to PINP penguins, and would likely also be significant exposure and resultant disease to seabirds and seals, dogs and humans. If were to occur would likely be managed centrally as per the Victorian Marine Biotoxin Management Plan (Ecowise Environmental 2008) under the Office of the Chief Health Officer, Public Health Division, DHHS (Victoria). Noting the 2012 red tide blooms in Florida were linked with approximately 11,000 hospital admissions and 4000 emergency department visits (Lim et al. 2023) demonstrating potential for moderate associated human consequences.

Risk evaluation

Preventative measures should be employed to reduce the disease risks.

Risk management options

Diagnosis:

To implicate HABs in bird mortality, extensive environmental and animal sampling is required. Future investigations should adopt a comprehensive approach that includes analysis of environmental factors, collection of water or benthic samples for microalgal and phycotoxin testing, conducting routine screening for phycotoxins in tissues and gastrointestinal contents, and documenting clinical, gross, and histopathological abnormalities in cases of suspected phycotoxicosis (Landsberg et al. 2014).

Domoic acid can be detected in stomach content, serum, faeces, and urine. However, collection of appropriate diagnostic samples is often impeded by the rapid clearance of the toxins by urination, defecation, and vomiting. Because of this, collection of samples early in affected animals, and collection of a variety of fresh (non-fixed) post-mortem samples is critical for proper diagnosis. Highest levels of toxin have been identified in urine and faeces. Scanning electron microscopy of gastric content and faecal samples can identify the presence of *P. australis* (St Leger et al. 2004).

Brevotoxin can be found in stomach content, liver, kidney, and lung tissues of affected animals via receptor assay, ELISA, and HPLC-mass spectrometry. Additionally, immunohistochemistry utilizing anti-brevotoxin antibody performed on fixed tissues has identified positive staining in lymphocytes and macrophages in respiratory, renal, and nervous tissue.

Toxin level determinations are required for definitive diagnosis and ecological assessment of marine biotoxin impacts. Because there are multiple biotoxins implicated and potentially affecting marine mammals, sample collection for analysis of unknown toxins should include multiple fresh or frozen samples. These should include gastric, intestinal, and colonic content; urine; bile; serum; CSF; brain; lung; liver; and kidney (St Leger et al. 2004).

Knowledge gaps -Further investigations are needed to determine dose response and toxicokinetics, the impacts of multiple biotoxins, as well as chronic, low-level exposure impacts in PINP LPs and sympatric species.

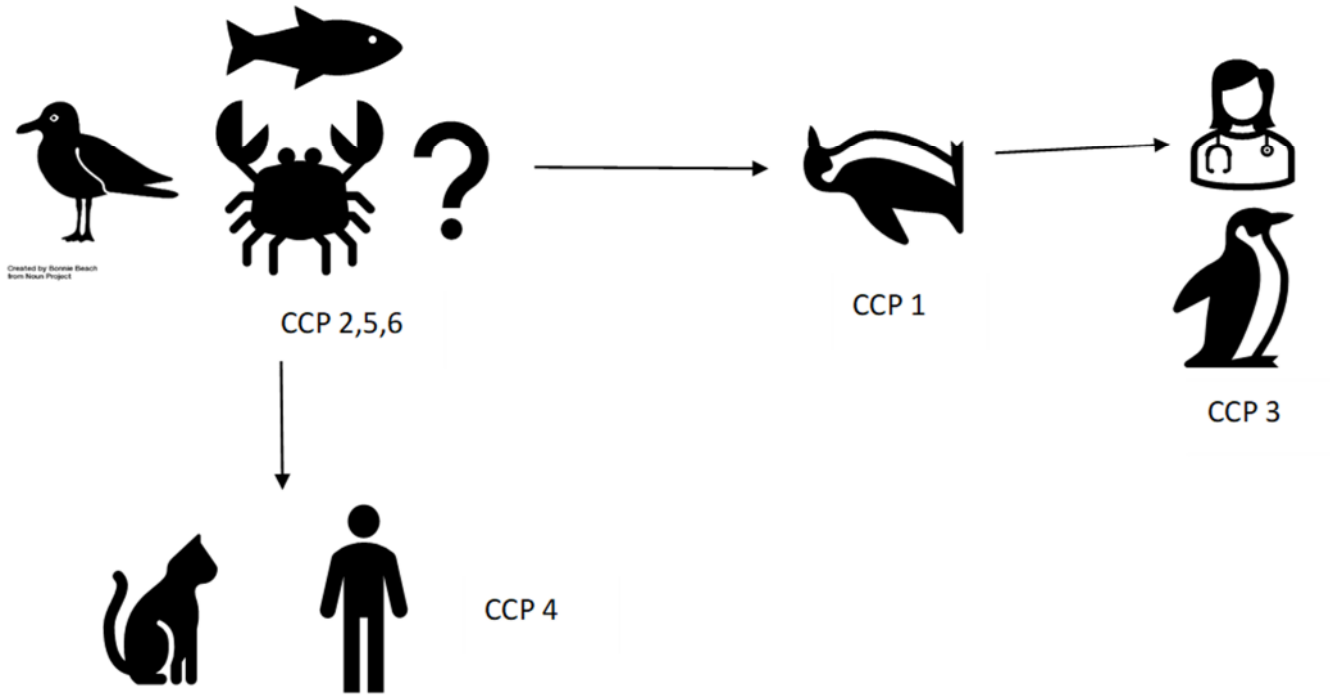


Figure 15. Critical control points for the hazard algal toxins

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Phytoplankton monitoring is used to provide early warning of the presence of phytoplankton with the potential to contaminate shellfish with marine biotoxins and is administered through the Victorian Marine Biotoxin Management Plan (2017) owing to the farming of mussels outside of the Port Phillip Bay area. Symbio Laboratories, a NATA certified laboratory in Sydney can perform the full range of biotoxin analyses required by the Food Standards Code which includes PSP, ASP, DSP and NSP. Periodic measurement of algal toxins indicated through penguin necropsy and tissue samples, and eggs.	Y
2	Environmental strategies			Remove or reduce the magnitude and potency of the HAB, to prevent bird exposures by managing the affected habitat or toxic prey, or to treat birds after they are affected. However many HABs occur offshore or in inaccessible areas so that bird carcasses are unnoticed or become too badly decomposed for accurate diagnosis. Significant events may go unnoticed or are severely underestimated (Landsberg et al. 2007)	Y

3	Treatment	Low	M	In the rehabilitation setting exposure often lethal and, or unwell birds are often not easily recovered from the wild. (Landsberg et al 2007). Birds suffering from brevetoxicosis are usually dehydrated, weak, and unable to stand. If birds are removed from the area of exposure and treated with supportive therapy, including but not limited to IV fluids, assisted feeding and nursing to hasten the elimination of the toxin then birds can recover and can be released. However in double-crested cormorants representation within five days was common Kreuder et al. (2002) suggesting that birds were either becoming re-exposed to brevetoxins or that they demonstrated signs of delayed effect from the initial exposure.	attempt
4	Prevent human and pet access too affected area			Make sure to gut fish, crayfish and crabs before cooking. Eating fish and shellfish from affected areas should be avoided. Pets should also be banned from area to minimise likelihood of exposure and ingestion. Requires appropriate signage and education of local community	Y
5	Environmental causes can be difficult to prevent but can be useful to educate public	M	H	Large numbers of dead or dying seabirds can create an awareness of offshore marine events, and provide important clues of ecosystem disturbances and hopefully action change	Y
6	Investigate algal toxins as a contributor to death in mass mortality events	M	M	Requires monetary resources and staff to facilitate. Small scale funding available through Wildlife Health Australia to investigate wildlife mortality events https://wildlifehealthaustralia.com.au/Portals/0/Incidents/NSDI_Wildlife_disease_investigation_funding.pdf	Y

4.4.13 Disease risk assessment for Persistent Organic Pollutants (POPs)

Justification of hazard	
Host range & impact: Persistent Organic Pollutants (POPs) are chemicals that are toxic, persist in the environment and animals, bioaccumulate through the food chain, and pose a risk of causing adverse effects to animal and human health and the environment even at low concentrations. Examples of POPs include DDT, polychlorinated biphenyls (PCBs) and some per- and polyfluoroalkyl substances (PFAS). Australia ratified the Stockholm Convention in 2004 which places controls on the import, manufacture, use and export of twelve chemicals listed as POPs. These include Aldrin, Chlordane, Dieldrin, Endrin, Heptachlor, Hexachlorobenzene (HCB), Mirex, Toxaphene, Polychlorinated biphenyls (PCB), DDT, Dioxins, Furans (Dept of Climate Change, Energy, Environment and Water 2024). Except for some perfluoroalkyl substances (PFAs), most of the chemicals defined as POPs under the Stockholm Convention on Persistent Organic Pollutants are bioaccumulative and lipophilic (UNEP 2009). In seabirds, high POPs concentrations in blood have been linked to oxidative stress in individuals (Costantini et al. 2017)) as well as wing feather asymmetry (Jenssen et al. 2010) and endocrine disruption. Lewis et al. (2020) reports 'On a population level, POPs contamination can cause changed reproductive behaviour and reduced offspring performance. Combined interactions with other pollutants such as mercury can affect individual survival and fecundity, and therefore population viability.' POPs have been linked to adverse effects on human health such as cancer, damage to the nervous system, reproductive disorders and disruption of the immune system (Lallas EP 2001, UNEP 2009). Due to their potential for long range movement (circulating via the atmosphere, and other pathways), significant concerns regarding persistence, bioaccumulation and toxicity have been raised. Seabirds have historically served as sentinels of marine environmental pollution, as they are long-lived, with wide-ranging foraging locations that can integrate contamination both spatially and temporally. Consequently, seabirds can show high concentrations of persistent organic pollutants (POPs) due to their position as apex predators, through biomagnification up marine food chains (Mallory and Braune 2012). Until recently there was little information in regards to baseline levels of these agents in southern hemisphere seabirds. Lewis et al. (2020) reported baseline information on POPs in migratory short-tailed shearwaters from Fisher Island, Tasmania, and LPs from Phillip Island (Millowl), Victoria. Levels of polychlorinated biphenyls (PCBs), organochlorine pesticides (OCPs) and brominated flame retardants (BFRs) were determined from blood samples, with total contamination ranging 7.6-47.7 ng/g ww for short-tailed shearwaters and 0.12-46.9 ng/g ww for LPs. In both species contamination followed the same pattern where PCBs>OCPs>BFRs. BFR levels included the presence of the novel flame retardant hexabromobenzene (HBB). In this case LPs were thought to be excellent indicators of regional pollution; given their defined foraging habits. The distribution of pollutants within the body varies with both pollutant and tissue type Penguins have a high lipid content and slow metabolism, therefore most species have a very slow process of pollutant detoxification (Jara-Carrasco et al. 2017). Despite small fractions eliminated by guano (Falkowska and Reindl, 2015), POPs can persist and accumulate in penguins, with enhanced circulation of pollutants when animals are fasting and mobilize fat reserves. Differing levels of body fat influence tissue levels of lipophilic contaminants found in Sydney and Phillip Island (Millowl) LPs (Norman 2005). Dichlorodiphenyltrichloroethylene (DDE), polychlorinated biphenyl (PCB), hexachlorobenzene (HCB) and heptachlorepoxide redistribute to liver and brain as body fat is depleted. Concentrations at PI and Sydney interestingly were higher than those reported in subantarctic and antarctic species (Lewis et al. 2020). Redistribution of organochlorines due to starvation has importantly also been noted in reports of other seabirds and Adelie penguins (Gibbs 1995).	
Risk assessment	
Entry and exposure assessment	Consequence assessment

Many pollutants are likely to be already present at variable levels. Contaminants such as heavy metals, dioxins and trace elements are known to be present in wastewater that the Western Treatment Plant processes (DAWE 2020). This combined with the work of Lewis et al (2020) in regards to entry and exposure would lead to a high likelihood of exposure for both LPs and sympatric species given they share a similar environmental niche at Phillip Island (Millowl). Migratory seabirds such as the short-tailed shearwater may have increased opportunities for exposure during migration between the north and southern hemisphere.	Many of these chemical compounds are stable in the environment and cause persistent contamination leading to biomagnification and bioaccumulation through food chains. However consequences are likely to vary depending on species affected, life stage, level of concentration and type of pollutant. Exposure likely in LPs owing to proximity to Port Phillip Bay, however overall low population consequence but may be additive over time and increasing effect with food shortages. Surrounding agricultural land is likely to result in historical and continuing runoff of agricultural chemicals as well as heavy metals and trace elements from soil works, quarries and mining. Port Phillip Bay and Corio Bay with associated port, industry and recreational uses are assumed to be contributing to pollutant risk.
Risk evaluation	
Preventative measures should be employed to reduce the disease risks.	
Risk management options	
Diagnosis: Multi-modal including environmental detection • Water samples • Soil samples • Vegetation samples • Sediment samples139 Animal detection • Blood sample • Necropsy and tissue samples • Feather samples • Egg shells Diagnostic tests • Clotting time tests • High performance liquid chromatography (HPLC) • Mass spectrometry Laboratory tests to identify pollutants are expensive and this often limits the number of tests performed. Many of the pollutants in question are present at low levels in soil, water and animal tissues, making interpretation of positive results challenging (Ramsar wetlands DRA 2020).	

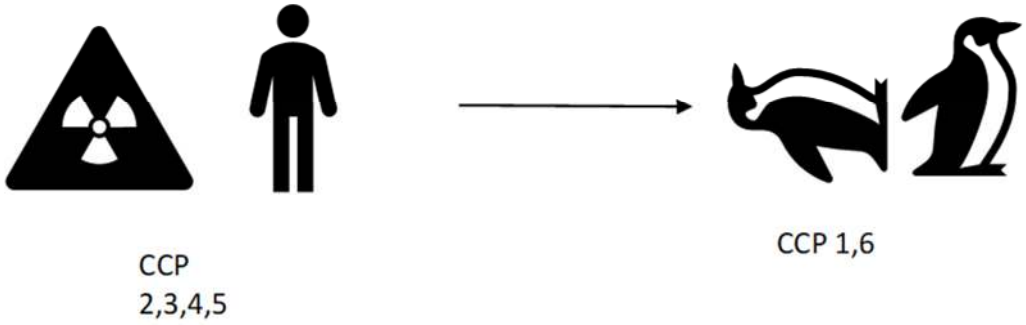


Figure 16. Critical control points for the hazard persistent organic pollutants (POP)

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive (and active) surveillance	Low	High	Periodic measurement of POPs indicated through penguin necropsy and tissue samples, feather samples and egg shells. Could also be expanded to regular surveillance of live population.	Y
2	Improve and enforce legislation on use and discharge of chemicals	High	Low/medium	Would potentially reduce presence in environment. Resources to enforce compliance and prosecution are not currently present.	T
3	Improve education on chemical disposal on a household level at Phillip Island (Millowl)	M	H	Requires behavioural change which is difficult to affect. Education programs would need to target industry and householders for best impact	Y

4	Integrated pest management to reduce reliance on higher-risk chemicals	M	M	Change in pest management has the potential to reduce chemicals in environment. Alternatives to currently used chemicals may have barriers that prevent use and may not be appropriate in all situations.	Y
5	Improve education on chemical disposal to farmers in Phillip Island (Millowl) and surrounds	M	H	Requires behaviour change, which can be difficult to affect through education alone.	Y
6	Investigate pollutants as a contributor to death in mass mortality events	M	M	Requires monetary resources and staff to facilitate	Y

4.4.14 Disease risk assessment for Per- and Polyfluoroalkyl substances (PFAS)

Justification of hazard

Per- and Polyfluoroalkyl substances (PFAS) a group of over 4000 anthropogenic chemicals have been extensively produced and used as surfactants and repellents for decades (Sun et al 2023). PFAS can be directly discharged into the environment during production and use, or indirectly generated via degradation of precursor compounds There is growing acknowledgement that PFAS are hazardous chemicals (Sun et al 2023). PFOS and PFOA were included in the Stockholm Convention on persistent organic pollutants in 2009 and 2019 (UNEP 2019). Tolerable or maximum contaminant levels have been set for PFAS including perfluorononanoic acid (PFNA) and perfluorohexane sulfonic acid (PFHxS) regionally in Australia and several U.S. states (Australian-Government 2019). The toxic and/or bioaccumulative potential of legacy PFAS such as PFOS and perfluorooctanoic acid (PFOA) has been well-established, and that of alternative PFAS are currently being discovered (Henry et al. 2018).
PFAS are of potential concern owing to their environmental persistence and ability to disperse over great distances in the marine environment. PFAS have been increasingly detected in wildlife and present unknown health risks. Most people living in Australia will have detectable levels of PFAS in their blood. Exposure to PFAS can be from a variety of sources such as stain and water protectants for carpets, fabric, furniture and apparel, paper coating (for some food packaging), metal plating, photographic materials, aviation hydraulic fluid, cosmetics and sunscreen and medical devices (Australian govt PFAS taskforce 2024).
There is a growing body of evidence describing PFAS in seabird species, however knowledge from temperate Southern Hemisphere regions is lacking other than in Tasmanian LPs where 14 PFAS in serum and scats were detected at nine sites. Perfluorooctanesulfonic acid (PFOS) and perfluorohexanesulfonic acid (PFHxS) were most commonly detected (Wells et al. 2024). Recently published PFAS concentrations in Australian fur seals and sea lions, breeding and foraging in close proximity to LPs detected concentrations comparable to northern hemisphere counterparts (Taylor et al. 2021), and concentrations detected in stranded dolphins from south-east Australia were the highest globally reported (Foord et al. 2024).

PFAS were widely detected in LPs around Tasmania. Urban intensity represented by total road length (km) within 1 km of each site, was positively associated with PFAS concentrations in soils and plasma of LPs (Wells et al. 2024). PFOS in female LPs was negatively associated with haematocrit and plasma proteins, while PFOS in male LPs was positively associated with erythrocyte nuclear abnormalities (Wells et al. 2024).
Biological consequences from PFAS exposure in seabirds include disruption of thyroid function, increased oxidative stress, effects on telomere length, reduction in body condition, lack of hatching success and weight gain (Costantini et al. 2019) and demographic responses which are typically initial symptoms in response to a stressor (Eckbo et al. 2019, Sebastiano et al. 2020).

Risk assessment

Entry and exposure assessment	Consequence assessment
LPs are exposed to PFAS predominantly through ingestion and nestlings may be affected by maternal transfer (regurgitative feeding) (Gebbink et al. 2011). It is highly likely PINP LPs have been exposed given the reported presence in LPs in Tasmania and the Port Phillip Bay and Corio Bay with associated port, industry and recreational uses are assumed to be contributing to pollutants. PFAS distribution and concentration in seabirds vary with trophic level, life history, behaviour and physiology, diet and habitat of the species (Hong et al. 2022). Sensitivity to contamination also varies depending on age, sex and growth stage (Burger and Gochfeld, 2004). Migratory species such as shearwaters that feed on zooplankton and other organisms can also transport marine-derived contaminants to remote areas including the Arctic, leading to the	LPs on Phillip Island (Millowl) likely to have been exposed given findings from Tasmanian study and levels detected in closely habiting sea lions however, further research required to understand trophic bioaccumulation pathways, and to understand sublethal consequences from exposure. Likely to become of increasing importance in the future.

contamination of ecosystem distant from pollution sources (Blais et al. 2005). There is a high likelihood of exposure to LPs and sympatric species exposed to PFAS	
Risk evaluation	
Preventative measures should be employed to reduce the disease risks.	
Risk management options	

Diagnosis:

PFAS are sequestered in eggs during their formation by the adult breeding female (Jouanneau et al. 2021), making the egg ideal for PFAS biomonitoring in seabirds, especially when blood or carcasses are not available.

Otherwise, liver, kidney, bile secretion and other tissues rich in protein are also suitable (Sun et al 2023).

Only a few studies have identified toxicity reference values (TRVs) in avian tissues. For instance, TRVs and predicted no effect concentrations (PNECs) have been established for serum (1700 and 1000 ng/mL, respectively), egg yolk (1700 and 1000 ng/mL, respectively), and liver (600 and 350 ng/g, respectively) based on acute and chronic dietary exposures of northern bobwhite quail (*Colinus virginianus*) and mallard (*Anas platyrhynchos*) (Newsted et al. 2005). In addition, a lowest-observed-adverse-effect level (LOAEL) of 100 ng/g has been approximated for eggs of white leghorn chicken (*Gallus domesticus*) based on reduced hatchability (Molina et al. 2006). See Sun et al. (2023) for a comprehensive summary of studies reporting the exposure to PFAS in seabirds worldwide. Also note that shorter-chain and branched PFAAs, as well as novel PFAS such as FTCAs, FTSs, FTOHs, ADONA, Gen-X and F–53B are much less studied and represent a significant knowledge gap.

Longitudinal studies from the PINP are ideally required to assess population significance.

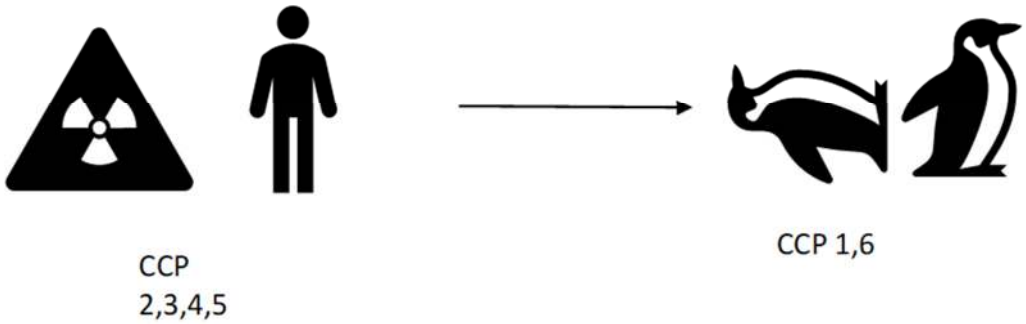


Figure 17. Critical control points for the hazard per- and polyfluoroalkyl substances (PFAS)

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive (and active) surveillance	Low	High	Periodic measurement of PFAS indicated through penguin necropsy and tissue samples, and eggs. Testing is expensive, however. Can also be expanded to regular surveillance in live populations.	Y
2	Improve and enforce legislation on use and discharge of chemicals	High	Low/medium	Would potentially reduce presence in environment. Resources to enforce compliance and prosecution are not currently present.	T
3	Improve education on chemical disposal on a household level at Phillip Island (Millowl)	M	H	Requires behavioural change which is difficult to affect. Education programs would need to target industry and householders for best impact	Y
4	Integrated pest management to reduce reliance on higher-risk chemicals	M	M	Change in pest management has the potential to reduce chemicals in environment. Alternatives to currently used chemicals may have barriers that prevent use and may not be appropriate in all situations.	Y
5	Improve education on chemical disposal to farmers in Phillip Island (Millowl) and surrounds	M	H	Requires behaviour change, which can be difficult to affect through education alone.	Y
6	Investigate pollutants as a contributor to death in mortality events	M	M	Requires monetary resources and staff to facilitate	Y

4.4.15 Disease risk assessment for environmental stressors including climate change

Justification of hazard
As stated in the Intergovernmental Panel on Climate Change's Fifth Assessment Report (IPCC 2014), greenhouse gases, such as carbon dioxide, have a warming effect on global climate. These gases absorb heat that would otherwise be lost to space and radiate it back into the atmosphere and to the Earth's surface. The IPCC concluded that it was extremely likely that more than half of the observed increase in global average surface air temperature from 1951–2010 have been caused by the anthropogenic increase in greenhouse gas emissions and other anthropogenic causes. Further increases in greenhouse gas concentrations resulting primarily from burning fossil fuel will lead to further warming, as well as other physical and chemical changes in the atmosphere, ocean and land surface (IPCC 2014). Changes in many extreme weather and climate events have been observed since about 1950. Some of these changes have been linked to human influences, including a decrease in cold temperature extremes, an increase in warm temperature extremes, an increase in extreme high sea levels and an increase in the number of heavy precipitation events in a number of regions. Climate change is unique as a driver of disease as it can affect all body systems, impair immune response and exacerbate the frequency, severity and spread of disease agents (Duncan et al. 2022). The mechanisms underpinning climate-associated illness in wildlife are multifactorial and interconnected. Climate change may compound the influence of other human-driven threatening processes such as habitat alteration, pollution, overexploitation, tourism and trade. As climate continues to change, novel conditions and disease risk factors will likely arise (WHA 2023f). Specific stressors of interest highlighted for seabird populations include sea level rise, decreased rainfall and humidity, increased fire risk, increase in extreme climate events, warmer oceans, ocean acidification, changing distributions of vectors and therefore potential for increase in vector-borne disease (eg arboviruses), increases in sea surface temperature (SST) and marine heatwaves. Rising average sea temperature and extended heat waves late summer and early autumn can create problems for LPs in catastrophic moult as they are unable to cool themselves in water and are subjected to thermal stress. Shifts in prey distribution due to climate change can also have an indirect effect on susceptibility to disease and can increase a host's exposure to new parasites (Xavier et al. 2013). Penguins appear to respond to changing environmental conditions in the short term through modifications in breeding parameters and in the long term by altering their distribution and abundance (Forcada and Trathan 2009). Changing environmental conditions place additional stressors on species and likely affect immunocompetence (Ropert-Coudert et al. 2019) and in doing so increase susceptibility to disease. Like many other species, penguin life cycles are affected by climate change directly through El Nino, increased frequency of heat waves and storms leading to egg and chick loss (Boersma and Rebstock 2014), or indirectly through increased frequency of bushfires (Chambers et al. 2010), and climate-driven changes in prey abundance and distribution (Trathan et al. 2006, Vargas et al. 2006). Climate variability and change affects seabirds, both directly (e.g. heat-related mortality) and indirectly (e.g. through the impact of climate on food webs) (Ainley et al. 2010). A report on climate change modelled on data from LPs at Phillip Island (Millowl) (Dann and Chambers 2009) predicted little direct impact of decreased rainfall and humidity. However, fire risk may increase leading to adult mortality and habitat loss and the fire season may be extended. Extreme climate events may also slightly reduce adult and chick survival. Warmer oceans were likely reported to improve recruitment into the breeding population (longer breeding season and greater availability and quality of feed predicted) but the effects on adult survival were not clear. Based on a projected tripling of days with temperatures >35°C by 2070 (the mid-range estimate for coastal regions of Western Port), heat stress in the LP is likely to increase. At temperatures >27°C, the daily energy budgets of penguins is expected to increase in conjunction with increasing temperatures, as the penguins expend energy to maintain core temperatures (Baudinette et al. 1986). Overall, many aspects of LP biology are likely to be affected by climatic change but no net negative effect on population size was projected. Climate change may also alter ecosystem properties for example air temperatures, precipitation, humidity, winds and rainfall, allowing disease-carrying vectors to establish where historically the climate was unsuitable (Tratham et al. 2014).

At Penguin Island in WA **i**ncreasing ocean temperature and strong winds have been linked to negative effects on adult foraging and chick survival (Cullen et al. 2009, Cannell et al. 2012, IUCN 2020). Under La Niña conditions, there is typically higher rainfall and more storm activity, while the opposite is true for El Niño conditions (Holbrook et al. 2009). The variability in rainfall across years will influence habitat quality for LPs. Higher rainfall and storm activity may increase erosion of pathways to nest sites, while less rainfall may reduce native vegetation cover ideal for nesting (Sutton 2022). Marine heatwaves are projected to become more frequent, intense, and persistent in Australia with climate change predictions (Commonwealth of Australia 2020). Following the marine heatwave of 2011 around Penguin Island WA, the spawning and migration patterns of many fish species were impacted for subsequent years (Caputi et al. 2014). Changes in the diet of LPs from Penguin Island were also noted. For the colony on Penguin Island, elevated SST prior to a breeding season has been linked to reduced courtship, delayed egg laying, and a higher occurrence of egg abandonment (Cannell et al. 2012). Following the marine heatwave in 2011, where elevated temperatures persisted above average for several years, overall chick production was low (Cannell 2017, 2018).

Exposure to these climatic changes will undoubtedly lead to changes in the marine and terrestrial environment of LPs and the likelihood of increased extreme weather events is also likely to be significant for the LP population and other sympatric species on Phillip Island (Millowl) and globally. Australia's 2019-20 "Black Summer" bushfires are an excellent example of a climate change induced event impacting wildlife health. Preceding severe drought, record temperatures and high winds, led to wildfires causing significant destruction to wildlife and the environment. The reported figures of 18.6 million hectares burnt and almost three billion animals killed or displaced are thought to significantly underestimate the impact of these wildlfires (van Eeden et al. 2020).

Specific identified stressors for LPs at Phillip Island (Millowl):

Sea level rise

Dann and Chambers (2013) systematic review on climate predictions for LPs at Phillip Island (Millowl)**,** included a small loss of penguin breeding habitat due to sea level rise. The sea level has risen at a rate of 1.4 mm/year across Australia's coastlines between 1966-2009 (CSIRO and Bureau of Meteorology 2022). This is of concern as rising sea levels can threaten LP burrowing and nesting sites through inundation or erosion of pathways to nests, however, the relative impact to the population was thought to be low.
Indirect threats such as habitat loss can impact the distribution and abundance of penguins.

Ocean acidification and contamination

LPs spend 80% of their lives at sea. The marine ecosystem is dynamic and multiple threats to habitat exist. Land-based activities modify the marine environment through runoff of sediments, nutrients, toxins, and pollutants, and even alter the flow of currents and tides (Tratham et al. 2014). Contamination of water sources by toxins and infectious disease agents can be exacerbated by increased precipitation and temperature. High levels of rainfall can introduce terrestrial pathogens to aquatic species via runoff, as well as contributing to the growth, survival and toxicity of aquatic microorganisms, significantly impacting food webs (WHA 2023f).
Dann and Chambers (2013) noted that ocean acidification has the potential to be a highly significant negative influence on food availability with climate change predictions, but present assessments are speculative. Greenhouse gas emissions are causing the oceans to become acidic, which threatens the entire ocean food web from plankton to penguins. The oceans absorb a large portion of carbon dioxide produced by our industrial society. As ocean waters have absorbed this excess carbon dioxide, the acidity of the ocean has increased by 30% and carbonate ions have become less available. Carbonate ions are used by calcifying creatures such as plankton, corals, and clams to build their shells. As ocean acidification reduces carbonate ion availability, these creatures will become increasingly unable to build new shells and existing shells will begin

to dissolve, which affects survivability and therefore, penguins' food supply. Indirect threats such as predicted ocean acidification may impact the distribution and abundance of penguins through altering available food sources.

Reduction in food supplies / starvation

Reduction in food supply through declining water quality or other factors to reduce fish stocks. Starvation has been identified as a major factor determining the survival of young and adult penguins (Norman et al. 1992). When prey abundance is low in foraging grounds LPs need to travel greater distances and expend more energy to find prey, which can be detrimental for raising chicks. Little penguins can also experience delayed breeding cycles, delayed growth, and delayed breeding success when food resources are low (Cannell et al. 2016). Egg desertion on Penguin Island in WA was associated with hunger as birds were thin (Wienecke, Wooler and Klomp 1995). Starvation was found to be the second highest cause of mortality in 163 deceased LPs collected in the Perth metropolitan coastal areas between 2003-2012 (Cannell et al. 2016). Most starved individuals were found in spring and summer. The increased rate of deaths of LPs from starvation following the marine heatwave in 2011 and subsequent La Niña years was attributed to higher sea surface temperatures reducing the abundance of coastal baitfish species in the Perth metropolitan region (Cannell et al. 2019).

Reduced food supplies may become more likely with predicted alterations in climate and overfishing, and this is significant as it may make the population more susceptible to other threats. Most affected birds are emaciated juveniles with reduced bodyweight compared to birds surviving migration, pectoral muscle atrophy, an empty stomach, complete fat exhaustion with serous atrophy, and multi-organ atrophy. In sea birds food shortages and extreme weather events often precede large-scale die-offs (Dann et al. 2000; Frederiksen et al. 2008, Rensburg 2010). Individuals may sustain low level injury or immediate death; may survive but later die from complications or systemic infection. May present as 'wrecks' washed up on shoreline.

Storm damage and extreme weather events

Storms and waves can cause erosion of sand banks and dunes and impact the ability of LPs to reach their nests (Cannell 2001). Storms have been suggested as a cause of death for LPs and eggs may be lost due to heavy rain (Reilly and Cullen 1981). More extreme climate events are predicted with climate change in certain regions (Dann and Chambers 2013) and this may impact chick growth rate. Wienecke et al. (1995) reported a decline in growth rate after parents were prevented from accessing nests to feed chicks following a significant storm event at Penguin Island.
Severe storms are likely to only be sporadic however may increase in frequency with climate change predictions. Likely to be of moderate consequence to the population if storms are severe however overall low population consequence.

Thermal stress -terrestrial

LPs can experience thermal stress in temperatures ≥ 30°C and hyperthermia at ≥ 35°C, which has caused mortalities of LPs from the Shoalwater islands WA particularly during the moult stage (Cannell et al. 2016). During daylight hours, nest temperatures exceed ambient temperatures, causing thermal stress in LPs on warmer days. Artificial nest boxes have higher maximum temperatures during the day, exceed upper thermoneutral limits more often & have prolonged extreme temperatures more so than natural nests (Sutton 2022). Ambient temperatures can create potentially lethal temperatures inside artificial nest boxes, however manipulation of artificial nest boxes on Penguin Island found improvements could be made to boxes to reduce this threat to LPs (Clitheroe 2021). Elevated temperatures during the day can also delay the onset of breeding for some LPs on Penguin Island WA including, pre-laying nest attendance and courtship (Wienecke 1993).
Thermal stress is likely to only be sporadic however, may increase in frequency with climate change predictions.

Thermal stress -marine

Ocean warming in south-eastern Australia is causing pole-ward shifts in species ranges, including penguins and their prey. The fish penguins feed on stay in cooler, deeper waters as surface temperatures rise. Penguins and petrels respond to these changes by foraging further away and diving deeper to find food. Starvation is a risk for themselves and their chicks if food is in short supply (Boresma and Rebstock 2014). Warming ocean waters can also change the distribution of water currents and lead to changes in prey recruitment and growth, making finding food more difficult for many of the world's penguin species (Ropert-Coudert et al. 2019). For example, the Galápagos penguin has proven to be extremely vulnerable to starvation during El Niño events. Adults are forced to abandon their eggs and chicks to search for food, leaving their chicks to starve. Regional instances of episodic warming of the marine environment have also been shown to cause mass starvation of penguins in Australia (Cannell et al. 2012) These changes can also cause a shift in abundance towards species tolerant of warmer waters (Last et al. 2011).

Additional effects of rising ocean water temperatures include the increased occurrence of harmful algal blooms. Algae grow rapidly and accumulate into dense patches near the ocean surface, promoting the absorption of solar radiation, exacerbating warming. At Penguin Island in WA increasing ocean temperature and strong winds have been linked to negative effects on adult foraging and chick survival (Cullen et al. 2009, Cannell et al. 2012, IUCN 2020). In 2011, temperature anomalies of 2-4°C above average (marine heatwaves) persisted for months along the WA coastline. Following the marine heatwave of 2011, the spawning and migration patterns of many fish species were impacted for subsequent years (Caputi et al. 2014). Changes in the diet of LPs from Penguin Island were noted following the marine heatwave. For the colony on Penguin Island, elevated SST prior to a breeding season has been linked to reduced courtship, delayed egg laying, and a higher occurrence of egg abandonment (Cannell et al. 2012). Increased rainfall and water temperature also favours the growth of many waterborne disease agents such as bacteria and fungi, as well as potential arthropod vectors as described below (WHA 2023f).

Vector-born disease

Climate change can affect disease occurrence directly, as rising temperatures may increase the geographic range and abundance of ectoparasites at lower altitude. For example ticks feeding on Adélie penguins during high temperature events in Antarctica (Benoit et al. 2009). Climate change influences the El Niño cycle that is known to be associated with increased risks of some mosquito borne diseases such as avian malaria, and as well as flaviviruses such as RRV, MVEV and Kunjin. In dry climates, heavy rainfall can provide good breeding conditions for the mosquitoes. Droughts may turn rivers into strings of pools, the preferred breeding sites of mosquitos. Ticks the presumed vectors of Babesia, and Saumarez reef virus, can also change their distribution based on changing climatic conditions.

Wildfire

Increasing temperatures (leading to drier summers) and likelihood of extreme weather events (for example lightning storms) may increase likelihood of wildfires. Wildfires can potentially decimate breeding habitat of LP populations. Severe drought periods may also increase the frequency of bushfires that destroy nesting habitat (Chambers et al. 2010).

Individuals may sustain low level burns or sudden death; or alternatively may survive the initial episode, but later die from complications. Likely will have increased exposure to predation following fire, and reduction in the habitat available for nesting. Overall potential for high consequences to LP population. PINP actively managing wildlife risk through strategic prescribed burns and other pre-suppression work to provide protection and reduce the risk of bushfire impact.

The impact of climate change is expected to intensify, potentially making populations less resilient to non-climate related impacts (Crawford et al. 2017). Therefore the ability of penguin populations to cope and respond to further climate change will likely depend as to how other current terrestrial and marine threats are addressed (Ropert-Coudert et al. 2019). Climate change does not inherently promote the proliferation of all disease, rather it disrupts the extremely complex balance between environment, host, agent and vector, thereby creating conditions that can alter overall disease dynamics (Duncan et al. 2022).

Risk assessment	
Entry and exposure assessment	Consequence assessment
Already evidence of potential indirect effects of climate change on the PINP LP population which likely impacts on con-specifics and all wildlife in the region and also globally as reported by Tratham et al. (2014). Wild animals' dependence on their natural habitat means that they are extremely vulnerable to environmental changes, more so than humans and domesticated animals which benefit from having a buffer between themselves and the natural world (Duncan et al. 2022). We also have a greater ability to actively manage individuals rather than wild populations.	Overall medium likelihood of occurrence with current climatic predictions and of moderate consequence to the population. Climate change is associated with an increased incidence and severity of extreme events or "natural disasters" such as bushfires, floods, storms, heat waves, cold waves and droughts (WHA 2023f). While direct injury may result from an extreme event, the long-term impacts can be equally concerning and may include loss of refuge areas, displacement, habitat fragmentation, food and water loss and contamination, increased predation risk and increased susceptibility to an infectious disease outbreak. In large scale environmental disasters, these impacts may result in population declines, local extinctions or even ecosystem collapse. (Duncan et al. 2022). While these events are currently sporadic they may increase in frequency with climate change predictions. Indirect threats such as habitat loss through sea level rise can impact the distribution and abundance of penguins. Ongoing threat with medium consequences to the population although is being actively managed by PINP. Indirect threats such as predicted ocean acidification may impact the distribution and abundance of penguins through altering available food sources. Multiple threats which LPs are currently exposed to and which are likely to escalate owing to human population growth. Ongoing threat with medium consequences to the population Alteration in prey availability may become more likely with predicted alterations in climate and overfishing, of medium consequence to population as may make population more susceptible to other threats if food supply is reduced. Algal blooms can potentially kill large numbers of seabirds, and will likely become a greater problem for penguins and other seabirds in the future if the frequency of harmful algal blooms increases as a result of regional warming and altered ecosystem properties (Shumway et al. 2003). Human physical and mental health may also be affected by climate change and this has the potential to impact wildlife welfare where it affects our capacity to provide animals with care and protection (WHA 2023f). The growing threats are likely to lead to significant

	mental health challenges in the fields of veterinary medicine, ecology, biology , conservation and wildlife rescue and rehabilitation. Climate change modelling should be updated as new information becomes available to assist with management decision making. However, we should also note that the predictive power of relationships built on past observations (when not only the average climatic conditions are changing but also the frequency of extreme climatic anomalies) may not be a good predictor of a species' future response to climate change (Tratham et al. 2014).
Risk evaluation	
Preventative measures should be employed to reduce the disease risks.	
Risk management options	
Diagnosis: An iterative process, ongoing collection of climate data, review and feeding information back into the modelling datasets to allow better informed ongoing predictive modelling to enable more informed management.	

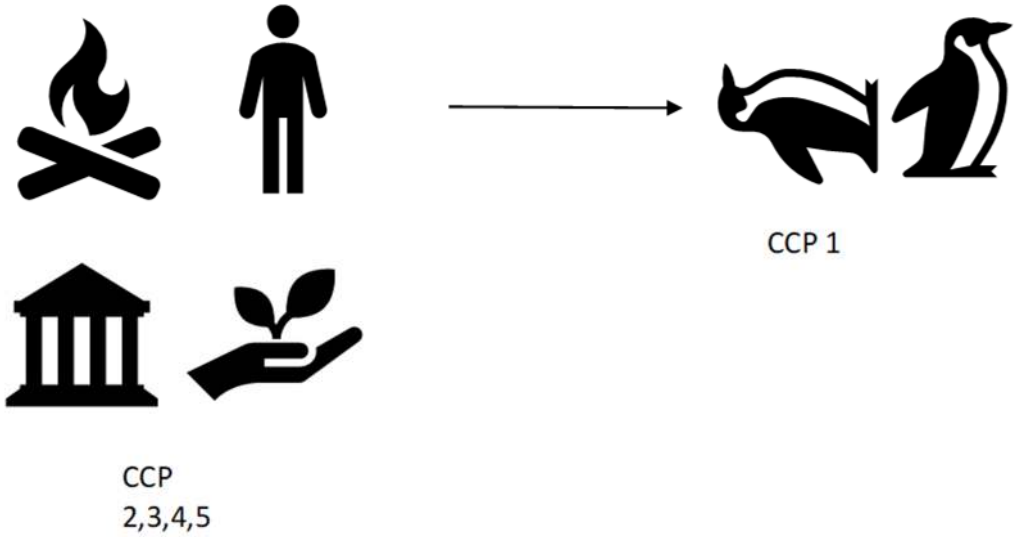


Figure 18. Critical control points for the hazard environmental stressors including climate change

CCP#	Mitigation Options	Effectiveness	Feasibility	Explanation (include any relevant sources of information)	Recommendation (Y/N)
1	Passive surveillance	Low	High	Ongoing climate modelling based on longitudinal data analysis to best predict likelihood of future threats	Y
2	Governmental pressure to limit coastal development and regulate marine fishing	High	Medium	Climate change is a global issue, not influenced by political boundaries, that requires intergovernmental effort at a global scale. However state and coastal lobbying to limit coastal development and regulate marine-capture fisheries is	Y

				justified when examining the potential for reduced prey availability secondary to climate change	
3	Establishment of further marine protection areas (MPSs) in LP range to include core foraging areas and traveling routes to and from foraging grounds identified through GPS tracking.	High	Medium	MPAs are an important management tool for conserving marine biodiversity because they allow for the sustainable and rational use of marine resources and potentially enhance fisheries management (Gell and Roberts 2003).	Y
4	Wildfire risk	High	High	PINP actively manages wildlife risk through strategic prescribed burns and other pre-suppression work to provide protection and reduce the risk of bushfire impact.	Y
5	Education and research	High	Low	State and local education to reduce emissions for a sustainable long-term future. Local action to research strategies to limit projected impacts of climate change see Penguin Foundation https://penguinfoundation.org.au/whats-new/	Y

5 General Recommendations for Disease Risk Management

Based on the Disease Risk Assessment, the following Risk Management processes will be required to mitigate residual risk.

- Increased **active and passive disease surveillance of population** is needed.
- Excellent **biosecurity and risk management practices** are required.
- Any suspicious clinical signs or lesions should lead to **specific sampling and intervention.**
- Strict **protocols during field work** should be observed to maintain biosecurity and maximise animal health and welfare.
- **Ongoing management** and surveillance of little penguins and in-contact species should be supported.
- Identified knowledge gaps should be addressed through **implementation of a research plan.**
- **Capacity and training of staff** in wildlife health management should be increased, and collaborations with wildlife health

5.1 Increased disease surveillance

Surveillance incorporates systematic collection and analysis of health information, followed by dissemination and effective communication leading to appropriate action; it provides a widely accepted approach for the identification of disease in wildlife populations, allowing wildlife managers to minimise the negative impacts of disease (Salman 2003). Disease surveillance can be divided into passive disease surveillance (focusing on detecting disease and/or cause of death) in sick and dead animals) and active disease surveillance (screening of populations for specific pathogens). Specific pathogen surveillance is helpful for the detection of changing levels and distribution of infection (e.g. through molecular testing) or exposure (e.g. through seroprevalence) in a population (for example, changes in seroprevalence) (Wildlife Health Australia, 2018). We propose both active and passive disease surveillance programs for the little penguins at Phillip Island (Millowl) to provide required baseline data and allow early diagnosis and detection of disease, as well as rapid responses to identified hazards. In the long-term, the programs will also contribute to closing the identified knowledge gaps and support a shift to preventative, or perhaps even pro-active, health management of the population.

The penguin population size and breeding success is currently closely monitored, using a combination of several rigorous survey techniques, including nightly counts of individuals crossing the beach to nest, colony-wide surveys and demographic models (Sutherland and Dann, 2014). The average number of chicks fledged per breeding pair is estimated each year. Multiple studies over the years have explored the LPs movements at sea, their foraging strategies and how they differ from nearby colonies (e.g. Venegas-Li et al. 2023; Chiaradia et al. 2012), using a range of technologies such as GPS trackers, PIT tags and automated weighbridges (Joly et al. 2022). The National Wildlife Biosecurity Guidelines (2018) further suggest the following demographic indicators of disease: increased morbidity or mortality; reduced fertility or recruitment; altered population structure (age or sex); reduced capture rate; changes (usually a reduction) in average body condition (assessed as weight or a specific condition score. Recent research in Tasmania reported moulting birds and breeding females to be particularly vulnerable to negative health impacts (Wells, M. pers. comm.).

It is recommended that further disease monitoring is undertaken, based on power analysis for the calculation of an adequate sample size to estimate the population prevalence of a particular pathogen or disease with good precision). Minimum sample sizes for pathogens can only be calculated when we have an estimation of the population size using statistical power analysis. The National Statistical Service sample size calculator, or the Epitools website, could be useful tools to facilitate this estimate:

https://www.abs.gov.au/websitedbs/d3310114.nsf/home/sample+size+calculator
https://epitools.ausvet.com.au/

From transect surveys of active nests performed across the entire population, the estimated number of breeding penguins on Summerland Peninsula in the 2022-2023 breeding season was 37,000 breeding birds (95% confidence interval of 30,000 to 45,000). In 2019-2020 the estimate was 40,000 breeding birds (32,000-49,000). The interpretation is that the breeding population remains strong. It does seem reasonable that fewer birds may have attempted breeding in 2022-2023 than in 2019-2020 which was a more successful breeding season (M. Schreider pers comm 7 Feb 2024). Given we do not have recent population estimates of disease prevalence, or, in fact, in some cases any prevalence data at all, it may be conservative to sample 381 individuals (based on a population size of 37 000) to enable a 95% level of certainty for the true population value of the hazard and/or disease.

This logistical effort and cost to sample 381 penguins would be substantial, therefore, ongoing budgetary allowances should be sought to facilitate the recommended disease surveillance programme in regards to costs for staffing, sample collection, processing and storage, sample testing and review of results which should then be integrated into an iterative annual health management plan. Even if this is not immediately possible, collection of samples and clinical examinations should be opportunistically undertaken when penguins are in hand to enable the collection of data and samples which would be highly useful for retrospective analysis should a disease event occur.

In addition, a passive disease surveillance system should be instigated, targeting little penguins that have been found recently dead due to unknown causes, or those dying in care. There is no minimum or maximum number for this type of surveillance, but the more cases that can be captured, the higher the likelihood of early detection of emerging disease.

While awaiting the surveillance results, the precautionary principle should be applied. In the context of wildlife disease risk management, the National Wildlife Biosecurity Guidelines state that “if there is a suspicion that an interaction or situation with wildlife may pose a disease risk (to other wildlife, domestic animals or humans), a risk is assumed (and managed appropriately) until proven otherwise” (Wildlife Health Australia, 2018).

5.2 General risk management and biosecurity practices

Strategies to minimise and monitor biosecurity and related risks as discussed in the National Wildlife Biosecurity Guidelines (2018) are recommended, including (citing and adapting the Guidelines):

- Standard precautions or basic biosecurity practices should be adopted, regardless of the perceived risk.
    - Basic biosecurity practices include:
      Hand hygiene, keeping the work environment clean and tidy, cleaning spills of blood and body substances, cleaning and disinfecting (or safely disposing of) equipment after use, appropriate management and disposal of waste material (including animal, food, water and clinical waste), appropriate management of laundry (bedding, towels and worker clothing), safe use and disposal of sharps such as needles and scalpel blades, as well as knives for

captive animal food preparation, managing accidental exposures to blood and body substances, as well as animal bites, scratches and sharps injuries, care with the movement of animals and items from one location to another, appropriate use of personal protective equipment (PPE), protection against biting insects and ticks that may transmit disease.

- Guided decision-making, through a risk assessment process, for health testing, health management, monitoring and surveillance.
- Maintaining basic hygiene and biosecurity protocols during capture, examination, sampling, movement and release of LPs undergoing surveillance and health screening
- Hygiene practices to reduce the risk of zoonotic disease, including hand washing with an appropriate disinfectant and provisions for clean dedicated clothing for field workers, gloves, respiratory (N95 masks) and ocular protection may also be indicated for example in cases of confirmed or suspicious HPAI
- Full and appropriate investigation and treatment of any sick or dead individuals, including full post-mortem examination and investigation to a reach a diagnosis (rather than just to rule out a suspected cause of death or disease of concern)
- Permanent identification of each LP that was sampled for disease surveillance and/or treated with this information recorded as per PINP (2020) "once a chick is classed as P1 - P4 and weighs over 700g, it is eligible to be microchipped by a qualified Nature Parks staff member or trained Higher Degree Research student. If a chick is at a P stage but weighs less than 700g then it is too small and must not be microchipped."
- Collection and recording of individual health and morphometric data associated with a handling event
- Maintenance of detailed individual animal records for an extended period, and collation of these into an accessible database
- Methods to track and monitor animals that have previously received care/treatment remotely, which may provide information on movement, interference, morbidity and mortality, e.g. GPS monitoring.

Biosecurity measures should be implemented **routinely** as standard practice whether or not an outbreak has been detected. However, the stringency of biosecurity measures may be altered in response to changes in the perceived level of risk.

In the **event of any unusual signs of disease or deaths** in wildlife (for example five cases of sudden death) contact the following organisations to assist in ongoing surveillance:

- your local State/Territory Wildlife Health Australia Coordinator (see www.wildlifehealthaustralia.com.au)
- In Victoria, Mark Hawes is the primary contact - **mark.hawes@agriculture.vic.gov.au** W: 03 9032 7275
- the 24-hour National Emergency Animal Disease Watch Hotline on 1800 675 888.
- your local veterinarian
- the Department of Primary Industry or Agriculture in the state or territory in which the event occurred

- Enhanced levels of biosecurity practices may be required depending on the situation and include: Additional levels of hygiene e.g. hand hygiene prior to commencement of the work, as well as between handling each animal (rather than between cohorts), use of additional methods of cleaning and/or disinfection e.g. use of heat, pressure, radiation or chemical disinfection or sterilisation for both equipment and personnel, use of disposable gloves (in addition to hand hygiene), use of appropriate personal protective equipment, use of isolation areas, use of dedicated equipment, temporary bagging, followed by cleaning and disinfection, of equipment and clothing after use, movement restrictions for animal and people, use of ventilation controls, respiratory protective equipment, entry/exit processes, chemical foot baths

Specific recommendations for PINP LP management:

- Exposure to pathogens can increase when species come into contact that would not normally do so. Wildlife rehabilitation and wildlife management programs are situations where abnormal species contact is highly likely to occur as a result of human intervention. These processes can result in abnormal mixing of species, and / or elevated population (stocking) density increasing the environmental load of pathogens and potential for disease transmission. LPs may have increased risk of exposure to pathogens when undergoing routine monitoring, health surveillance and if deemed abnormal and requiring further intervention when they are transferred to a wildlife hospital setting.

- Direct contact and indirect contact (e.g. through contaminated clothing, equipment, facilities, food, water or waste) between wildlife in care and free-ranging wildlife, pets, therapy animals, and feral or domestic animals can pose biosecurity risks to all the animals involved and should be minimised.

- Physical separation (barrier keeping and nursing) is required as well as dedicated equipment, tools, clean clothing and footwear and appropriate PPE e.g. gloves, facial protection in the hospital which is distinct from rehabilitation facilities and those used in the field. This is an important tool supporting the management of biosecurity and defines the use of stringent control measures designed to minimise the spread of infectious disease from one animal, group or environment to another. If used carefully, barrier keeping and nursing practices can minimise the risks associated with working with multiple different individuals or groups of animals on the same day, or within the same treatment area or facility.

- Isolation (typically in the hospital setting) involves the following: separation of newly arrived cases to allow for examination, treatment, and monitoring; clinical and laboratory diagnostic tests as required, and veterinary treatment for disease or injury. Isolation is typically undertaken for a defined, appropriate period based on the incubation period of pathogens of concern to ensure animals are free from infectious (including zoonotic) disease. It also enables appropriate veterinary care and treatment to be undertaken while protecting other populations from infectious disease transmission.

- PPE the choice of PPE should be based on the method of transmission of the infectious agent. PPE must be a suitable size and fit for the individual (fit testing may be required in certain high risk scenario's e.g. for those working in the hospital) worn correctly; fit for purpose in the conditions in which it will be worn (e.g. does not cause heat stress or fogging and allows for dexterity); and if disposable, it should be only used once, or alternatively appropriately cleaned and stored.

- Staff in contact with LPs should have the ability to disinfect footwear and equipment before entering the specific site they are stationed to. Facilities for disinfection and stocking of appropriate PPE for the designated area should be available on entry to and exit from the area.

- Please note specific biosecurity management of the wildlife hospital and rehabilitation facility is undertaken by Zoos Victoria.

- In outbreak scenarios, managers should consider dedicated staff be posted in each facility (e.g. field, hospital and rehabilitation) to minimise potential for disease transfer via fomites.

- If dedicated staff are not possible, workflow should be organised with the aim of working from lower biosecurity risk to high biosecurity risk areas. For example, those working in the field (lower risk) could later work in the wildlife hospital (higher risk) but ideally not vice versa. Noting that risk levels of these facilities are iterative based on pathogens isolated.

- In outbreak scenarios, the movement and/or introduction of livestock, people, vehicles or equipment into the Summerland Peninsula may need to be minimised or at least controlled, particularly so during periods of increased risk, or if significant morbidity or multiple mortalities have occurred and staff are awaiting diagnostics to enable diagnosis of disease.

- In outbreak scenarios, it would be beneficial to minimise the number of people participating in procedures to those workers with appropriate training and protection and only those required for the procedure.

- In outbreak scenarios, it would also be beneficial to reduce access of people with increased infection risk (e.g. those not immune to a relevant vaccine-preventable disease, pregnant or with a medical condition that increases infection risk for example individuals on immunosuppressive medication).

Disinfection

- While broad spectrum disinfectants such as bleach are reasonable for general disinfection, certain pathogens will require a higher concentrations of bleach, or alternative disinfectants based on the pathogen of concern (Figure 19 and 20).

- For example, disinfectants active against HPAI include: 70% ethanol, Virkon S, F10, soap + 10% bleach solution, or 0.1% iodine solution.

- Prepare a 1: 10 (10%) Household Bleach Solution as follows:

    - 62 ml (1/4 cup) household bleach + 562 ml (2 1/4 cups) water
    - 250 ml (1 cup) household bleach + 2250 ml (9 cups) water

- Current PINP protocols advocate the use of 1% (as compared to 10%) bleach; for ease, this should be continued when disease risk is low. However, the concentration needs to be increased from 1% to 10% as directed above, for widespread pathogen disinfection, for example in a viral outbreak scenario (such as HPAI). Please note to be effective, ALL organic matter must first be removed otherwise the virus will potentially not be killed.

- The bleach and water solution should also be mixed daily to preserve its strength.

- The advantages and disadvantages of a product such as bleach, when compared to Virkon S, are highlighted below.
  Household bleach (5% sodium hypochlorite)

  Advantages

    - Inexpensive and readily available
    - Effective in hard water

- Can be used in a wide range of dilutions between 1:10 and 1:100

Disadvantages

- May bleach clothing in higher concentration range
- Corrosive; long term use may degrade rubber parts
- Rapidly deactivated in organic matter

Virkon S

is a peroxygen compound chemical disinfectant (active ingredient potassium peroxomonosulfate) and is highly effective in the inactivation of most non-enveloped viruses. Non-enveloped viruses are more resistant to extreme pH, heat, dryness, and simple disinfectants. Some examples of non-enveloped viruses include norovirus, enterovirus, adenovirus, and rhinovirus. To be effective against these viruses, Virkon needs to be used at the dilution rate of **2% with a contact time of at least 10 minutes**. It is stable for seven days following dilution. Metal equipment and handling tools should be rinsed following disinfection with Virkon to prevent corrosion.

Advantages

- Wide spectrum of activity including non-enveloped viruses
- More effective than chlorine bleaches in the presence of organic matter
- Less corrosive than chlorine bleaches
- Made up dilutions have very low toxicity and no irritancy (powders are irritants)
- Good detergent properties combine cleaning with disinfection
- Suitable for use in many applications

Disadvantages

- Although considered relatively safe, prolonged exposure may cause irritation to skin (consider gloves if available)
- The powder dust is a respiratory irritant: Virkon S concentrate should be mixed in well ventilated areas
- Can cause eye damage. Avoid contact with the eyes.
- Flammable
- Corrosive (but less so than bleach)

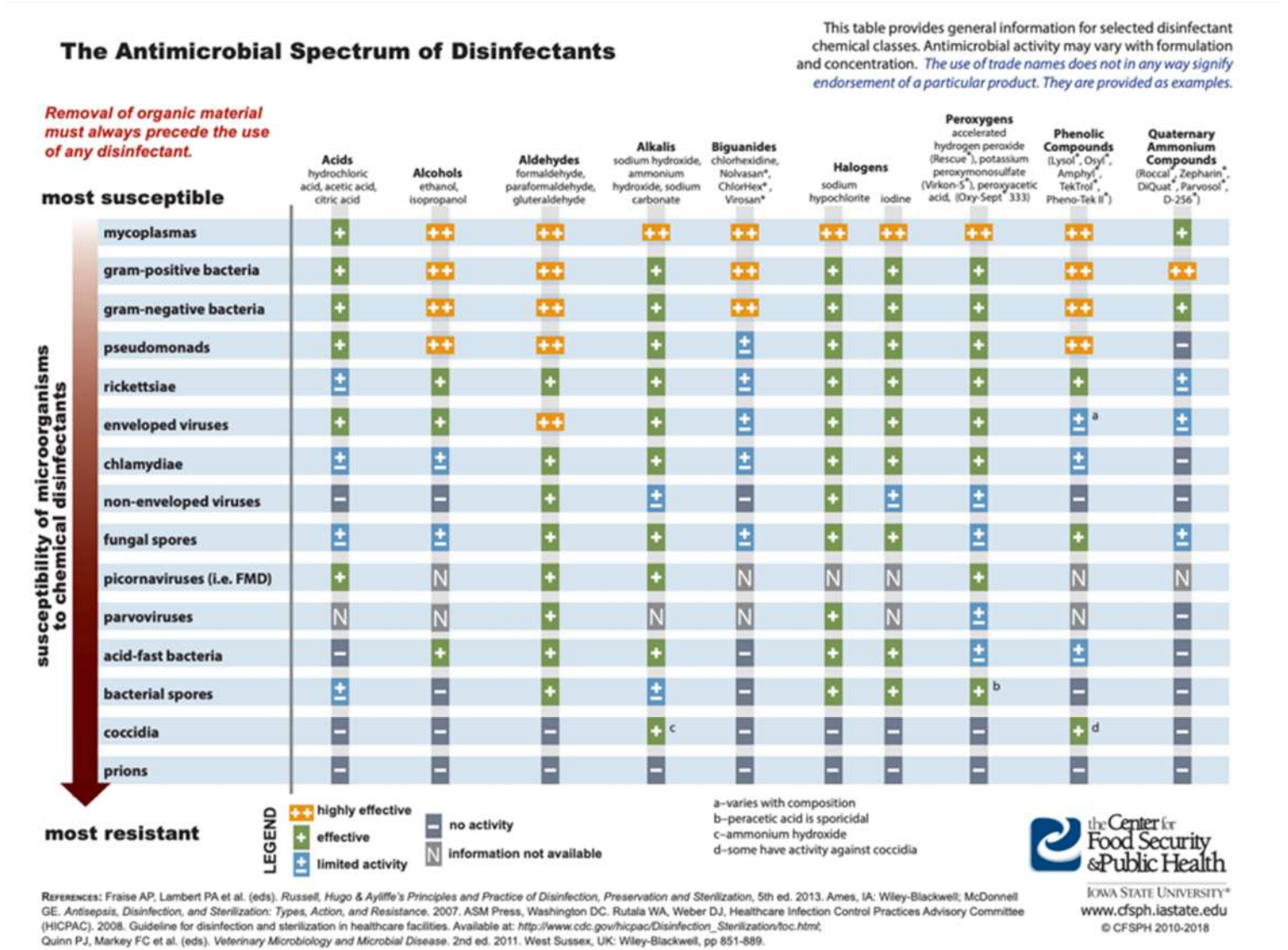
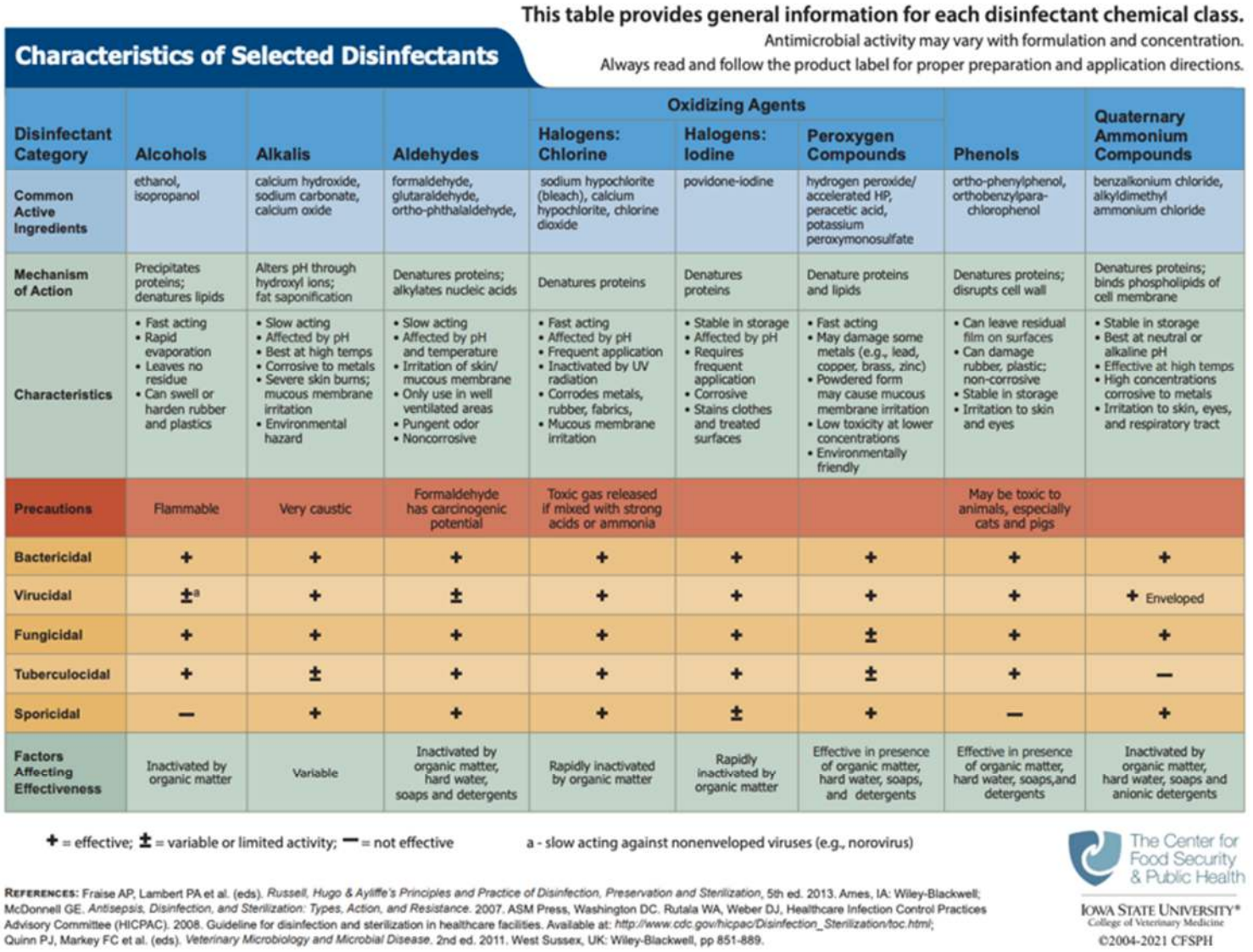


Figure 19. The antimicrobial susceptibility of disinfection (CFSPH 2023)



This table provides general information for each disinfectant chemical class.

Antimicrobial activity may vary with formulation and concentration.
Always read and follow the product label for proper preparation and application directions.

Characteristics of Selected Disinfectants

Disinfectant Category	Alcohols	Alkalis	Aldehydes	Oxidizing Agents			Phenols	Quaternary Ammonium Compounds
				Halogens: Chlorine	Halogens: Iodine	Peroxygen Compounds		
Common Active Ingredients	ethanol, isopropanol	calcium hydroxide, sodium carbonate, calcium oxide	formaldehyde, glutaraldehyde, ortho-phthalaldehyde,	sodium hypochlorite (bleach), calcium hypochlorite, chlorine dioxide	povidone-iodine	hydrogen peroxide/ accelerated HP, peracetic acid, potassium peroxymonosulfate	ortho-phenylphenol, orthobenzylpara-chlorophenol	benzalkonium chloride, alkyldimethyl ammonium chloride
Mechanism of Action	Precipitates proteins; denatures lipids	Alters pH through hydroxyl ions; fat saponification	Denatures proteins; alkylates nucleic acids	Denatures proteins	Denatures proteins	Denature proteins and lipids	Denatures proteins; disrupts cell wall	Denatures proteins; binds phospholipids of cell membrane
Characteristics	• Fast acting • Rapid evaporation • Leaves no residue • Can swell or harden rubber and plastics	• Slow acting • Affected by pH • Best at high temps • Corrosive to metals • Severe skin burns; mucous membrane irritation • Environmental hazard	• Slow acting • Affected by pH and temperature • Irritation of skin/ mucous membrane • Only use in well ventilated areas • Pungent odor • Noncorrosive	• Fast acting • Affected by pH • Frequent application • Inactivated by UV radiation • Corrodes metals, rubber, fabrics, • Mucous membrane irritation	• Stable in storage • Affected by pH • Requires frequent application • Corrosive • Stains clothes and treated surfaces	• Fast acting • May damage some metals (e.g., lead, copper, brass, zinc) • Powdered form may cause mucous membrane irritation • Low toxicity at lower concentrations • Environmentally friendly	• Can leave residual film on surfaces • Can damage rubber, plastic; non-corrosive • Stable in storage • Irritation to skin and eyes	• Stable in storage • Best at neutral or alkaline pH • Effective at high temps • High concentrations corrosive to metals • Irritation to skin, eyes, and respiratory tract
Precautions	Flammable	Very caustic	Formaldehyde has carcinogenic potential	Toxic gas released if mixed with strong acids or ammonia			May be toxic to animals, especially cats and pigs	
Bactericidal	+	+	+	+	+	+	+	+
Virucidal	±[a]	+	±	+	+	+	+	+ Enveloped
Fungicidal	+	+	+	+	+	±	+	+
Tuberculocidal	+	±	+	+	+	±	+	—
Sporicidal	—	+	+	+	±	+	—	+
Factors Affecting Effectiveness	Inactivated by organic matter	Variable	Inactivated by organic matter, hard water, soaps and detergents	Rapidly inactivated by organic matter	Rapidly inactivated by organic matter	Effective in presence of organic matter, hard water, soaps, and detergents	Effective in presence of organic matter, hard water, soaps,and detergents	Inactivated by organic matter, hard water, soaps and anionic detergents

+ = effective; ± = variable or limited activity; — = not effective

a - slow acting against nonenveloped viruses (e.g., norovirus)

REFERENCES: Fraise AP, Lambert PA et al. (eds). *Russell, Hugo & Ayliffe's Principles and Practice of Disinfection, Preservation and Sterilization*, 5th ed. 2013. Ames, IA: Wiley-Blackwell; McDonnell GE. *Antisepsis, Disinfection, and Sterilization: Types, Action, and Resistance*. 2007. ASM Press, Washington DC. Rutala WA, Weber DJ, Healthcare Infection Control Practices Advisory Committee (HICPAC). 2008. Guideline for disinfection and sterilization in healthcare facilities. Available at: *http://www.cdc.gov/hicpac/Disinfection_Sterilization/toc.html*; Quinn PJ, Markey FC et al. (eds). *Veterinary Microbiology and Microbial Disease*. 2nd ed. 2011. West Sussex, UK: Wiley-Blackwell, pp 851-889.

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Figure 20. Characteristics of certain disinfectants (CFSPH 2023)

As per the current PINP (2020) biosecurity protocols (cited below):

- Any clothing that has been in contact with other birds or poultry must be put through a hot wash with laundry detergent before being worn in the penguin colony.

- Any shoes that have foreign material (e.g. mud) not from the Summerland Peninsula should be scrubbed to remove the debris. All shoes must then be sprayed with a 1% bleach solution prior to entering the penguin habitat. The sides and bottom of the shoes should be thoroughly covered.

- If any vehicles are to be taken off road (e.g. on fire breaks) the vehicle must first be properly washed down in the wash-bay facility at the Koala Conservation Reserve. This wash down must include removing all mud and debris from tyres and washing down grills, undercarriages and trays.

- Further precautions need to be undertaken if moving between penguin colonies, both before entering a new colony and upon return. As well as wearing clean clothing and shoes (see above), all penguin weigh bags need to be washed at 60 degrees with laundry detergent and then tumble dried on a hot setting.

- All equipment used, such as callipers and balance scales must be wiped down with 70% alcohol wipes (PINP 2020).

Stressor management and planning for the future

- Exposure to stress, especially chronic stress, may result in negative impacts on the immune system and predispose hosts infection and disease. Subsequently, stressors can affect the disease dynamics and probability of disease outbreaks in wildlife populations (Cromie et al. 2015). They can be multifactorial and additive and vary in their impact on the individual's physiology. Generally speaking, short term or acute stress, such as wild animals experience during capture, handling and sample events, is likely to have fewer negative impacts on individuals than ongoing, chronic stress such as food shortages, prolonged interventions or close proximity to predator species and other threats (Wildlife Health Australia, 2018). Stressors evident in the PINP LP population likely include environmental pollution, reductions in prey abundance, human disturbance for example tourism (albeit currently well managed) and habitat destruction, predation, competition, extreme weather and climate events.

- PINP Management plans have been formulated to identify and manage these stressors, for example the need for habitat protection, including foraging hotspots, consideration of interactions with shipping, controlling impacts of tourism, domesticated animals and road traffic, education of relevant stakeholders including the public, and consideration of penguin conservation and habitat needs in urban development planning processes.

- There are existing efforts by PINP researchers to predict changes to penguin food security and assess ecosystem health within Bass Strait, through the development of ecosystem models (Penguin Foundation 2021). In addition, PINP are involved in the Government's 'Marine Spatial Planning' process for Phillip Island (Millowl) to protect little penguins in Bass Strait (Penguin Foundation 2021). Because the marine ecosystem is frequently changing, extensive collation of data on penguin diet, prey biomass, fishery landings, and primary production, as well as research on other species within the ecosystem is needed. This information is critical to building and strengthening the ecosystem models and the Marine Spatial Plan, which will then be implemented and both used to future proof LPs and other species which share the ecosystem.

Cultural awareness

- The Bunurong and Boon Wurrung Peoples are the Traditional Owners of Summerland Peninsula on Phillip Island (Millowl). There are many culturally sensitivity locations at the site, such as middens (feeding and gathering spots), including at the Penguin Parade. Accordingly, any management actions at the site, including ongoing disease risk management and outbreak responses need to take this into consideration. The disturbance or destruction of an Aboriginal place, including knowingly walking over a midden, is illegal.

- Likewise, it may be beneficial to educate local Elders about the DRA process and findings, which may be pertinent in regards to ongoing fostering of a strong connection to Country.

5.3 Specific sampling, active surveillance and intervention for penguins presented for care

- Ideally, a clinical examination of little penguins being sampled for disease surveillance should be conducted by a veterinarian with avian expertise. The examination should include an assessment of weight, body condition score, age and include a detailed examination of all body systems.

- In the absence of a universally defined body condition scoring system for little penguins, the published scoring system in Magellanic penguins should be used by all personnel for consistency of reporting (Clements and Sanchez 2015) (Figure 21). This is a one to five system, one being emaciated and five being obese. This system was designed for clinical assessment of captive penguins, and wild penguins are most likely to range from 1-3 in the scoring system; however, there would still be value in using this system for the wild LPs at PINP in addition to using body weight. Body weight can vary substantially depending on the duration since the last feed, and muscle mass around the keel is a sensitive clinical indicator of bird body condition in experienced hands.

- Ideally, any observed sick little penguin would be brought to the clinical for examination by a veterinarian with avian expertise; however, if this is not feasible, than this should be focused on those that appear significantly affected (e.g. appear unusually thin for the time of year and age, show specific clinical signs such as incoordination, lameness, external signs of disease such as extensive atypical feather loss, signs of respiratory disease such as marked respiratory discharge, or are unable to stand or walk), or are representative of a broader disease outbreak. See also below for more details for specific conditions. If infectious disease is suspected, consideration should be given to holding such individuals in quarantine to enable further disease investigation and to monitor clinical progression. A quarantine facility would require strict biosecurity isolation practices, which ideally includes (i) dedicated personnel (ii) barrier clothing (overalls, boots, gloves) (iii) dedicated tools (iv) disinfectant footbath at the barrier and (v) disinfection of tools and equipment using a virucidal and bactericidal disinfectant.

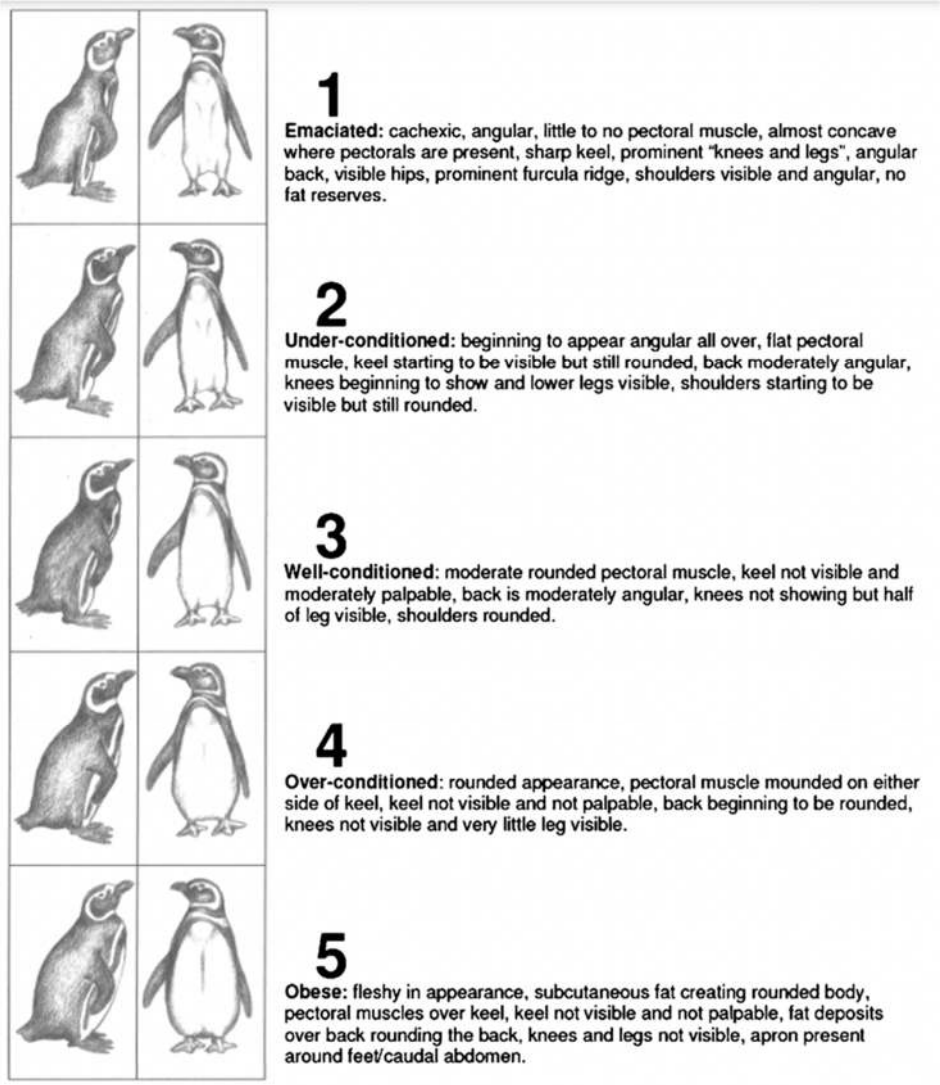

Figure 21. Illustrations and descriptors for categories 1- the most under-conditioned to 5 the most over conditioned (Clements and Sanchez 2015)

Screening and actions for specific clinical presentations are outlined below and summarised in Figure 23 and Table 16. The below recommendations are a guide only and each bird will need to be assessed on a case-by-case basis.

- Any LPs with respiratory signs of disease, including increased respiratory rate and effort, eye discharge, sneezing, eye abnormalities including eyelid swelling, nasal discharge, sneezing, and/or a thin body condition score (BCS) should have swabs collected from the oral cavity and cloaca for avian chlamydia PCR analysis and potentially an oropharyngeal swab for avian cholera *(Pasteurella multocida)* culture and clinical ophthalmological and physical exam.

- If associated with multiple deaths, also consider the possibility of zoonotic highly infectious agents such as APMV-1 and HPAI and undertake appropriate reporting to the state government authorities and veterinarians.

The current recommendations for surveillance for HPAI in wild birds are as outlined below (cited/adapted from Wildlife Health Australia 2023c):

HPAI should be considered as a differential diagnosis in the following scenarios for wild birds:

• Small groups or clusters (5 or more; spatial or temporal clusters) of sick or dead wild birds of any species.

• Individual or sick or dead wild birds: (5 or more) seabirds, waterbirds, shorebirds or birds of prey or any other bird species with signs of avian influenza infection as outlined below.

Infected live birds may show a wide range of clinical signs, including:

• Neurological signs (lack of normal response to stimuli, ataxia (poor coordination), paralysis, seizures, tremors, abnormal posture)

• Respiratory signs (conjunctivitis (inflamed and "weepy" eyes),, increased nasal secretions, oedema (swelling) of the head, dyspnoea (difficulty breathing))

• Gastrointestinal signs (diarrhoea)

• Sudden death

Some species may be asymptomatic or show only very mild clinical signs. In some cases, birds may die suddenly without displaying any clinical signs (WHA 2023d)

- It is recommended that any clinically infected LPs with suspicious respiratory and eye lesions should be isolated and will likely need to be transferred to another facility for further diagnostics and investigation and treatment.
    - ENSURE staff wear appropriate PPE consisting of disposable gloves, overalls, goggles, and an N95 facemask should be worn.
    - All boots, exterior clothing, and equipment should also be decontaminated with a broad spectrum disinfectant such as 70% ethanol, Virkon S, F10, soap + 10% bleach solution, or 0.1% iodine solution.
- LPs presenting with neurological signs including blindness, ataxia, circling, incoordination, nystagmus, head tilt, hind limb paralysis, altered mentation and dysphagia should be screened for *Toxoplasma* infection and potential HPAI and APMV-1 as outlined above. In regards to Toxoplasma, disease screening via the Modified Agglutination Test (MAT) testing would help to determine if actively infected or exposed. Treatment is often unrewarding as once clinical signs are present disease is often advanced and euthanasia of affected individuals should be considered.
- Individuals presented for care with ectoparasites, including fleas, ticks and mites, should be assessed on a case-by-case basis and only treated if the burden is thought to significantly impair their health

(Table 15). Parasite conservation is increasingly being integrated into methods of analysing risk of disease in wild populations (Gompper and Williams 1998, Perez et al. 2006, Sainsbury and Vaughan-Higgins 2012). This is because loss of one parasite species can alter competitive interactions among remaining parasite species (Fernandez and Esch 1991), possibly to the detriment of the host.

- To detect ectoparasites, skin scraping and clinical examination for signs of dermatitis and ectoparasites, should be performed. If indicated, individuals should be treated (Table 15). Ivermectin is effective against ticks, mites and fleas. In moderate to severe infections treatment should be repeated in 2-4 weeks, however, this would require holding in a facility prior to release which may not be practical. Research into alternative parasiticides may be helpful to reduce reliance on repeat treatment with ivermectin. However, even a single treatment will still substantially, albeit potentially only temporarily, reduce parasite burdens. For general parasite treatment regimes, see Table 15.

Table 15. Treatment regimes for parasitism (Dr David Roberts SANCCOB, 2024)

ANTIPARASITIC

Trade Name	Active Ingredient	Concentration	Dose mg/kg	Volume dose & Route	Duration	What is it?	What's it used for?
BAYCOX	Toltrazuril	2.5%m/v 25mg/ml	20mg/kg	**0.8ml/kg Oral**	2 days	Anticoccidial	Treatment of coccidia parasite
IVOMEC oral liquid	Ivermectin	0.08%m/v 0.8mg/ml	0.2mg/kg	**0.25ml/kg**	Q 2 weeks	Anthelmintic	Part of deworming protocol for roundworms and lice
IVOMEC injectable		1%m/v (10mg/ml)	0.5mg/kg	**0.05ml/kg**	Q 10 days		
KARBADUST	Carbaryl	50g/kg	**Dust body surface avoiding beak and eyes**		Once off but can reapply if needed	Insecticide	Ectoparasites on seabirds
MAXILINT	Praziquantel	2.5%m/v 25mg/ml	7.5mg/kg	**0.3ml/kg Oral**	Q 2 weeks	Anthelmintic	Part of deworming protocol for tapeworms
MIRQUIN	Chloroquine	10mg/ml	10mg/kg	**0.5ml/kg Oral**	10 days for avian malaria	Anti-haemoparasite	Avian Malaria protocol
PANACUR	Fenbendazole	50mg/ml	50mg/kg OID	**1ml/kg OID Oral**	3 days	Anthelmintic	Against parasites **Do not use in chicks or moulting birds**
PRIMAQUIN	Primaquin	1.76%m/v	1mg/kg	**0.06ml/kg OID Oral**	5 days for babesiosis 10 days for malaria	Anti-haemoparasite	Treatment of blood parasites

Medication and supplement doses for African Penguins and other seabirds as used by SANCCOB
www.sanccob.co.za – April 2023

SANCCOB

- If ticks are localised (typically around the face, including ear canal, and feet) and thought to bother the presented animal, manual removal by experienced personnel could be considered.

- Ideally a standard health screening protocol (Table 16) for any LP presented for veterinary care or as part of an active disease surveillance system would involve the collection of multiple diagnostic specimens (Table 17). These results would help to inform the DRA and allow hazards and associated management to be adjusted accordingly. Diagnostic screening samples should ideally be batched to minimise costs of shipping. Attempts should be made to collect a thorough suite of samples as recommended (Table 17 and 18). If funding is limited, samples should still be collected and stored, as it is useful to obtain samples which can be retrospectively analysed should a disease event subsequently occur. Alternatively, unwell individuals presenting with clinical signs including dehydration and lethargy should be clinically examined and samples and treatment undertaken as indicated.

- Field examination and collection of samples should include strict application and use of PPE and processes when collecting samples, undertaking post-mortem examinations and disposing of wastes.

- The National Biosecurity Guidelines (Wildlife Health Australia 2018) recommend this to include "strict hand and equipment hygiene prior to and after handling specimens, use of appropriate PPE when handling specimens, placing specimens in appropriate leak-proof containers (such as screw-top vials, plastic bags) for transport and storage, storing specimens at appropriate temperature and in appropriate containers (e.g. fridge, esky with ice), and careful handling to minimise contamination of external surfaces of containers. Cleaning, and if necessary, disinfection, of containers and surfaces, especially prior to re-use, safe and appropriate disposal of products, carcasses and other waste e.g. by clinical waste disposal service, deep burial, incineration, adhering to safe packaging and handling regulations and contacting receiving laboratories prior to the submission of high-risk material."

- Removal of dead carcasses during mortality events is an important management tool for multiple highly infectious aetiologies such as HPAI, APMV-1 and avian cholera. However, it must only be undertaken by trained personnel with appropriate PPE and understanding of the disease and disease transmission.

- Provision of suitable sample storage facilities e.g. -20°C freezer space and -80°C freezer space which is remotely monitored to ensure temperature stability and sample viability is also recommended.

- In the event of a large-scale outbreak, advice should be sought from experts in epidemiology; however, the basic steps in disease investigation generally include:

1) Field history (Work 2015)

- Collect a thorough history/information about the event to rule in or out possible causes of death, such as:
  - Extent, onset, and duration of the outbreak; species, sex, and age classes of animals affected; clinical signs manifested by sick animals; and species not affected by the event.
- Record environmental factors, such as:
  - Unusual weather, changes in land use by animals, changes in habitat management, or distribution of affected animals on the landscape.
- Take photographs of the environment, as well as close up photos or videos of sick or dead animals.
- Summarise data in a mortality event log (see Figure 7).

(2) Collection of tissues from sick or dead animals (necropsy)

- Conduct gross necropsy and histopathological investigation, to help guide laboratory investigations.
- SUITABLE PPE ESSENTIAL
- Collect representative samples of affected individuals
- Fresh tissue samples have a higher likelihood of disease detection, decomposed samples loose tissue architecture and are overgrown with secondary invaders

(3) Laboratory analyses

- Complete post -mortem exam and histopathology useful screening tool to allow further, more specialized testing based on necropsy results.
- Ensure samples when shipped are appropriately packaged at the correct temperature and double bagged to prevent specimen leakage and maintain biosecurity.
- Ensure all samples labelled appropriately with though history provided to laboratory.

(4) Communication of results to stakeholders

- Preliminary results should be distributed to stakeholders once received and may prompt more targeted surveillance to reach further diagnoses.
- Preliminary results may also require further consultation with stakeholders regarding recommendations for biosecurity, containment, isolation, treatment or management of the species and other wildlife, livestock and pet populations including humans.

(5) Implementation of appropriate management actions

- Often requires a multi-disciplinary team to best manage wildlife disease events including but not limited to wildlife veterinarians, veterinary pathologists, park manager and research team, ecologists, biologists, government representatives to ensure recommended actions are logistically sound, safe, feasible and within current recommended state, national and international legislation.

5.4 Specific field protocols for biosecurity and review

- Specific biosecurity guidelines for LPs should be developed and regularly reviewed based on current pathogens of concern.

- Care must be taken to prevent indirect transmission through fomites (objects or materials which are likely to carry infection, such as clothes, utensils, and furniture). All bags, crates, equipment and tools used should be thoroughly cleaned to remove organic matter then disinfected prior to use. For example, at Zoos Victoria transport boxes that have been used for other species are cleaned before use with soap and water then disinfected with F10 (Chemical Essentials, Mitcham North Victoria Australia). New transport bedding material is used e.g. shredded paper substrate. The paper is discarded following use and the transport box scrubbed then cleaned with F10 disinfectant (Jakob-Hoff et al. 2016).

- Staff conducting handling in the field should be competent, aware of fomite transmission and take appropriate precautions. For example, staff should wear clean clothes which have not come into contact with other species wear clean footwear which is disinfected before leaving the site; wash and disinfect hands (or wear gloves) before and after handling penguins; use dedicated tools e.g. nets and weigh bags.

- All staff involved in field work should wear appropriate clothing to minimise the likelihood of mosquito and tick bite e.g., a long-sleeved shirt, long pants tucked into socks and light coloured clothing to make it easier to see ticks on clothes before skin attachment occurs. If field workers have tick bites and there is any difficulty removing the tick or symptoms occur post removal, medical attention should be sought.

- PINP staff / wildlife veterinarians should prepare an avian sampling and emergency field kit for veterinary use (Appendix 3.3). Ideally, a veterinarian with avian expertise should be present during sampling associated with active disease surveillance to enable prompt disease or trauma management as required. This will also allow the veterinarian to connect clinical findings with any diagnostic/surveillance results.

- Handling time should be kept as short as possible to minimise stress to the LPs and handling should cease if birds are showing signs of undue stress, which staff are trained to recognise.

- Any LPs that die in care or during handling, as well as those found freshly dead with unknown cause of death, should have a post-mortem examination undertaken by a veterinarian or pathologist with avian expertise. A range of tissues (including brain, lung, heart, liver, kidney, spleen, pancreas, stomach, small and large intestine, bursa of Fabricius), plus any lesions should be fixed in 10% formalin, and the same complement of samples also stored fresh frozen for further diagnostic testing if indicated. If post-mortem

examination is delayed, seek advice from a veterinarian with avian expertise for advice on storage of the body and any field sampling requirements.

- Regular review of disease risk management, disease screening and necropsy results, survivability, and population size estimates with principal stakeholders should be undertaken. Refinement of ongoing disease risk management should be based on the review which should be formally documented and discussed with relevant staff.

- It is possible that there are other, currently unknown, parasites present in LPs which might cause disease in LPs or other populations of interest at PINP. Therefore, it will be important to monitor the LPs at PINP for these currently unknown hazards through, for example, post-mortem examination of LPs and other species found dead in the vicinity of the site (passive disease surveillance).

- It is also important to understand that the changing environmental factors and external stressors can affect the impact and prevalence of disease in the population, including new, but also existing pathogens. It is important to consider this in relation to future disease management.

5.5 Specific recommendations for data recording

- It is recommended to establish a health database for record keeping, ensuring the approach to managing data is clean, systematic, and enables cross-referencing and a clear audit trail.

- Documents should be easily accessible and ideally online for universal access for example Microsoft OneDrive or a similar web-based server.

- PINP field and health data should be collated into spreadsheets to allow ease of data retrieval and analysis as required.

- In the event of multiple unusual clinical presentations involving morbidity or mortality, a wildlife disease case log should be created to identify demographic data which may aid in disease investigation (Figure 22). Spreadsheet column titles may include factors such as age, sex, species, identification, location found, time of day presented, presenting signs, BCS, weight, diagnostic testing and results of any testing. This will enable spatial analysis of trends for example region/s of interest, time course, species affected.

- Ideally all paper records should be retrospectively transcribed into the electronic database in due course.

- External researchers should submit their raw data and publications to this database for collation and potential future retrospective analysis.

Date of admission	Species	ID	Sex	Weight	Age	location found	Presenting signs								keel trauma (Y/N)	BCS (0-3)	depression/ altered mentation (Y/N)	Fate**	If recovered: number of days to full recovery (consult nurses notes)	Plasma CHE*	Necropsy/histopath findings	If CHIPS case? (fill in at the end of case management)	Comments
							clenching strength in feet (0= none; 3=normal)		able to actively unclench feet (0= none; 3=normal)		ability to withdraw from deep pain (0= none; 3=normal)		ability to move legs (0= none; 3=normal)		cloacal tone (0-3)				D=died E= euthanased R=recovered DOA = dead on arrival					
							Right	Left	Right	Left	Right	Left	Right	Left									
17/10/16	Carnaby's	B1610312	U	526	SA	Martin	0	0	?	?	3	3	?	?	?	N	2.0	Y	R				legs flaccid on arrival but very quick improvement. Some depression, quiet initially

Figure 22. Example of a case log for neurological disease presentation in Carnaby's cockatoos in Western Australia

5.6 Identification of knowledge gaps and specific recommendations for further health research

- Ongoing research should aim to address the knowledge gaps identified in the DRA, this includes:
- Identify outbreaks early in the disease course of avian cholera *Pasteurella multocida,* and further describe the epidemiology in wild birds in Australia.
- Isolate and characterise Chlamydiaceae-like bacteria, and investigate their epidemiology, pathogenicity, and zoonotic potential (Gallo Vaulet et al. 2022).
- Further investigation is required into the presence or absence of tick-borne diseases in Victorian ticks and what is capable of causing disease in LPs, including reference to changing environmental conditions (e.g. climate change).
- Further investigations are needed to determine dose response, toxicokinetics and the impacts of multiple algal biotoxins, including chronic, low-level exposure, LPs and sympatric species.
- There is limited understanding of the type and level of pollutants in the Phillip Island (Millowl) environment and the impact of these pollutants on bird health and populations. Further investigation is required. Unfortunately, laboratory testing can be expensive, and collaborative research with relevant scientists may be a practical way of approaching this. Nonetheless, pollutants are often present at low levels and almost always in combination with other pollutants, in both the environment and animal, making interpretation of positive results challenging. Therefore, a targeted approach based on what has been detected in close proximity for example in Port Phillip Bay is recommended.
- The consequences of releasing rehabilitated individuals back into the wild, after they have potentially been exposed to other species they would usually not be in contact with, should also be acknowledged as a disease risk to the population unless rehabilitated individuals are screened for known, higher risk hazards prior to release and managed with strict biosecurity.
- Ongoing collaboration with universities and local veterinarians to bolster and advance health and disease knowledge of the species is recommended.

5.7 Increasing capacity and training of staff in wildlife health management and collaborations with wildlife health authorities

The following text cites/adapts text from the Ramsar Technical Report Number 7 Ramsar Wetland Disease Manual Guidelines for Assessment, Monitoring and Management of Animal Disease in Wetlands (Cromie et al., 2012).

- Developing capacity to undertake disease management may involve formal education and training of key personnel from wildlife veterinarians for example to land managers, PINP staff and research assistants. Ideally, disease training should be part of other management and biosecurity training to convey its integral nature and to avoid it becoming detached from day-to-day practices.

- Frequency of training will depend on the disease issue e.g. there may be merit in provision of brief annual refresher training for diseases such as HPAI, or to coincide with changes in management practices and legislative Occupational Health and Safety Requirements e.g. fit testing for N95 facemasks. Education and training for those involved in high-risk activities e.g. a large-scale disease control operation, are essential to protect public health (if the disease is zoonotic) and potential for further spread of disease. Communication networks of key stakeholders, including disease control authorities, should be established when 'all is well' to facilitate rapid disease control responses should the need arise.

- Consideration should be given to running a 'mock' disease outbreak to evaluate current management plans and assess the need for further interventions. Lessons learnt from such exercises should be used to further refine and improve your contingency plan. These exercises are essential for building effective teams, ensuring that there are adequate resources and for training staff.

- National (Wildlife Health Australia) and international collaborations with long-standing institutions that have decades of experience in these research areas such as the USGS National Wildlife Health Center could help to rapidly build research capacity in Australia, and foster wildlife and ecosystem health in Australia. This could provide a strategic, efficient, and wise method of collaboration for future disease events.

1) **Identify age group, sex, check for identifiers, note date, time and capture location**
2) **Weigh**

ID: .....
WEIGHT:.....
TIME / DATE OF CAPTURE .....
:.....

3) Clinical examination			Sampling	Action	Clinical notes
Body condition score (BCS) (1- 5)		Body Condition -Pectoral muscles convex– (good BCS) Pectoral muscles concave– (poor BCS)	n/a	Assess with weight, and score 1 (emaciated) to 5 (obese) see Figure 6	
Behaviour & demeanour		Assess for signs of stress associated with capture & handling			
Check all body systems including:	Integument	Examine feathers & overall condition, feathers should be clean, groomed & glossy, note any change to plumage colour. Examine for ectoparasites.	Collect & store ectoparasites in ethanol	Photograph bird & any unusual feathering Assess severity of ectoparasitism –treat only if indicated (moderate to severe burden)	
	Head	Check head–eyes, ears, face, mouth and beak, check top of head for feather loss or abrasions.		Photograph any lesions	
	Ears	Check ears for exudate, obstructions, Ticks or wounds.		Photograph any abnormalities	
	Eyes	Check eyes for pupillary reflex (response to light) and cataracts (lens cloudiness).		Photograph any abnormalities	
	Beak	Check beak (top and bottom) and cere for crusty lesions, swellings, fractures or deformity.		Photograph any abnormalities	
	Oral cavity	Check mouth, choanal slit and tongue for foreign objects, swellings and growths.		Photograph any abnormalities	
	Musculo- skeletal system	Palpation of long bones for any joint crepitus or instability. Examine wings, joints and feather shafts. Palpate legs, toes and nails. Check for joint mobility, grip response, crusty lesions, lumps or swellings.		Photograph any abnormalities	
	Rest of body	Palpate crop and abdomen and note if anything is not bilaterally symmetrical. Examine tail feathers, preengland &cloaca. Check nail length, examine feet for bumblefoot like lesions	Collect faecal if possible	Photograph any abnormalities	

Figure 23. Example of a penguin examination/record sheet

Please circle and annotate below drawings to identify abnormal clinical findings.

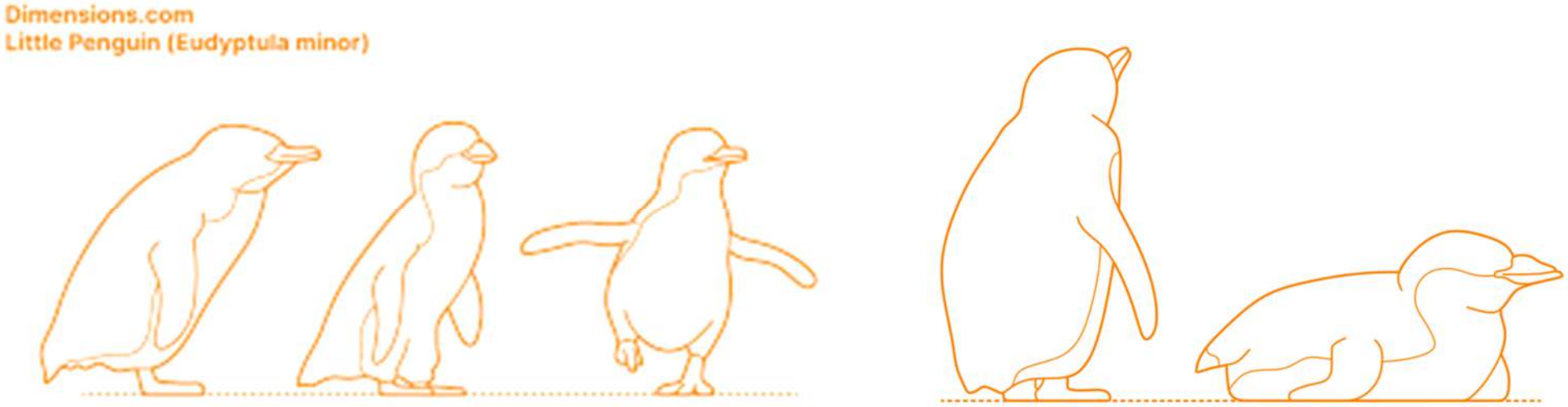

Table 17. Ante-mortem diagnostic testing & screening including sample collection, laboratory and storage details for identified disease hazards

Disease	Test	Sample required	Number of samples	Send to	Store	Cost
*Avian influenza viruses	PCR	Cloacal and oropharyngeal swab	1 of each	AgriBio state laboratory La Trobe University Main Loading Dock 5 Ring Road Bundoora Victoria 3086 (03) 9032 7515	Collect in viral transport medium; store at 4°C until shipping	National wildlife surveillance
*Avian Paramyxovirus APMV-1	PCR	Tracheal and cloacal swab	1 of each	AgriBio state laboratory La Trobe University Main Loading Dock 5 Ring Road Bundoora Victoria 3086 (03) 9032 7515	Collect in viral transport medium	National wildlife surveillance
Avian Chlamydiosis	PCR	Combined dry oral and cloacal swab	1 swab for PCR	APCAH Laboratory Melbourne Veterinary School Dr Alistair Legione B400, 30 Flemington Road Parkville, VIC 3052 0431 847 295	Hold at -80°C until couriered	tbc
Avian cholera *(Pasteurella multocida)*	Culture	Oropharyngeal swab	1 swab for culture	APCAH Laboratory Melbourne Veterinary School Prof Amir Noormohammadi 250 Princes Highway Werribee VIC 3030 03 9731 2275 0428 502 324		tbc
Enteric pathogens - helminths and trematodes	Faecal microscopy,	Fresh faeces - Fresh faeces is best and could be stored in fridge (4°C) for 2-3 days before analysis	5g minimum	Melbourne Veterinary School Prof Abdul Jabbar 250 Princes Highway Werribee VIC 3030 03 9731 2022	Can fix in formalin for later analysis	tbc
Flaviviruses	Whole blood Ticks	Blood collected in serum tube for conventional PCR	0.2ml whole blood	Victorian Infectious Diseases Reference Laboratory 792 Elizabeth Street, Melbourne, 3000	Hold at -80°C until couriered	tbc

				Victoria, Australia T +61 3 9342 9379 F +61 3 9342 9666 E VIDRL.Enquiries@mh.org.au W www.vidrl.org.au		
Intracellular haematozoa – *Babesia, Plasmodium*	peripheral blood smear	Fresh blood smear Whole blood -Nested PCR	1 blood smear	Macquarie University, School of Natural Sciences Prof Michelle Power +61 2 9850 6974	Hold at room temperature	
Toxoplasma gondii	MAT	1ml whole blood	0.5ml serum	Melbourne Veterinary School Jasmin Hufschmid or Abdul Jabbar 250 Princes Highway Werribee VIC 3030 03 9731 2020\03 9731 2022	Can batch and freeze then submit	
General health profile (if clinically compromised)	Blood & blood smear	Blood collected in lithium heparin and always collect a fresh blood smear	0.8ml blood 1 blood smear	Melbourne Veterinary School Clinical Pathology 250 Princes Highway Werribee VIC 3030 Dr Astrid Oscos Snowball 03 9731 2273	Hold at 4°C and ship as soon as possible (within 24 hours or less)	

Table 18. Post-mortem diagnostic testing & screening including sample collection, laboratory and storage details for identified disease hazards

Disease	Test	Sample required	Number of samples	Send to	Store	Cost
Multiple	Post-mortem exam and histopathology	Whole body	As described on necropsy form	Veterinary Anatomic Pathology Melbourne Veterinary School 250 Princes Highway Werribee VIC 3030 03 8344 1269	Can store frozen however then can't submit for histopathology Can store individual tissues frozen Can store tissues in 10% formalin for histopathology	tbc
Algal toxins	LCMS	Proventriculus contents and scrapings of proventricular mucus for liquid chromatography–mass spectrometry for ASP, DSP, NSP, PSP			Can store frozen	tbc
Per- and Polyfluoroalkyl substances (PFAS)	Soil samples Egg samples Blood samples	100 g of soil using polypropylene collection jars and their lids. 1ml whole blood in EDTA		Soil samples -ADE consulting Unit 4/95 Salmon Street, Port Melbourne VIC 3207 1300 796 922 **info@ade.group** Egg and blood samples Ultra Trace Laboratory at the National Institute of Measurement (NMI) 105 Delhi Rd, North Ryde NSW 2113 Phone: +61 2 9449 0111	Samples were stored at 4 °C until processed Plasma was pipetted off and stored at −80 °C in polypropylene tubes until analysis by the	Up to $400 per sample
Persistent organic pollutants (POPs) and other emerging contaminants	Blood (GC–MS/MS)	0.5ml whole blood		Centre for Environmental Sustainability and Remediation (EnSuRe), School of Science, RMIT University, GPO Box 2476, Melbourne, Victoria 3001, Australia	Transferred into solvent-washed polypropylene Eppendorf® tubes and kept cold in the field for 1-4 h, before being frozen at −20 °C until analysis.	tbc

5.8 Recommended minimal sample collection protocol

Definitive diagnosis in wildlife is often difficult and a presumptive diagnosis may be made based on the species and clinical signs (e.g. progressive weakness, respiratory or neurological signs), ruling out other diseases on necropsy as well as taking environmental factors into account (see necropsy submission form).

Combined dry oral and cloacal swab to screen for

- *Chlamydia psittaci*

Separate oral and cloacal swab in VTM for APMV-1 testing
Separate oropharyngeal/tracheal swab in VTM for HPAI testing

Blood collection (2.5ml total volume) using a 26G needle

- Collect blood spot on Whatman's filter paper
- Blood smear
- 0.5ml in a lithium heparin anticoagulant paediatric blood tube for CBC and biochemistry
- 2ml serum store
- 200uL whole blood Flavivirus PCR

Faecal (when possible)

- 3g formalin-fixed (parasitology)
- 2g faecal flotation (parasitology looking for *Contracaecum* and *Mawsonia* eggs)

If collecting external parasites, place into 70% ethanol.

5.8.1 Blood collection

Typically, blood is collected from the inter-digital, medial tarsometatarsal, ulnar, and jugular veins. The jugular is commonly used under general anaesthetic because of the speed and ease of acquisition of large quantities of blood. The medial tarsometatarsal (as shown in Figure 24) is preferred for small volumes and can be safely obtained under manual restraint post appropriate cleaning of the site given likelihood of soiling. The animal's body weight determines the amount of blood that may be taken safely, but normal avian standards are no more than 1% body weight.

Figure 24. Photo of Little penguin feet showing location of medial tarsometatarsal vein for blood collection

5.8.2 Blood smear protocol

Blood smear examination is useful to look at blood cell populations and to detect any haemoparasites. A small droplet of blood is typically placed on the end of the slide closest to the frosted edge and then smeared with the aid of a spreader slide and then allowed to air dry for future analysis (Figure 25). This should be undertaken by someone with relevant experience to avoid wasting precious samples.

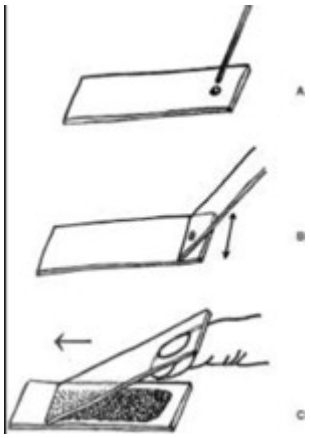

Figure 25. Technique for undertaking a blood smear

5.8.3 Swab collection

Please make sure you discuss the pathogen specific protocol with the relevant diagnostic laboratory prior to collection. However, generally speaking:

Oral swab – The bird should be held with its head up, and the swab is then inserted into the oral cavity; the tip should be directed up into the choanal slit, the swab rotated gently. Swabs that are not tested immediately should be placed into transport medium.

Cloacal swab- The birds should be held gently, and the tail bent back to expose the cloaca. The swab is then gently inserted into the cloaca and gently rotated to collect a small amount of faecal material. Swabs that are not tested immediately should be placed into transport medium.

The below video is a useful guide to ensure an anatomically correct and diagnostic sample
https://www.youtube.com/watch?v=gIbw1SkowfU

5.8.4 Necropsy protocol for Phillip Island (Millowl) little penguins

As of February 2024, HPAI should remain at the forefront of small group or clusters of sick or dead wild birds.

https://wildlifehealthaustralia.com.au/Portals/0/Incidents/HPAI_Advice_for_bird_banders_wildlife_rangers_and_researchers.pdf

High Pathogenicity Avian Influenza and Wild Birds

Advice for bird banders, wildlife rangers and researchers

Version 3.1, December 2023

What to look out for

- Small groups or clusters (5 or more) of sick or dead wild birds of any species.
- Individual or <5 sick or dead wild birds:
  - seabirds, waterbirds, shorebirds or birds of prey
  - any other bird species with signs of avian influenza infection as outlined below.
- Sick or dead wild marine mammals, predators or scavenger species with signs of avian influenza infection as outlined below.

Infected live birds may show a wide range of clinical signs, including:

- incoordination, tremors, swimming in circles
- twisted necks or other abnormal posture
- inability to stand or fly
- diarrhoea
- difficulty breathing, coughing or sneezing
- swelling around the head, neck and eyes
- cloudiness or change in colour of the eyes
- sudden death

Infected live mammals may show a wide range of clinical signs, including:

- incoordination and tremors
- seizures
- difficulty breathing
- nasal discharge or drooling
- death, including the potential for mass mortality events

Can people be infected?

- HPAI can infect people, although infections with the current strains are uncommon and have typically only occurred in people who have had close contact with infected birds. Infection can cause a wide range of symptoms from no symptoms at all, to severe illness and death (see Australian Department of Health and Aged Care).
- Contact with sick or dead birds or other animals should be avoided.
- Contact with land or waterways in which sick or dead birds or other animals have been observed should also be avoided.

- If you develop flu-like symptoms after handling wild birds, contact your health care provider.

What to do

GENERAL ADVICE:

- Become familiar with the clinical signs associated with avian influenza in wild birds.

DURING ROUTINE BIRD HANDLING ACTIVITIES:

- Operate with an increased awareness of the potential risk of disease in wild birds.
- Always maintain good hygiene and biosecurity practices before, during and after working with wildlife (see Hygiene and biosecurity below).
- Where possible, before undertaking activities, observe for any unusual signs of sickness or deaths in wild birds.

IF DEAD OR SICK WILD BIRDS OR WILD MAMMALS ARE OBSERVED:

- Do not handle or remove sick or dead wild birds or mammals unless instructed to do so by government authorities.
- **RECORD** what you see, the species and estimated numbers sick and/or dead, the location (GPS reading), and take photos or videos.
- **REPORT** any unusual signs of disease or mass mortality events in wild birds or mammals (see above) via the Emergency Animal Disease Hotline on 1800 675 888. Reporting will alert authorities to the event so they can evaluate the need for diagnostic testing or other investigation. Even if testing is not undertaken, all reports help inform our understanding of the disease and how to manage it.
- Further actions should only be undertaken following advice from government authorities.

Hygiene and biosecurity

Avian influenza viruses can be transmitted between birds or to other animals via direct contact with respiratory secretions and faecal material, as well as indirect exposure to contaminated environments or objects (e.g. clothing, boots, equipment, etc.).

To prevent spread and protect the health of wildlife, domestic birds, and humans, good hygiene and biosecurity practices should be maintained when visiting sites, moving between sites and when handling wildlife. This includes measures prior to arrival, during activities and after departure.

- **Review and implementation of appropriate biosecurity measures are recommended in the field,** following the National Wildlife Biosecurity Guidelines.
- Use appropriate personal protective equipment (PPE), including gloves, a facemask and eye protection, and ensure PPE is removed properly to avoid self-contamination.
- Particular attention should be given to hand washing after handling birds, after contact with potentially contaminated materials and after removal of gloves. Hands and arms should be washed with abundant soap and warm water. Hand sanitizer (gel with 60 to 90% ethanol concentration) can be applied to reinforce disinfection but should not replace proper handwashing.
- Take care to avoid rubbing eyes or touching the mouth, eating, drinking, or smoking until hands are clean.
- Handle birds in a well-lit and well-ventilated area to minimise the possibility of inhaling dried faecal or other material.
- Use clean bird bags for each bird to minimise the risk of transmitting disease between birds.
- Where practicable, use a clean or new pair of gloves for each bird, especially for species which do not congregate or live in close proximity to each other.

- Clothing, shoes and equipment (e.g. used for capture, handling, marking, holding [e.g. transport boxes/bags]) should be thoroughly cleaned after use, followed by disinfection and waste material (e.g. disposable gloves) should be disposed of appropriately (e.g. sealed in plastic bags).
- Bird banders, wildlife rangers, researchers and other wildlife professionals should avoid contact with domestic birds and poultry for 48 hours after handling wild birds, and should avoid visiting multiple field sites in one day.
- Further advice on PPE and disinfection, directed to people currently working in locations impacted by HPAI, can be found in the WOAH recommendations.

Unusual signs of disease or mass deaths in wildlife

Please remember, if you see any other unusual signs of disease or mass deaths in wildlife you can report it to:

- Your local State/Territory WHA Coordinator
- The 24-hour Emergency Animal Disease Hotline on 1800 675 888
- The Department of Primary Industries or Agriculture in the State/Territory in which the event has occurred.
- Your local veterinarian.

IN OTHER CASES:

- In the event of death or mortality first refrigerate the bird and consult with your local veterinarian and provide history and background to the event.

- If it is less than 24 hours since death and the bird has not been necropsied on-site, the bird should be transported for further processing.

- The bird should be double bagged (ziplock or similar bag which is watertight) and placed in an esky with a frozen ice brick and scrunched up newspaper as filling. Always wear gloves when handling dead birds and a P2 face mask is recommended, particularly if the bird had ocular or respiratory signs prior to death, or the cause of death is unclear.

- The esky should be labelled and a submission form recording the following details: Bird ID, sex, whether adult, juvenile, the date of collection/time of death, any abnormal clinical signs of findings on exam, and what signs were observed prior to death.

- Transport via courier or other means should then be arranged. Discuss relevant history with veterinarian, they will help with transport as needed / organise a submission form and ring the duty pathologist.

- If it is 24-48 hours since death it may or may not be worthwhile sending a fresh bird to the laboratory.

- It will depend on how soon after death the bird was collected / refrigerated, and the environmental conditions, discuss these factors with the veterinarian. If you are unable to speak with veterinarian over 48hrs from death double bag and freeze bird.

- The veterinarian /pathologist will collect a range of tissues (including brain, lung, heart, liver, kidney, spleen, pancreas, stomach, small and large intestine, bursa of fabricius), plus any lesions, fixed in 10% formalin, and the same complement of samples will also be stored fresh frozen for further diagnostic testing if indicated. Histopathology will also be conducted

Hocken (2002) Post-mortem examination of penguins. New Zealand Department of Conservation Science Internal Series 65 is an excellent technical resource for penguin post-mortem examination with useful diagrams
https://www.doc.govt.nz/globalassets/documents/science-and-technical/dsis65.pdf

A modified version of the 2014 Association of Zoos and Aquariums Recommended Penguin Egg, Chick & Adult Bird Necropsy Protocol follows:
https://assets.speakcdn.com/assets/2332/penguin_care_manual_aza_final_2014.pdf

Egg Necropsy:

Note that little penguin eggs remain viable for 7-10 days after abandonment (P. Wasiak, pers. comm.).

1. Refrigerate the egg if there will be a delay before necropsy. Do not freeze eggs or embryos unless the primary goal is virus isolation or bacterial culture, rather than histologic evaluation.
2. Weigh and measure the egg as soon as possible after the embryo is confirmed dead.
   a. Record weight in grams.
   b. Measure length and greatest diameter of egg in centimeters.
3. Describe egg shell characteristics (abnormal shape, shell thickness, presence of cracks, degree of faecal staining, external calcium deposits, etc.).
4. Open the egg by carefully removing the shell overlying the aircell. This can be accomplished with a pair of sharp-blunt scissors, or by gently cracking the shell and removing fragments with forceps.
   a. Examine the aircell membrane for integrity, thickenings, haemorrhages, etc.
5. For small (early stage) embryos, obtain separate swabs of yolk and albumen for culture and cytology. Skip to step 7 for larger embryos.
   a. Peel back the aircell membrane and insert a swab to obtain the albumen culture. Note: if the fluid is watery, it is likely allantoic fluid rather than albumen.
   b. The egg contents may have to be dumped out in order to obtain the yolk cultures.
   c. A second swab of yolk (not a culture swab) may then be taken and rolled onto three microscope slides. The smears should be as thin as possible.

NOTE: Avoid vigorous swabbing of the internal aspect of the yolk sac; hematopoietic cells which reside there may be dislodged and give a false impression that there is inflammation in the yolk sac. Recommended stains include Wright-Giemsa (or Diff-Quik) and gram. Save the third slide for additional stains, if needed.

6. For larger (late stage) embryos, remove enough egg shell to expose the embryo. Note the position of the head relative to other body parts, and in relation to the aircell. The normal position for embryos ready to pip is head under the right wing, with the tip of the beak pointing up toward the aircell.
   a. If the yolk sac is still external (has not retracted into the body cavity), and is accessible, puncture the wall with a sterile scalpel and obtain a culture. If the yolk sac is inaccessible, skip to step 8.
   b. Obtain a second swab of yolk for cytology as described above.
   c. Save the yolk sac (in formalin) for histopathology
   d. Record the colour and consistency (relative thickness or viscosity) of the yolk.
7. Remove the embryo and membranes from the shell by gently dumping the contents into a clean shallow container.
   a. If swabs of yolk for culture and cytology have not yet been collected, obtain them now (as described step 6). Record the colour and consistency (relative thickness or viscosity) of the yolk.
   b. Weigh the embryo with and without the yolk sac (if external).

c. Measure the length of the embryo and if possible estimate the stage of development using The Normal Stages of The Chick as a guideline.
d. Note any external abnormalities, such as musculoskeletal deformities, abnormal skin colour, skin haemorrhages, oedema, dryness, residual albumen, etc. If possible photograph any abnormalities.
e. Record the degree of internalization (retraction) of the yolk sac.
f. Examine the pipping muscle at the back of the neck for oedema or hemorrhages.
g. Note the contents of the mouth, nares, and gizzard.

8. Small embryos along with yolk sac and foetal membranes may be immersed whole in formalin. The volume of formalin should be at least ten times the total volume of the tissues.
9. If the embryo is large enough, conduct a mini-necropsy, retaining representative samples of all organs and tissues for histopathology.
    a. Open the coelomic cavity by making a ventral midline incision with a scalpel or scissors, being careful to avoid tearing the yolk sac if it is internalized.
    b. Proceed with yolk sac cultures and cytology as described under steps 6 and 7 above.
    c. Save the yolk sac (in formalin) for histopathology along with the embryo and membranes. The volume of formalin should be at least ten times the total volume of the tissues.

Chick and Adult Necropsy:

1. Refrigerate the body at 5°C if there will be a delay before necropsy. **Do not freeze the body** unless the primary goal is virus isolation or bacterial culture, rather than histologic evaluation, or neither refrigeration nor immediate necropsy are an option.
2. Record all relevant historical information as indicated on the necropsy form.
3. Weigh the bird as soon as possible after death.

EXTERNAL EXAMINATION:

4. For chicks, note condition of the umbilicus or seal, particularly whether it dry and completely closed.
5. Note any musculoskeletal abnormalities, ectoparasites, evidence of trauma, proliferative skin lesions, etc.
6. Examine the feet carefully for evidence of pododermatitis (bumblefoot).
7. Examine body orifices for patency, exudates, faecal staining around cloaca, etc.
8. Make an evaluation of nutritional condition based on fat stores and relative muscle mass.

INTERNAL EXAMINATION:

9. Make a ventral midline skin incision from the mandible to the cloaca with a sharp scalpel or scissors, being careful to avoid rupturing the yolk sac in young birds.
    a. If the yolk sac ruptures, immediately obtain a yolk culture as the yolk spills out and prepare smears for cytology.
    b. Note the size of the yolk sac and, if sufficient yolk remains, obtain separate swabs for culture and cytology.
10. Remove the keel to expose the thoracic organs. a. Note any accumulations of fluid or exudate in the body cavity and obtain a swab for bacterial and/or fungal culture if appropriate.
11. Obtain blood for smears and bacterial culture by direct heart puncture using a 1 to 3 ml syringe with a 20 to 22 gauge needle.
    a. Prepare at least two blood smears for haemoparasite screening (only a few drops of blood are needed).
    b. If enough blood was obtained, bacterial cultures should be submitted on young birds to rule out septicaemia.

c. If no blood can be obtained from the heart by syringe, smears can be prepared by dabbing the cut surface of the lung or liver onto two or three microscope slides.

Collect the thyroids (with parathyroids), thymus, and spleen for histopathology.

12. Determine gender by examining the gonads prior to removal.
13. Remove the internal organs and examine each systematically.
    a. Obtain samples for histopathology using the tissue list below as a guide. Save samples of all lesions.
    b. Note especially the quantity and nature of the ingesta throughout the GI tract.
    c. The bursa of Fabricius lies dorsal to the cloaca, close to the cloacal orifice (vent). Make sure the bursa does not remain attached to the body when the GI tract is removed.

TISSUE CHECKLIST

All following tissues may be placed together in a single container of 10% neutral buffered formalin.

THE VOLUME OF FORMALIN SHOULD BE 10 TIMES THE VOLUME OF ALL TISSUES COLLECTED.
The tissues should be no thicker than 0.5cm to ensure proper fixation.

- ❑ Skin Muscle (pectoral and thigh)
- ❑ Sciatic nerve (with thigh muscle)
- ❑ Tongue
- ❑ Oesophagus
- ❑ Crop
- ❑ Proventriculus
- ❑ Gizzard
- ❑ Duodenum
- ❑ Jejunum
- ❑ Ileum
- ❑ Caecum
- ❑ Colon
- ❑ Cloaca with Bursa of Fabricius
- ❑ Liver with gallbladder
- ❑ Pancreas
- ❑ Spleen
- ❑ Kidney with Gonad
- ❑ Oviduct
- ❑ Adrenal (with kidney)
- ❑ Thyroid and Parathyroid Thymus
- ❑ Trachea
- ❑ Lung
- ❑ Heart
- ❑ Aorta
- ❑ Pituitary
- ❑ Eye
- ❑ Brain
- ❑ Femoral Bone Marrow

FREEZE PORTIONS OF THE FOLLOWING, IF POSSIBLE, FOR FURTHER TESTING:

- ❑ Liver
- ❑ Spleen
- ❑ Lung
- ❑ Brain

- ❑ Heart
- ❑ Skeletal Muscle

Freeze each tissue separately by placing in separate bags (at least 10 grams of each tissue if large enough). Check with testing laboratory on best storage approach (e.g. foil vs plastic). These tissues can be valuable for ancillary diagnostics. They may be discarded after a diagnosis is reached or stored for potential research or later retrospective analysis.

6 Risk communication

As discussed and reported in Figure 3, effective communication with all relevant stakeholders is central to the success of a wildlife DRA. Prior to the DRA workshop, principal funders of the DRA identified stakeholders and participants with expertise in penguin health, disease, pathology, ecology and biology. Individuals with broader wildlife and livestock health knowledge and government representatives were also invited to participate to help build and shape the DRA. This was done with the intention of creating a report which would have specific recommendations for PINP LPs but could also be extrapolated to other populations globally, and which looked at hazards that, based on expert opinion, were likely to increase in risk in the future (for example climate change and other environmental stressors). We identified potential communication challenges based on recommended risk mitigation and also discussed if there were any experts missing from our round table and online discussions.

The face-to-face workshop was invaluable in the discussion of risk, hazards and prioritisation and achieved most of the aims of the workshop, namely to finalise the hazard list for further risk assessment and facilitate conversation and identify knowledge gaps to build a research plan. As always, it was difficult to arrange mutually agreeable times for international colleagues. However, by organising an online component running simultaneously via Zoom, our international colleagues could connect where possible throughout the process. Keeping to the timeline of the agenda, while promoting discussion is always a challenge and meant certain aspects, such as identification of critical control points (CCPs), were not able to be conducted at the workshop. Ideally, in future a two-day workshop would be ideal, although we also respect two days is a considerable time commitment for stakeholders. Follow up on CCPs was undertaken via email.

We attempted to facilitate group discussion in-person and online. However, this proved challenging for our colleagues in certain time zones and meant they were unable to attend for the full length of the workshop. Nonetheless, following the workshop, our international colleagues have proven invaluable in ongoing support, advice and providing early copies of papers in print to ensure the data presented is both cutting edge and relevant. The value of holding the workshop to build these relationships and foster ongoing collaboration to create an accessible and hopefully useful final written report can not be underestimated. We also invited two individuals, including one student working in wildlife DRA, to teach the process and see the outcomes to assist in the building of capacity in the Australian wildlife DRA space.

A list of identified stakeholders and experts is provided in Table 19.

Challenges in communication

Experts in Australian ticks and tick-borne disease were highlighted as an exclusion of the DRA workshop. However, subsequent collaboration with the Murdoch University One Health Vector and Water-borne Pathogen Research Team has bridged this knowledge gap and may pave the way for future research activities.

Challenges in communication identified included money, time and other resources available. Difficulties around exerting authority locally, when many of the hazards identified required state, national and international environmental management change to effectively mitigate risks were also identified. These challenges are universal and have been identified in previous Australian multi-stakeholder wildlife DRA collaborations and will remain challenges in ongoing DRA implementation and review.

Table 19. Identified stakeholders, experts and researchers

Category	Stakeholder
Phillip Island (Millowl)	Phillip Island Nature Park
	Parks Victoria
	Local council
Community	Local community
	Local veterinarians
	Traditional Owners of Millowl (the Bunurong People)
Government	Victorian Department of Environment, Land, Water and Planning
	Victoria Fisheries Authority
	Agriculture Victoria
University	University of Melbourne Veterinary School experts in One Health, wildlife pathology, parasitology
Surrounding industry	Commercial companies e.g. fishermen, tourism operators
Other Institutions	Australian Centre for Disease Preparedness (formerly AAHL)
	Birdlife Australia
	Commonwealth Scientific and Industrial Research Organisation
	Countries in migration flyways
	Universities
	Wildlife Health Australia
	Zoos Victoria veterinarians

The most time sensitive and current high-risk hazard, HPAI, will require high level and significant ongoing communication with state and national government officials should the recommendation for vaccination be pursued. This recommendation will require significant support from multiple stakeholders, including the PINP Board to enable further actioning and would also ideally involve a vaccination trial in captive penguins to assess efficacy in LPs via measuring antibody responses. It is currently thought that the risks of vaccination, for example stressors associated with manual handling, identification and restraint, are effectively counter balanced by the perceived benefits of immunity provided through vaccination and would result in significantly reduced morbidity and mortality based on experiences in similar species globally. However, to facilitate the trial, government support (via permits) as well as multi-institutional support will be required, including ethics approvals and centralised reporting and evaluation of the trial. Based on international expert opinion, vaccination is thought to be the only available risk mitigation tool that will minimise morbidity and mortality. The WOAH now also endorses emergency vaccination for wildlife in certain circumstances (WOAH 2023), so this recommendation and document should provide some high-level justification for consideration of vaccination for the highly charismatic and economically important little penguin population of Phillip Island (Millowl).

Communication about the DRA process and the importance of wildlife DRAs in providing a framework to manage and mitigate identified hazards in our changing climate is of national significance. Ensuring the PINP staff are aware of and educated about these potential hazards and have the tools to best manage these identified risks is also instrumental in reducing the likelihood of significant disease incursion. This document aims to identify and mitigate these hazards and set up a broader scale systematic approach to health monitoring through necropsy investigations and targeted surveillance of hazards while also identifying knowledge gaps and future research priorities for the populations of interest at PINP. Implementation of these processes will undoubtedly require ongoing review and collaboration and is an iterative process.

A DRA is a living, evolving document reliant on stakeholder collaboration and communication to achieve its purpose. We have provided a structured, evidence-based and iterative approach to assessing the disease risks of the PINP LP population. We have transparently assessed both published and unpublished information through expert and stakeholder consultation to identify 80 hazards of concern. We have built on current protocols and biosecurity practices and conducted detailed risk assessment of 15 high and medium risk

hazards. We have formulated practical disease risk management guidelines and strongly recommend annual review of disease threats and recommendations to monitor and evaluate effectiveness of risk mitigation measures. Ideally, this document, while identifying the importance of disease and infectious disease risk, will also aim to promote the health of the population through acknowledging the importance of non-infectious disease risks which in many accounts can be more problematic to manage and require large scale multi-national, longitudinal, management strategies.

There are processes that drive penguin survival that are essentially 'natural' and those that are anthropogenically altered. There is little that can be done to manage natural cycles and processes governing fish die-offs and marine productivity in general, however, if there is an anthropogenic element to the origin, frequency or extent of these events, then management may be feasible (Dann 1996. It is also important to understand that the changing environmental factors and the presence of external stressors in the future could lead to changes in the clinical expression of disease. Disease may therefore become more or less apparent relative to environmental change, and this needs to be considered when managing the species into the future.

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8 Appendices

8.1 Resident species on Phillip Island (Millowl) (Phillip Island Nature Park Management Plan 2006-2011)

appendix 3 – bird species occurring on phillip island

The following bird species have been identified within the Nature Park. Their status under the Flora and Fauna Guarantee Act 1988 (FFG), JAMBA (Japan Australia Migratory Bird Agreement) and CAMBA (China Australia Migratory Bird Agreement) (J/C), Victorian Status (DSE 2003), and listing under the EPBC Act (1999), is specified.

ORDER Family Common name	*Scientific Name*	FFG	J/C	Victorian Status	EPBC
STRUTHIONIFORMES					
Casuariidae					
Emu (locally extinct)	*Dromaius novaehollandiae*				
GALLIFORMES					
Phasianidae					
Stubble Quail	*Cotumix pectoralis*				
Brown Quail	*Cotumix ypsilophora*			NT	
ANSERIFORMES					
Anseranatidae					
Magpie Goose	*Anseronas semipalmata*	Listed		VU	Listed
Anatidae					
Blue-billed Duck	*Oxyura australis*	Listed		EN	
Musk Duck	*Biziura lobata*			EU	Listed
Black Swan	*Cygnus atratus*				
Greylag Goose*	*Anser anser var. domesticus*				
Cape Barren Goose	*Cereopsis novaehollandiae*			NT	Listed
Australian Shelduck	*Tadorna tadornoides*				
Australian Wood Duck	*Chenonetta jubata*				
Pacific Black Duck	*Anas superciliosa*				
Australasian Shoveler	*Anas rhynchotis*				
Grey Teal	*Anas gracilis*				
Chestnut Teal	*Anas castanea*				
Pink-eared Duck	*Malacorhynchus membranaceus*				
Hardhead Duck	*Aythya australis*				
PODICIPEDIFORMES					
Podicipediae					
Australasian Grebe	*Tachybaptus novaehollandiae*				
Hoary-headed Grebe	*Poliocephalus poliocephalus*				
Great Crested Grebe	*Podiceps cristatus*				
SPHENISCIFORMES					
Spheniscidae					
Rockhopper Penguin	*Eudyptes chrysocome*				Listed
Fiordland Penguin	*Eudyptes pachyrhyncus*				Listed
Erect-crested Penguin	*Eudyptes sclateri*				Listed
Little Penguin	*Eudyptula minor*				Listed
Magellanic Penguin	*Spheniscus magellanicus*				Listed
PROCELLARIIFORMES					
Procellariidae					
Common Diving-Petrel	*Pelecanoides urinatrix*			NT	
Southern Giant-Petrel	*Macronectes giganteus*	Listed (A)		VU	Threatened Species, Endangered Migratory
Northern Giant-Petrel	*Macronectes halli*	Listed (A)		NT	Threatened Species, Endangered Migratory
Southern Fulmar	*Fulmarus glacialoides*				Listed
Cape Petrel	*Daption capense*				Listed
Kerguelen Petrel	*Lugensa brevirostris*				Listed
Great-winged Petrel	*Pterodroma macroptera*				Listed
White-headed Petrel	*Pterodroma lessonii*				Listed
Mottled Petrel	*Pterodroma inexpectata*				Listed
Gould's Petrel	*Pterodroma leucoptera*				Listed
Blue Petrel	*Halobaena caerulea*				
Broad-billed Prion	*Pachyptila vittata*				Listed
Salvin's Prion	*Pachyptila salvini*				Listed
Antarctic Prion	*Pachyptila desolata*				Listed
Slender-billed Prion	*Pachyptila belcheri*				Listed
Fairy Prion	*Pachyptila turtur*			VU	Listed
Flesh-footed Shearwater	*Puffinus carneipes*				Listed
Sooty Shearwater	*Puffinus griseus*				Listed
Short-tailed Shearwater	*Puffinus tenuirostris*				Migratory
Fluttering Shearwater	*Puffinus gavia*				Listed
Hutton's Shearwater	*Puffinus huttoni*				Listed
Diomedeidae					
Wandering Albatross	*Diomedea exulans*	Listed (A)		EN	Threatened Species, Vulnerable Migratory
Southern Royal Albatross	*Diomedea epomophora*	Listed (A)		EN	Threatened Species, Vulnerable Migratory

ORDER Family Common name	Scientific Name	FFG	J/C	Victorian Status	EPBC
Black-browed Albatross	*Diomedea melanophris*			EN	Threatened Species, Vulnerable Migratory
Shy Albatross	*Diomedea cauta*	Listed		VU	Threatened Species, Vulnerable Migratory
Grey-headed Albatross	*Diomedea chrysostoma*	Listed (A)		VU	Threatened Species, Vulnerable Migratory
Yellow-nosed Albatross	*Diomedea chlororhynchos*	Listed		VU	Threatened Species, Vulnerable Migratory
Hydrobatidae					
Wilson's Storm-Petrel	*Oceanites oceanicus*		J		Listed
White-faced Storm-Petrel	*Pelagodroma marina*			NT	Listed
PELECANIFORMES					
Sulidae					
Australasian Gannet	*Morus serrator*				Listed
Anhingidae					
Darter	*Anhinga melanogaster*				
Phalacrocoracidae					
Little Pied Cormorant	*Phalacrocorax melanoleucos*				
Black-faced Cormorant	*Phalacrocorax fuscescens*			NT	Listed
Pied Cormorant	*Phalacrocorax varius*			NT	
Little Black Cormorant	*Phalacrocorax sulcirostris*				
Great Cormorant	*Phalacrocorax carbo*				
Pelecanidae					
Australian Pelican	*Pelecanus conspicillatus*				Listed
Fregatidae					
Frigatebird species	*Fregata spp.*				Listed
CICONIIFORMES					
Ardeidae					
White-faced Heron	*Egretta novaehollandiae*				
Little Egret	*Egretta garzetta*	Listed		EN	Listed
Eastern Reef Egret	*Egretta sacra*				Listed
White-necked Heron	*Ardea pacifica*				
Great Egret	*Ardea alba*			VU	Listed
Cattle Egret	*Ardea ibis*		J/C		Listed

ORDER Family Common name	Scientific Name	FFG	J/C	Victorian Status	EPBC
Nankeen Night Heron	*Nycticorax caledonicus*			NT	Listed
Australasian Bittern	*Botaurus poiciloptilus*	Listed		EN	
Threskiornithidae					
Glossy Ibis	*Plegadis falcinellus*			NT	Listed
Australian White Ibis	*Threskiornis molucca*				Listed
Straw-necked Ibis	*Threskiornis spinicollis*				Listed
Royal Spoonbill	*Platalea regia*			VU	
Yellow-billed Spoonbill	*Platalea flavipes*				
FALCONIFORMES					
Accipitridae					
Osprey	*Pandion haliaetus*				Listed
Black-shouldered Kite	*Elanus axillaris*				
Letter-winged Kite	*Elanus scriptus*				
Whistling Kite	*Haliastur sphenurus*				Listed
White-bellied Sea-Eagle	*Haliaeetus leucogaster*	Listed		VU	Migratory
Swamp Harrier	*Circus approximans*				Listed
Brown Goshawk	*Accipiter fasciatus*				Listed
Grey Goshawk	*Accipiter novaehollandiae*			VU	
Wedge-tailed Eagle	*Aquila audax*				
Little Eagle	*Hieraaetus morphnoides*				
Brown Falcon	*Falco berigora*				
Australian Hobby	*Falco longipennis*				
Black Falcon	*Falco subniger*			VU	
Peregrine Falcon	*Falco peregrinus*				
Nankeen Kestrel	*Falco cenchroides*				Listed
GRUIFORMES					
Rallidae					
Buff-banded Rail	*Gallirallus philippensis*				Listed
Lewin's Rail	*Rallus pectoralis*	Listed		VU	
Baillon's Crake	*Porzana pusilla*	Listed		VU	
Australian Spotted Crake	*Porzana fluminea*				
Purple Swamphen	*Porphyrio porphyrio*				Listed
Dusky Moorhen	*Gallinula tenebrosa*				
Eurasian Coot	*Fulica atra*				
TURNICIFORMES					
Turnicidae					
Painted Button-quail	*Turnix varia*				

ORDER Family Common name	Scientific Name	FFG	J/C	Victorian Status	EPBC
CHARADRIIFORMES					
Scolopacidae					
Latham's Snipe	*Gallinago hardwickii*			NT	Migratory
Black-tailed Godwit	*Limosa limosa*		J/C	VU	Migratory
Bar-tailed Godwit	*Limosa lapponica*		J/C		Migratory
Whimbrel	*Numenius phaeopus*		J/C	VU	Migratory
Eastern Curlew	*Numenius madagascariensis*		J/C	NT	Migratory
Common Greenshank	*Tringa nebularia*				Migratory
Wood Sandpiper	*Tringa glareola*		J/C	VU	Listed
Common Sandpiper	*Actitis hypoleucos*		J/C	VU	Listed
Grey-tailed Tattler	*Heteroscelus brevipes*	Listed	J/C	CR	Migratory
Ruddy Turnstone	*Arenaria interpres*		J/C		Listed
Great Knot	*Calidris tenuirostris*	Listed	J/C	EN	Listed
Red Knot	*Calidris canutus*		J/C	NT	Listed
Sanderling	*Calidris alba*		J/C	NT	Listed
Red-necked Stint	*Calidris ruficollis*		J/C		Migratory
Pectoral Sandpiper	*Calidris melanotos*		J	NT	Listed
Sharp-tailed Sandpiper	*Calidris acuminata*		J/C		Listed
Curlew Sandpiper	*Calidris ferruginea*		J		Migratory
Burhinidae					
Bush Stone-curlew	*Burhinus grallarius*			EN	Listed
Haematopodidae					
Pied Oystercatcher	*Haematopus longirostris*				
Sooty Oystercatcher	*Haematopus fuliginosus*			NT	
Recurvirostridae					
Black-winged Stilt	*Himantopus himantopus*				Listed
Charadriidae					
Pacific Golden Plover	*Pluvialis fulva*		J/C	NT	Migratory
Grey Plover	*Pluvialis squatarola*		J/C	NT	Listed
Red-capped Plover	*Charadrius ruficapillus*				
Double-banded Plover	*Charadrius bicinctus*				Migratory
Black-fronted Dotterel	*Elseyornis melanops*				
Hooded Plover	*Thinornis rubricollis*	Listed (A)		VU	Listed
Banded Lapwing	*Vanellus tricolor*				
Masked Lapwing	*Vanellus miles*				
Laridae					
Great Skua	*Catharacta skua*				Listed
Pomarine Jaeger	*Stercorarius pomarinus*		J/C		Listed
Arctic Jaeger	*Stercorarius parasiticus*		J		Listed
Pacific Gull	*Larus pacificus*			NT	Listed
Kelp Gull	*Larus dominicanus*				Listed
Silver Gull	*Larus novaehollandiae*				Listed
Caspian Tern	*Sterna caspia*	Listed	C	NT	Listed
Crested Tern	*Sterna bergii*		J		Listed
White-fronted Tern	*Sterna striata*			NT	Listed
Fairy Tern	*Sterna nereis*	Listed		EN	Listed
Little Tern	*Sterna albifrons*	Listed		VU	Listed
COLUMBIFORMES					
Columbidae					
Rock Dove*	*Columba livia*				
Spotted Turtle-Dove*	*Streptopelia chinensis*				
Common Bronzewing	*Phaps chalcoptera*				
Brush Bronzewing	*Phaps elegans*				
Crested Pigeon	*Ocyphaps lophotes*				
PSITTACIFORMES					
Cacatuidae					
Gang-gang Cockatoo	*Callocephalon fimbriatum*				
Galah	*Cacatua roseicapilla*				
Sulphur-crested Cockatoo	*Cacatua galerita*				
Cockatiel	*Nymphicus hollandicus*				
Psittacidae					
Rainbow Lorikeet	*Trichoglossus haematodus*				
Musk Lorikeet	*Glossopsitta concinna*				
Scaly-breasted Lorikeet	*Trichoglossus chlorolepidotus*				
Little Lorikeet	*Glossopsitta pusilla*				
Australian King-parrot	*Alisterus scapularis*				
Crimson Rosella	*Platycercus elegans*				
Eastern Rosella	*Platycercus eximius*				
Australian Ringneck	*Barnadius zonarius*				
Swift Parrot	*Lathamus discolor*	Listed		EN	Threatened Species, Endangered
Blue-winged Parrot	*Neophema chrysostoma*				Listed
Orange-bellied Parrot	*Neophema chrysogaster*	Listed (A)		CR	Threatened Species, Endangered

ORDER Family Common name	Scientific Name	FFG	J/C	Victorian Status	EPBC
CUCULIFORMES					
Cuculidae					
Pallid Cuckoo	*Cuculus pallidus*				Listed
Fan-tailed Cuckoo	*Cacomantis flabelliformis*				Listed
Horsfield's Bronze-Cuckoo	*Chrysococcyx basalis*				Listed
Shining Bronze-Cuckoo	*Chrysococcyx lucidus*				Listed
STRIGIFORMES					
Strigidae					
Powerful Owl	*Ninox strenua*	Listed		VU	
Barking Owl	*Ninox connivens*	Listed		EN	
Southern Boobook	*Ninox novaeseelandiae*				Listed
Tytonidae					
Barn Owl	*Tyto alba*				
CAPRIMULGIFORMES					
Podargidae					
Tawny Frogmouth	*Podargus strigoides*				
Aegothelidae					
Australian Owlet-nightjar	*Aegotheles cristatus*				
APODIFORMES					
Apodidae					
White-throated Needletail	*Hirundapus caudacutus*				Migratory
Fork-tailed Swift	*Apus pacificus*				Listed
CORACIIFORMES					
Alcedinidae					
Azure Kingfisher	*Alcedo azurea*				
Halcyonidae					
Laughing Kookaburra	*Dacelo novaeguineae*				
Sacred Kingfisher	*Todiramphus sanctus*				Listed
PASSERIFORMES					
Maluridae					
Superb Fairy-wren	*Malurus cyaneus*				
Southern Emu-wren	*Stipiturus malachurus*				
Pardalotidae					
Spotted Pardalote	*Pardalotus punctatus*				
Striated Pardalote	*Pardalotus striatus*				
White-browed Scrubwren	*Sericornis frontalis*				
Striated Fieldwren	*Calamanthus fuliginosus*				
Brown Thornbill	*Acanthiza pusilla*				
Yellow-rumped Thornbill	*Acanthiza chrysorrhoa*				
Yellow Thornbill	*Acanthiza nana*				
Striated Thornbill	*Acanthiza lineata*				
Meliphagidae					
Red Wattlebird	*Anthochaera carunculata*				
Little Wattlebird	*Anthochaera chrysoptera*				
Spiny-cheeked Honeyeater	*Acanthagenys rufogularis*				
Noisy Friarbird	*Philemon corniculatus*				
Noisy Miner	*Manorina melanocephala*				
Yellow-faced Honeyeater	*Lichenostomus chrysops*				
Singing Honeyeater	*Lichenostomus virescens*				
White-eared Honeyeater	*Lichenostomus leucotis*				
Yellow-tufted Honeyeater	*Lichenostomus melanops*				
White-plumed Honeyeater	*Lichenostomus penicillatus*				
Brown-headed Honeyeater	*Melithreptus brevirostris*				
White-naped Honeyeater	*Melithreptus lunatus*				
Crescent Honeyeater	*Phylidonyris pyrrhoptera*				
New Holland Honeyeater	*Phylidonyris novaehollandiae*				
Eastern Spinebill	*Acanthorhynchus tenuirostris*				
White-fronted Chat	*Epthianura albifrons*				
Petroicidae					
Jacky Winter	*Microeca fascinans*				
Scarlet Robin	*Petroica multicolor*				
Rose Robin	*Petroica rosea*				
Flame Robin	*Petroica phoenicea*				Listed
Pink Robin	*Petroica rodinogaster*				Listed
Eastern Yellow Robin	*Eopsaltria australis*				
Cinclosomatidae					
Spotted Quail-thrush	*Cinclosoma castanotus*			NT	
Pachycephalidae					
Crested Shrike-tit	*Falcunculus frontatus*				
Golden Whistler	*Pachycephala pectoralis*				
Rufous Whistler	*Pachycephala rufiventris*				
Grey Shrike-thrush	*Colluricincla harmonica*				
Dicruridae					
Leaden Flycatcher	*Myiagra rubecula*				
Satin Flycatcher	*Myiagra cyanoleuca*				Migratory
Magpie-lark	*Grallina cyanoleuca*				Listed
Rufous Fantail	*Rhipidura rufifrons*				Migratory
Grey Fantail	*Rhipidura fuliginosa*				
Willie Wagtail	*Rhipidura leucophrys*				

ORDER Family Common name	Scientific Name	FFG	J/C	Victorian Status	EPBC
Spangled Drongo	*Dicrurus bracteatus*				
Campephagidae					
Black-faced Cuckoo-shrike	*Coracina novaehollandiae*				Listed
White-bellied Cuckoo-shrike	*Coracina papuensis*				Listed
White-winged Triller	*Lalage sueurii*				
Oriolidae					
Olive-backed Oriole	*Oriolus sagittatus*				
Artamidae					
White-browed Woodswallow	*Artamus superciliosus*				
Dusky Woodswallow	*Artamus cyanopterus*				
Grey Butcherbird	*Cracticus torquatus*				
Australian Magpie	*Gymnorhina tibicen*				
Pied Currawong	*Strepera graculina*				
Grey Currawong	*Strepera versicolor*				
Corvidae					
Australian Raven	*Corvus coronoides*				
Little Raven	*Corvus mellori*				Listed
House Crow*	*Corvus splendens*				
Alaudidae					
Skylark*	*Alauda arvensis*				
Motacillidae					
Richard's Pipit	*Anthus novaeseelandiae*				Listed
Passeridae					
House Sparrow*	*Passer domesticus*				
Eurasian Tree Sparrow*	*Passer montanus*				
Red-browed Finch	*Neochmia temporalis*				
Chestnut-breasted Mannikin	*Lonchura castaneothorax*				
Fringillidae					
European Greenfinch*	*Carduelis chloris*				
European Goldfinch*	*Carduelis carduelis*				
Dicaeidae					
Mistletoebird	*Dicaeum hirundinaceum*				
Hirundinidae					
Welcome Swallow	*Hirundo neoxena*				Listed
Tree Martin	*Hirundo nigricans*				Listed
Fairy Martin	*Hirundo ariel*				
Sylviidae					
Clamorous Reed-Warbler	*Acrocephalus stentoreus*				Listed

ORDER Family Common name	Scientific Name	FFG	J/C	Victorian Status	EPBC
Little Grassbird	*Megalurus gramineus*				
Brown Songlark	*Cincloramphus cruralis*				
Golden-headed Cisticola	*Cisticola exilis*				
Zosteropidae					
Silvereye	*Zosterops lateralis*				Listed
Muscicapidae					
Bassian Thrush	*Zoothera lunulata*				
Common Blackbird*	*Turdus merula*				
Sturnidae					
Common Starling*	*Sturnus vulgaris*				
Common Myna*	*Acridotheres tristis*				

Key to abbreviations

J=Japan-Australia Migratory Birds Agreement (JAMBA)

C=China-Australia Migratory Birds Agreement (CAMBA)

Victorian Categories – Vertebrate Fauna

- **CR- Critically Endangered:** A taxon is Critically Endangered when it is facing an extremely high risk of extinction in the wild in the immediate future.
- **EN- Endangered:** A taxon is Endangered when it is not Critically Endangered but is facing a high risk of extinction in the wild in the immediate future.
- **VU- Vulnerable:** A taxon is Vulnerable when it is not Critically Endangered or Endangered but is facing a high risk of extinction in the wild in the medium-term future.
- **NT- Low risk- near threatened:** A taxon is Low Risk- near threatened when it has been evaluated, does not satisfy the criteria for any of the threatened categories, but which is close to qualifying for Vulnerable. In practice, these species are most likely to move into a threatened category if the population decline continues or catastrophes befall the species.

Flora and Fauna Guarantee Act 1988 *(Threatened List February 2006)*

- **Listed:** Listed under the Act
- **Nominated:** Nominated for listing under the Act
- **Recommended:** Recommended for listing under the Act
- **Rejected:** Rejected for listing under the Act
- **(A)** indicates an action statement has been prepared

appendix 4 – mammal species occurring on and near phillip island

The following mammal species have been identified on and near Phillip Island. Their status under the *Flora and Fauna Guarantee Act 1988* (FFG), Victorian Status (DSE 2003), and listing under the *Environmental Protection Biodiversity and Conservation Act 1999* (EPBC), is specified.

ORDER Family Common name	*Scientific Name*	FFG	Victorian Status	EPBC
CARNIVORA				
Canidae				
Red Fox	*Vulpes vulpes*		Declared vermin	
Felidae				
Cat (feral)	*Felis catus*		Considered to be a serious threat to wildlife	
CETACEA				
Balaenidae				
Southern Right Whale	*Eubalaena australis*	Listed	Critically endangered	Threatened Species, Endangered
Balaenopteridae				
Fin Whale	*Balaenoptera physalus*		Protected numbers low	Vulnerable
Humpback Whale	*Megaptera novaeangliae*	Listed	Endangered	Threatened Species, Endangered
Delphinidae				
Common Dolphin	*Delphinus delphinus*		Common and protected	
Bottlenose Dolphin	*Tursiops truncatus*		Common and protected	
Long-finned Pilot Whale	*Globicephala melas*		Protected	
Killer Whale	*Orcinus orca*		Protected	Migratory
Kogiidae				
Pygmy Sperm Whale	*Kogia breviceps*		Protected	Migratory
Physeteridae				
Sperm Whale	*Physeter macrocephalus*		Protected	
Physeteridaeiphiidae				
Southern Beaked Whale	*Mesoplodon grayi*		Protected	
CHIROPTERA				
Pteropodidae				
Grey-headed Flying-fox	*Pteropus poliocephalus*	Listed	Vulnerable	Threatened Species, Vulnerable
Vespertilionidae				
Gould's Wattled Bat	*Chalinolobus gouldii*		Secure	
Chocolate Wattled Bat	*Chalinolobus morio*		Secure	
Lesser Long-eared Bat	*Nyctophilus geoffroyi*		Widespread and common	
Common Bent-wing Bat	*Miniopterus schreibersii*	Listed	Vulnerable	
Large Forest Bat	*Vespadelus darlingtoni*		Widespread and common	
Southern Forest Bat	*Vesadelus regulus*		Common and secure	
Little Forest Bat	*Vespadelus vulturnus*		Common and secure	
White-striped Freetail Bat	*Tararida australis*		Abundant	
Eastern False Pipistrelle	*Falsistrellus tasmaniensis*		Uncommon	
DIPROTODONTIA				
Macropodidae				
Eastern Grey Kangaroo	*Macropus giganteus*		Abundant throughout range	
Swamp Wallaby	*Wallabia bicolor*		Common	
Phalangeridae				
Common Brushtail Possum	*Trichosurus vulpecula*		Common	
Phascolarctidae				
Koala	*Phascolarctos cinereus*		Secure (introduced onto Phillip Island)	
Pseudocheiridae				
Common Ringtail Possum	*Pseudocheirus peregrinus*		Abundant	
LAGOMORPHA				
Leporidae				
Brown Hare	*Lepus capensis*		Declared vermin	
European Rabbit	*Oryctolagus cuniculus*		Declared vermin	
MONOTREMATA				
Tachyglossidae				
Short-beaked Echidna	*Tachyglossus aculeatus*		Not considered to be threatened	
PINNIPEDIA				
Otariidae				
New Zealand Fur Seal	*Arctocephalus forsteri*		Secure	Listed
Australian Fur Seal	*Arctocephalus pusillus doriferus*		Vulnerable	Listed
Subantarctic Fur Seal	*Arctocephalus tropicalis*		Secure within protected Victorian reserves	Listed
Australian Sea Lion	*Neophoca cinerea*		Secure	Listed
Phocidae				
Leopard Seal	*Hydrurga leptonyx*		Secure	Listed
Southern Elephant Seal	*Mirounga leonina*		Rare and protected	Listed

ORDER Family Common name	*Scientific Name*	FFG	Victorian Status	EPBC
RODENTIA				
Muridae				
Water Rat	Hydromys chrysogaster		Protected, reasonably common	
House Mouse	Mus musculus		Introduced pest	
Brown Rat	Rattus norvegicus		Declared vermin	
Black Rat	Rattus rattus		Declared vermin	

Data from the Atlas of Victorian Wildlife (24 March 2004), supplemented with data from the Nature Park.
Conservation Status in Victoria (DSE 2003a). Categories:

Victorian Categories - Vertebrate Fauna

- **Critically Endangered:** A taxon is Critically Endangered when it is facing an extremely high risk of extinction in the wild in the immediate future.
- **Endangered:** A taxon is Endangered when it is not Critically Endangered but is facing a high risk of extinction in the wild in the immediate future.
- **Vulnerable:** A taxon is Vulnerable when it is not Critically Endangered or Endangered but is facing a high risk of extinction in the wild in the medium-term future.
- **Low risk- near threatened:** A taxon is Low Risk- near threatened when it has been evaluated, does not satisfy the criteria for any of the threatened categories, but which is close to qualifying for Vulnerable. In practice, these species are most likely to move into a threatened category if the population decline continues or catastrophes befall the species.
- **Data Deficient:** A taxon is Data Deficient when there is inadequate information to make a direct or indirect assessment of its risk of extinction based on its distribution or population status. Listing of taxa in this category indicates that more information is required and acknowledges the possibility that future investigation will show that a threatened classification is appropriate.

Flora and Fauna Guarantee Act 1988

- **Listed:** Listed under the Act
- **Nominated:** Nominated for listing under the Act
- **Recommended:** Recommended for listing under the Act
- **Rejected:** Rejected for listing under the Act

References

'Museum Victoria, Melbourne, Australia (2002). Bioinformatics Victorian Faunal Web Site. Published on the Internet; http://www.museum.vic.gov.au/bioinformatics/ [accessed 23 February 2006].

reptiles, amphibian & freshwater fish species (phillip island)

The following amphibian and reptile species have been identified on and near Phillip Island. Their status under the *Flora and Fauna Guarantee Act 1988* (FFG), Victorian Status (DSE 2003), and listing under the *Environmental Protection Biodiversity and Conservation Act 1999*, is specified.

Order Family Common name	*Scientific Name*	FFG	Victorian Status	EPBC
FROGS				
ANURA				
Hylidae				
Southern Brown Tree Frog	*Litoria ewingii*			
Growling Grass Frog	*Litoria raniformis*	Listed	Threatened	Vulnerable
Verreaux's Tree Frog	*Litoria verreauxii*			
Leptodactylidae				
Common Froglet	*Crinia signifera*			
Southern Bullfrog	*Limnodynastes dumerilli*			
TORTOISES				
CHELONIA				
Chelidae				
Common Long-necked Tortoise	*Chelodina longicollis*		Introduced to Phillip Island	
SKINKS				
SQUAMATA				
Scincidae				
Eastern Three-lined Skink	*Bassiana duperreyi*			
White's Skink	*Egernia whitii*			
Garden Skink	*Lampropholis guichenoti*			
Metallic Skink	*Niveoscincus metallicus*			
Southern Grass Skink	*Pseudemoia entrecasteauxii*			
Weasel Skink	*Saproscincus mustelina*			
Blotched Blue-tongued Lizard	*Tiliqua nigrolutea*			
SNAKES				
SQUAMATA				
Elapidae				
Lowland Copperhead	*Austrelaps superbus*			
FRESHWATER FISH				
TELEOSTOMI				
Anguilliformes				
Short-finned Eel	*Anguilla australis*			
TELEOSTOMI				
Galaxiiformes				
Common Galaxias	*Galaxias maculatus*			
Spotted Galaxias	*Galaxias truttaceous*		Rare	

Data from the Atlas of Victorian Wildlife (24 March 2004), supplemented with data from Nature Park.
Conservation Status in Victoria (DSE 2003a). Categories:

Victorian Categories - Vertebrate Fauna

- **Critically Endangered:** A taxon is Critically Endangered when it is facing an extremely high risk of extinction in the wild in the immediate future.
- **Endangered:** A taxon is Endangered when it is not Critically Endangered but is facing a high risk of extinction in the wild in the immediate future.
- **Vulnerable:** A taxon is Vulnerable when it is not Critically Endangered or Endangered but is facing a high risk of extinction in the wild in the medium-term future.
- **Low risk- near threatened:** A taxon is Low Risk- near threatened when it has been evaluated, does not satisfy the criteria for any of the threatened categories, but which is close to qualifying for Vulnerable. In practice, these species are most likely to move into a threatened category if the population decline continues or catastrophes befall the species.
- **Data Deficient:** A taxon is Data Deficient when there is inadequate information to make a direct or indirect assessment of its risk of extinction based on its distribution or population status. Listing of taxa in this category indicates that more information is required and acknowledges the possibility that future investigation will show that a threatened classification is appropriate.

Flora and Fauna Guarantee Act 1988

- **Listed:** Listed under the Act
- **Nominated:** Nominated for listing under the Act
- **Recommended:** Recommended for listing under the Act
- **Rejected:** Rejected for listing under the Act

References

'Museum Victoria, Melbourne, Australia (2002). Bioinformatics Victorian Faunal Web Site. Published on the Internet; http://www.museum.vic.gov.au/bioinformatics/ [accessed 23 February 2006)].

8.2 LP workshop agenda Thursday 30 Nov 2023

We are currently conducting a disease risk analysis (DRA) on the Phillip Island (Millowl) little penguin population and seek your expertise to collaborate in this process. A DRA is a structured, evidence-based process that can assist in decision making in the face of uncertainty and determine the potential impact of infectious and non-infectious diseases on ecosystems, wildlife, domestic animals and people. The results from this disease risk analysis can then be used to help decision makers to consider an evidence-based range of options for the prevention and mitigation of disease in the Philip Island Little penguin population.

We are planning to conduct a full day workshop on Thursday 30th November at the Werribee University of Melbourne campus and would be very keen to hear if one, or several, of you, are available on this date, or could nominate a suitable Zoos Vic veterinarian to participate.

A summary agenda for the day includes:

8.30 or 9am (TBC) - welcome and introductions
9:10 - participants working agreement & introduction to the DRA process
9:40 - project summary: ensure problem description, questions, goal, scope & focus, assumptions, limitations, hazard identification & prioritisation, risk assessment, risk management aligns with principal stakeholders views
10:00 - define acceptable levels of risk
10:05 - present the hazard list to stakeholders, provide information on the identified hazards infectious & non-infectious and receive feedback on their perceived & actual significance (emailed 1 wk prior to enable review prior to workshop) discuss as a group any other hazards that have not been identified in process and where further information in regards to these hazards may be found

10:45 - 15 min break

11:00 - Introduction to hazard prioritisation
11:10 - Decision making in the face of uncertainty
11:20 - Group work (approx. 6 individuals / group) to rank hazards based on likelihood and consequence to population of Phillip Island (Millowl) little penguin population, other wildlife, livestock and humans. Ideally four groups, two ranking infectious hazards and two ranking non-infectious hazards
12:20 - Each group presents summary of ranked infectious and non-infectious hazards to whole group (10 mins each)

13:00 - 45 min break for lunch

13:45 - Identification of knowledge gaps and prioritisation of how to further investigate and assess significance
14:05 - Next steps -prioritisation of hazards as a whole group and defining which hazards require detailed risk assessment
15:00 - Risk management general options

15:30 - 15 min break

15:45 - Risk communication strategies
16:00 - Further monitoring, surveillance needs
16:45 - Summary and wrap up
17:00 – end

8.3 Minutes and notes from workshop held Thursday 30 Nov 2023

Little penguin Disease Risk Analysis Workshop

Werribee Campus, The University of Melbourne, November 30th 2023

8.3.1 Participants

In person

1	**Rebecca Vaughan-Higgins** (RVH)
	Senior Lecturer Wildlife Conservation Medicine at Murdoch University, clinical veterinarian at Perth Zoo, involved in DRAs for translocation and conservation while working for the Species Recovery Programme at the Zoological Society of London
2	**Liz Dobson** (LD)
	Pathologist, The University of Melbourne
3	**Franciscus Scheelings** (FS)
	Postdoctoral Research Fellow, The University of Melbourne; did the DRA 8 months ago
4	**Ian Beveridge** (IB)
	Emeritus Professor of Veterinary Parasitology at The University of Melbourne
5	**Michael Lynch** (ML)
	Head of Veterinary Services at Zoos Victoria, worked with FS on the DRA 8 months ago
6	**Lee Skerratt** (LS)
	Head of the One Health Research Group at The University of Melbourne
7	**Jasmin Hufschmid** (JHu)
	Senior Lecturer in Wildlife Health at The University of Melbourne
8	**Jenny Hibble** (JHi)
	Veterinarian on Phillip Island (Millowl), works with sick/ injured penguins
9	**Rachel Gibney** (RG)
	Vet at Agriculture VIC working in emergency animal disease preparedness, AIV
10	**Dave Ramsey** (DR)
	Quantitative ecologist at the Arthur Rylah Institute
11	**Maria Schreider** (MS)
	Research manager at Phillip Island (Millowl) National Park (PINP)
12	**Leanne Renwick** (LR)
	Research officer at PINP, involved in the development of the emergency response planning, would implement plans
13	**Anna Langguth**
	Transcriptionist, Ph.D. Candidate One Health Research Group, The University of Melbourne

Online

1 **Mark Hawes** (MH)

Veterinary Pathologist for WHA, WHA VIC state coordinator

2 **Michelle Wille** (MW)

Senior Research Fellow at The University of Melbourne, specialises on avian viruses, works on prep for HPAI

3 **Erin Davis** (ED)

Veterinary project officer on the WHA international team, collaborates with the ICUN

4 **Brett Gartrell** (BG)

Professor in Wildlife Health at Massey University

5 **David Roberts** (DRo)

Clinical veterinarian at the Southern African Foundation for the Conservation of Coastal Birds, works mainly with African penguins

6 **Laura Roberts** (LRo)

Government veterinarian, completed a PhD on Avian influenza in African penguins and coastal seabirds

7 **Arlene Rutherford** (AR)

Veterinarian with WHA, Senior Project Advisor in the One Health International Group, economist

8 **Sarah Michael** (SM)

Wildlife veterinarian with the state government in Tasmania, and WHA state coordinator for TAS

9 **Andre Chiaradia** (AC)

Marine biologist at PINP, focusing on environmental impacts

10 **Mel Wells** (MWe)

Ph.D. candidate at The University of Tasmania, studying the health and ecology of penguins in TAS, also looking into blood parasites

11 **Ralph Vanstreels** (RV)

Wildlife veterinarian, works on blood parasites, has a background in penguin ecology, currently involved with HPAI outbreaks in Brazil

8.3.2 Background

Briefly explained by **RVH**

- Quick overview of background on little penguins
- DRA will be following IUCN guidelines (Jakob-Hoff *et al.* 2014)
- DRA will mainly led by The University of Melbourne

- Workshop goals and aims (refer to DRA document)

- Agenda:
    - Morning session: Process of the DRA, Problem description, Hazard identification
    - Afternoon: Group discussions, risk assessment, risk management, risk communication

- Participants' working agreement
    - Focus on agreed objectives
    - Other business and agendas are put on hold
    - Respectful communication
    - Everything recorded
    - Everyone participates
    - All ideas, comments, opinions are openly shared
    - All ideas valis
    - Actively listen to each other
    - […]

      → **All participants agree**

- Group work, expectations and roles
    - Split based on area of expertise
    - Facilitator, padlet and document manager, note-taker, time-keeper, presenter

8.3.3 Padlet

https://The University of Melbourne.padlet.org/rebeccavaughanhiggins/little-penguin-dra-workshop-thursday-30-november-2023-y3l8yz8sn3sv0ej

8.3.4 Definitions

- IUCN Guidelines (Jakob-Hoff *et al.* 2014):
  - **Disease:**
    Any impairment of the normal structural or physiological state of a living organism resulting from its physiological response to a hazard
  - **Hazard:**
    A biological, chemical or physical agent, or a condition of an animal or animal product with the potential to cause an adverse health effect
    > includes parasites and non-infectious agents (e.g. toxins, trauma)
  - **Parasites:**
    Agents that live on or within a host and that survive at the expense of the host regardless of whether disease follows or not
    > includes both microparasites (viruses, bacteria, fungi) and macroparasites (helminths, parasitic arthropods)
  - **Pathogens:**
    Any disease-causing parasite

In this DRA:

- **Parasites**
  Macroparasites and microparasites (fungi, bacteria, viruses)

- **Risk**
  Likelihood x Consequences

8.3.5 DRA Risk Matrix

	Consequence of entry, establishment and spread in the specified population				
		Negligible	**Minor**	**Moderate**	**Major**
Likelihood of entry, establishment and spread in the specified population	**Very low**	Very low	Very low	Low	Low
	Low	Very low	Low	Low-Moderate	Moderate
	Moderate	Low	Low-Moderate	Moderate	High
	High	Low	Moderate	High	High

How to include uncertainty:
If it's a high level: Either risk cannot be defined, or we may choose a higher risk level automatically

8.3.6 DRA Terminology for "Likelihood"

Terminology and classification system for likelihood of hazard entry, establishment and spread	
Very low	Very unlikely to occur – hazard has not been recorded in the geographic range of the species at Phillip Island (Millowl)
Low	Hazard has been recorded in the geographic range of LPs at Phillip Island (Millowl), but it is unlikely this hazard would occur or become established

Moderate	Hazard occurs in other, similar ecosystems or geographically / climatically similar sites and is able to persist at geographically similar sites, but has not been recorded at Phillip Island (Millowl)
High	Hazard is known or expected to occur / persist in the Phillip Island (Millowl) population of LPs.

- **RG:** May not make sense for non-infectious hazards

8.3.7 DRA Terminology for "Consequences"

Terminology and classification system for consequences for LPs and extant wildlife	
Negligible	No detectable effects on the individual, no detectable population consequences, no socio-economic consequences.
Low	Individual morbidity or mortality, but no detectable population consequences, minimal socio-economic consequences in the short or long term. Low impact on welfare
Moderate	Temporary detectable population decline without risk of extinction from this disease, low socio-economic consequences. No long term population impact on the expected population growth / population levels of birds
High	High risk of local extinction, due to significant population decline at unsustainable levels, moderate to major socio-economic consequences. Changes status listing of bird species, impacts multiple bird species, flow on impacts to ecosystem processes. Has a high impact on welfare – many animals affected and prolonged suffering.
Proposed classification for consequences for humans and domestic animals	
Low	No effects
Moderate	Self-limiting morbidity, not requiring treatment
High	Individual morbidity, requiring treatment or not self-limiting.

8.3.8 DRA Terminology for "Uncertainty"

Proposed classification of level of uncertainty adapted from Wieland, Dhollander et al. (2011)	
Low	Solid and complete data available; strong evidence provided in multiple references
Medium	Some but no complete data available with some information gaps; evidence provided in small number of references.
High	Scarce or no data available with significant information gaps; evidence is not provided in references but rather in unpublished reports, based on observations, or personal communication

8.3.9 DRA – The Australian Situation

- While a pre-requisite for translocations and managed by legislation in other countries (e.g. UK), DRAs are currently not commonly undertaken in Australia
- New paper published in Canberra mid-November 2023 which mentioned the risk of disease
  à important first step

- Communicative Approach
  - Multi-stakeholder approach is 'best practice'
    - Slower, may be difficult logistically
    - But higher chance of getting stakeholder support
  - Ideally with legislator involvement

- Approach
  - Qualitative DRA – likelihood of outcome high, medium, low
    à most commonly used in Australian wildlife DRAs

  - Quantitative DRA – numerical expression of outcome
    à but hard to do for wildlife populations

- Gold standard:
  IUCN Manual of Procedures of Wildlife Disease Risk Analysis

8.3.10 Sources of Information

- **Lack of information specifically for little penguins (LPs)**
  - Extrapolate from other penguin species
  - Acknowledge assumptions (e.g. if a disease can affect another penguin species, we assume that LPs are also susceptible, but will acknowledge uncertainty)
  - Stakeholder and expert involvement; ideally well-prepared, funded workshop with an appropriate range of experts, stakeholders, and decision-makers

- **Scope of information collected**
  - Spheniscidae wild & captive 1974 – 2024
  - Necropsy reports 1990 – Nov 2023 collated by PINP
  - Experts involved with Spheniscidae (information on diseases not reported by other means)

  - Other info on scope of information collected
    - Species ecology: What other species are LPs interacting with (disease transmission)?
    - Disease screening: Currently not a lot of data on LPs at PINP

- **Wildlife Health Australia**
  - Collates and manages national disease surveillance – free-ranging wildlife
  - National wildlife surveillance data collected by zoos, selected private veterinary practices and universities

  Australian registry of Wildlife Health
  - Diagnostic pathology service
  - Archived material and records on Australian wildlife disease

- **Other sources**
  - Online database searching
  - Management plans
  - Published and unpublished literature
  - Expert and stakeholder opinion

8.3.11 Problem description

Outline and framework of the LP DRA

- **RVH: Any general feedback?**

- **Lee Skerratt (LS):**
  Rather than talking about 'disease', we should think more about the overall 'health' of the population, as disease is natural. We are trying to create 'resilient', 'healthy' species, mitigating as best as we can against the effects of the Anthropocene

- **Dave Ramsey (DR):**
  Major threat would be collapse of the food supply – overfishing

- **Arlene Rutherford (AR):**
  Should actually call it the "Wildlife 'health' risk analysis"
  Knowledge other than veterinary knowledge is also very valuable

- **Andre Chiaradia (AC):**
  Questions whether diseases of e.g. African penguins are applicable at all
  - **RVH:** first look was very broad, some hazards can potentially be excluded from the get-go
  - **Michael Lynch (ML):** We have to be careful about making assumptions, because infection may not always lead to disease that affects populations

- **LS:**
  Row 'Assumptions' in Table 1 (DRA Draft, p. 13): Can we really say "There are no other novel, unknown, or yet to be determined disease risks"?
  - **RVH:** Cannot assess "unknown unknowns" but should acknowledge them

Defining "acceptable risk"

RVH: <u>Who or what is at risk?</u>

- **Franciscus Scheelings (FS):** People, predators that rely on LPs
- **Rachel Gibney (RG):** Livestock

- **LS**
  Risk on the conservation status of the species ≠ Risk as perceived by the public/ politicians

- **Jenny Hibble (JH):**
  Phillip Island (Millowl) is dependent on PINP economically

- **Jasmin Hufschmid (JHu):**
  Acceptable risk may also be something that the public rather than the scientific community/ experts would classify as such

- **Dave Robers (DR):**
  Zoonotic diseases would likely confer a lower "acceptable risk"

- **RVH: <u>Definition of Species Groups?</u>**
  - Species Group 1: Little penguins at Phillip Island (Millowl)
  - Species Group 2: Wildlife resident at the site (including introduced or feral species)
    - **FS:** add wildlife that is not resident, penguins go out to sea
    - **Liz Dobson (LD):** consider migrating wildlife
  - Species Group 3: People and domestic animals (livestock and pets)

RHV: <u>Definitions of "low", "moderate", "high: risk?</u>

- **LS:**
  High= Anything that would change the conservation status of LPs

- **Michael Lynch (ML):**
  - High/ Moderate: Welfare impacts on a significant number of animals without affecting the conservation status
    à Group agrees that this would also be "high"

  - **DR:** Might have economic impact
  - **LD:** If the impact is on reproduction, then should probably be classified as "high"
  - **Maria Schreider (MS):** Welfare is a very important consideration in terms of the wellbeing of the animals and the perception of the public, visitors may be concerned for the birds
  - **RVH:** Public may also be concerned about the impacts of potential hazards on themselves
  - **MS:** Especially international visitors may panic seeing "unwell birds"

- **RVH: "Low" = individual, sporadic disease?**
  - Participants agree.

  - **Leanne Renwick (LR):** Occasional findings of dead penguins are currently already considered "normal" at PINP

  - **ML:**
    Something we accept as "normal" now, may not be "normal" in the future, so something classified as "low" would infer that we are confident that prevalence/ impact of the hazard is going to stay at current level and

won't have future population impacts

- **AC:**
  Localised disease may not change the IUCN status of the species
  DR:
  - But may change it under FFG – Flora and Fauna Guarantee Act VIC
  - Morbidity is also important, not just mortality; some chronic processes may affect morbidity now and then lead to low recruitment in following years

- **RVH: What is an "acceptable risk" for LPs? Individual disease and mortality?**
  - **Brett Gartrell (BG):**
    Any disease or pathogen that has trans-species impacts (e.g. HPAI) has to receive special consideration
    Chlamydia – have been finding avian strains in wading birds in New Zealand; have to look beyond the penguins themselves

  - **LD/ LS:**
    Defining "low risk" as "individual deaths" might be tricky if you don't know what the animal died from
    > **LR** and **DR** agree

  - **RG:** "Baseline mortality rate" at PINP?
    LR: Not usually quantified among the adult population; usually more focused on breeding success

  - **RHV/ JHu:**
    We're likely going to have to look at the different species groups differently

- **RVH: What is an "acceptable risk" for people?**
  - **LS:**
    If someone contracts a disease from the PINP penguins, that would be a disaster

  - **ML:**
    Will also come down to risk of likelihood

  - **JHu:**
    If your "acceptable risk" in humans is higher, then that would mean that the threshold for interventions is higher as well, which we would probably not want

  - **RVH:**
    Humans – acceptable risk will probably "negligible".
    > Participants agree

- **RVH: What is an "acceptable risk" for companion animals?**
  - **RG:**
    For livestock, it will probably depend on something quantifiable and the economic impact

  - **Mark Hawes (MH):**
    Not much livestock on Phillip Island (Millowl), so may depend on the geographic location of the risk

  - **JHi:**
    When Bandicoots were released, some farmers were quite paranoid about them being released near their farms
    ML: comes down to communication

- **JHi:**
  For pets, people would be more proactive, would probably receive higher media coverage than livestock disease

- **DR:**
  Consider potential EAD response; e.g. destruction of the flock would have economic impacts

- **JHi/ FS:**
  There is a chicken farm on Phillip Island (Millowl), and there is also backyard poultry
  AC: But has been discontinued

- **BG:**
  Significance of some diseases might be of national concern (e.g. HPAI), shouldn't think of it as just a local issue necessarily

- **Michelle Wille (MW):**
  Good idea to look at AgVic's actions in 2020

Hazard Identification and Categorisation

- Complete list put together; hazards will further be assessed based on acceptable risk
- Categorisation based on consequences

- 63 hazards identified

- **RVH: Have we missed any hazards?**
  - **BG:**
    Avian malaria probably increasing in frequency (in yellow eyed penguins and LP in New Zealand)
    Avian chlamydia probably increasing in frequency as well

  - **FS:**
    Contamination with PFAS, no references known

  - **ML:**
    Entanglement as a separate hazard

  - **LR:**
    Predation by ravens: Take eggs and chicks, scavenge
    In isolated areas, rates of chick fledging can be down to 0 %
  - **MS:**
    Long-nosed fur-seal predation

  - **Mel Wells (MWe):**
    Emerging contaminants, other things that have just been found and described
    Pesticide contamination may not cover all contaminants
    > Studies in penguins and seals have been done

  - **AC:**
    Contaminants such as Mercury, works on another report right now, penguin-based monitoring

- **RVH: Any other stakeholders we may have missed?**
  - **MS:**
    People that specialise on ectoparasites, not specifically penguins

8.3.12 Hazard list discussions by group

Hazard list – Online Group

Group left most things as they were, notes on changes and justifications below:

Disease	Parasite	Hazard population	Likelihood rating	Consequence rating	Uncertainty rating	Final rating
VIRAL						
High pathogenicity avian influenza	High pathogenicity avian influenza virus	1	-	-	-	-
		2	M	H	-	-
		3	H	H	-	-
Risk of non-resident birds should be increased						
BACTERIAL						
Avian chlamydia	*Chlamydia psittaci*	1	*Couldn't take notes; see uploaded documents*			
		2				
		3				
Bumped up the rating because of the perceived risk to human and companion animal health (specifically pets)						
-HAEMOPARASITES						
Haemoparasites – Avian malaria	*Plasmodium* *Haemoproteus* *Leukocytozoon* *Babesia*	1	*Couldn't take notes; see uploaded documents* *Rated "likelihood" as "low"*			
Plasmodium should be acknowledged as having higher consequences than laid out in the draft						

CONGENITAL/DEVELOPMENTAL					
Beak malformation, carpal rotation, ventricular septal defects, splay leg	1	L	M	-	-
Could be a warning sign for other issues in the colony					
INTOXICATION					
Algal toxins	1	M	M	-	-
Probably higher likelihood in the future due to climate change					
Heavy metal intoxication	1	M	L	-	-
Probably higher likelihood in the future due to climate change					

Comments:

- **FS:**
  Plasmodium is presumable relatively prevalent; why the low likelihood rating?
  DR: Agrees, likelihood probably increasing in terms of changes in vector distribution
- **Ralph Vanstreels (RV):**
  Probably small, localised clusters rather than country-wide problem.
  Might be good to separate all hemoparasites and assess separately.

- **DR:** Plasmodium is a lot worse than others, e.g. haemoproteus

- **RV:** Plasmodium is known to potentially be lethal, Babesia and Haemoproteus may not be. We also know that plasmodium is in Australia.

  è General consensus that Plasmodium should be assessed separately and may be of high importance (high likelihood, potentially high consequences)

Hazard list – In-person Group A

Participants: Liz Dobson, Fran Scheelings, Rachel, Leanne Renwick, Lee Skerratt, (Anna Langguth)
Notes from the group discussion:

	Hazard population	Likelihood rating	Consequence rating	Uncertainty rating	Final rating
CONGENITAL/DEVELOPMENTAL					
Beak malformation, carpal rotation, ventricular septal defects, splay leg	1	L	L	L	L
LR: See congenital abnormalities rarely **LD:** Low likelihood unless caused by toxin, as genetic diversity decreases incidence is increasing **LS:** May want to check genetics if there was an increasing rate; in that case might bump up risk to "medium" Risk management strategy: Any increase needs to be monitored **LR:** A certain proportion of burrows are monitored (~ 800), could pick up increased mortality, necropsies on chicks are rarely done, but congenital malformations that are easily visible are noted; currently not allowed to keep them indefinitely without a specific purpose See beak malformation a bit more often à Uncertainty rating: Low, a lot of monitoring being done					
DEGENERATIVE					
Cataracts	1	L	L	L	L
LD: Likely mostly affecting aged animals **FS:** Occasionally seen in captive penguins, but wild penguins may not get to that stage **LR:** Cataracts would affect LPs vision and ability to catch prey; sometimes they do see penguins with cataracts in hospital					
Degenerative joint disease	1	L	L	M	L
LD: Aged individuals, would not have a population impact					
INTOXICATION					
Algal toxins	1	L	L	M	L
LR: Very rarely seen **LS - Reference:** https://www.mdpi.com/1660-3397/15/2/33#					
Heavy metal intoxication	1	H	M	M	M
FS: Would be surprised if LPs weren't exposed **LR:** Had a student investigating heavy metals in LPs (Annette Finger) – more of a problem at St Kilda than Phillip Island (Millowl) but increasing annual trend and negative association with flipper length at St Kilda; differences in seasons and location https://www.sciencedirect.com/science/article/abs/pii/S0269749116317808?via%3Dihub **Not enough data**					
Industrial chemical intoxication – oiling	1	H	H	L	H
LR: Risk huge, some of the highest shipping traffic in VIC **FS:** Nearest oil rig is bass strait, probably too far away **Consequence?** **LR:** Penguins feed in 3 different distinct areas, so a spill may not affect all birds equally, but depends; worst case 3.000 penguins affected in as					

little as one hour; May not die from it, but would be of high public concern (moderate-major socio-economic consequences)					
Pesticide contamination	1	L	L	M	M
LD: Probably constant low-grade contamination, but uncertain what the level of contamination is exactly					
PFAS	1	H	L	L	M
FS: Quite certainly exposed, but probably low exposure					
ENVIRONMENTAL					
Botulism	1	L	L	L	L
Probably not in marine environment					
Environmental stressors including climate change -sea level rise -decreased rainfall & humidity -increased fire risk -extreme climate events -warmer oceans -ocean acidification -vector-borne disease -SST & marine heatwaves	1	H	H	L	H
LR: Starvation events increasing, effect of climate change **LS:** "Bad" years are now turning into "really bad" years and are increasing in frequency					
Habitat loss - terrestrial -trampling by humans / livestock, introduced species, invasive weeds, housing developments	1	M	M	L	M
LR: For the most part these points are well managed, still a bit of a weed problem; some new colonies are establishing themselves around Phillip Island (Millowl) and they are difficult to manage So some areas are at risk **LS:** Population is quite limited geographically so vulnerable **LD:** Should also look at ocean – added "Habitat loss – aquatic"					

Habitat loss – aquatic	1	M	M	L	M
LR: A lot of areas where they feed are unprotected Noise pollution needs to be considered as well **LS:** Oceans are getting warmer, more acidic, there's more microplastic					
Predation	1	H	L	L	M
LR: Island is fox-free, but big feral cat problem, incidents with domestic dogs (especially with the new colonies forming), ravens At the moment, ravens seem to be targeting only two particular areas 2023 is the 4th year where PINP is seeing 0 % breeding success. No noticeable impact on population yet but will probably see it soon. **FS:** Why are there so many ravens preying on nests? **LR:** Uncertain, probably learned behaviour. <u>Mitigation actions:</u> Destroy raven nests? But currently no actions undertaken.					
Reduction in food supplies	1	H	H	L	H
Group agrees on the importance of this.					
Starvation	1	H	H	L	H
Group agrees on the importance of this.					

Storm damage	1	M	L	L	L
LR: Some cliffing of dunes which prevents penguins accessing their burrows Floods may cause individuals to be unable to access burrows, not common but seen every few years and only seen in isolated areas					
Thermal stress	1	H	M	L	M
LR: Probably no population-level impacts at the moment On average one heat-stress event every 4-5 years, not entirely sure how many animals are being lost Worse than the storms					
Wildfire	1	H	H	L	H
Group agrees on this. Could wipe out the whole population/ destroy the whole island					
OTHER					
Cardiac (heart) disease	1	L	L	L	L
LD: Spontaneous and random, would only affect individuals					
Entanglement	1	L	L	M	L
LR: Not common **FS:** But you may not find animals entangled at sea; uncertainty higher **LD:** But not a lot of industrial fishing in the area					

Gastrointestinal foreign bodies	1	H	L	H	M
LD/ FS: Problem in all seabirds, but uncertain what the numbers are					
Hepatic (liver) disease	1	L	L	L	L
LD: Random if not infectious					
Neoplasia	1	L	L	L	L
See above					
Pododermatitis (bumblefoot)	1	L	L	L	L
Of low importance in wild birds					
Tourism	1	H	L	M	M
LR: Plans for a lot of future development, impact of tourism depends on people management; No management of new colonies that are popping up around the park **LD:** Should be on the radar, so shouldn't be too low					
Trauma	1	H	L	L	L
Cars, boats **LR:** Has been seen, but only low numbers					

Comments:

- MS:
  - Cape Barren geese are also presumed to be destroying habitat
  - Ravens are sacred birds for the Bunurong peoples – very challenging to figure out management strategies

Hazard list – In-person Group B

Participants: Jasmin Hufschmid, Michael Lynch, Maria Schreider, Ian Beveridge, Jenny Hibill, Dave Ramsey

Only points that were filled out/ commented on are listed

Disease	Parasite	Hazard population	Likelihood rating	Consequence rating	Uncertainty rating	Final rating
ENDOPARASITES						
-PROTOZOANS						
Coccidiosis	*Eimeria*, *Isospora*, & *Cryptosporidium*.	1	-	-	-	-
Flagellates: Giardia, Trichomoniasis	Giardia *Trichomonas gallinae*	1	L	L	M	H
		3	L	M	-	-
Sarcocystis and Toxoplasmosis	*Sarcocystis spp.* *Toxoplasma gondii*	1	-	-	-	-
Toxoplasma could have a reasonably big impact on the population						
-NEMATODES						
Capillariasis	*Capillaria spp.*	1	-	-	-	-
Contracaecum - helminths	*C. eudyptulae*, *C. spiculigerum*	1	H	M	L	H
		3	Negligible	-	-	-
Very common in little penguins, specifically Contracaecum. Could contribute to mortalities						
-TREMATODES						
Trematodiasis	*Cardiocephaloides physalis*	1	-	-	-	-
Trematodiasis	*Galactosomum angelae*	1	-	-	-	-
Trematodiasis	*Mawsonotrema eudyptulae*	1	-	-	-	-
Renal fluke	*Renicola*	1	-	-	-	-
Some of these may severely impact specifically penguin chicks						
ECTOPARASITES						
Lice	*Austrogonoides waterstoni*	1	H	L	L	-
Mites	*Ingrassia eudyptula,* & *Veigaia* sp.) and others	1	H	L	M-H	-
Ticks	*Ixodes eudyptidis, Ixodes uriae*	1	H	M	M-H	-
		2	H	M	-	
Lots of knowledge gaps overall						

Comments:

- A bit of confusion around definitions
  Low likelihood = has been recorded at the site before, not all groups initially used this definition

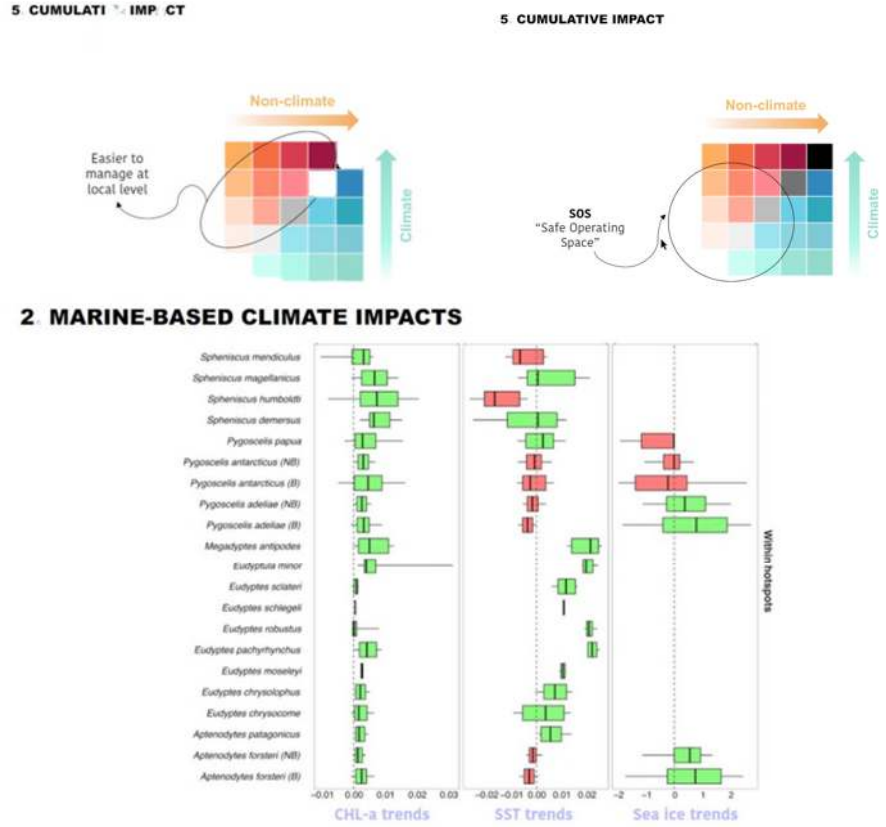
- **RV:**
  Ixodes ticks have a history of significant mortalities in penguins
  > references available

8.3.13 Prioritising hazards

Look at all risk levels, go through all hazards and assign what is most important

Non-Infectious

CONGENITAL/DEVELOPMENTAL	Hazard population	Final rating
Beak malformation, carpal rotation, ventricular septal defects, splay leg	1	L
Final risk rating: Online Group: Might be an indication of some other issue; **LRo:** would probably retract higher impact rating as only individuals affected In -person A: - In- person B: Low, **LS:** Surveillance should include an awareness of this		
DEGENERATIVE		
Cataracts	1	L
Final risk rating: Online Group: Low In -person A: Low In- person B: -		
Degenerative joint disease	1	L
Final risk rating: Online Group: Low In -person A: Low In- person B: -		
INTOXICATION		
Algal toxins	1	M
Final risk rating: Online Group: M In -person A: M In- person B: -		
Heavy metal intoxication	1	M
Final risk rating: Online Group: M In -person A: M In- person B: -		
Industrial chemical intoxication - oiling	1	M-H
Final risk rating: Online Group: Low likelihood, moderate consequence In -person A: H In- person B: -		
Pesticide contamination	1	L
Final risk rating: Online Group: L, Site-specific; **AC:** more likely for St. Kilda penguins, less likely for penguins at PINP In -person A: M, farming (industrial and hobby), moderate uncertainty hence rating bumped up, needs further investigation In- person B: - **Probably knowledge gap.**		

PFAS	1	M
Final risk rating: Online Group: - In -person A: Moderate, only group to assess In- person B: - Group agrees.		
ENVIRONMENTAL		
Botulism	1	L
Final risk rating: Online Group: - In -person A: Low In- person B: - Group agrees.		
Environmental stressors including climate change: sea level rise/ decreased rainfall & humidity/ increased fire risk/ extreme climate events/ warmer oceans/ ocean acidification/ vector-borne disease/ SST & marine heatwaves	1	H
Final risk rating: Online Group: - In -person A: High In- person B: - **AC:** Is steadily increasing **RV:** Useful to split these, so we can look at mitigation factors separately | **JHu:** Might be better to look at the effect of these stressors on the penguins and identify those (e.g. starvation) as hazards **RVH:** Look at starvation more so than "climate change" as a whole Comment **AC:**  		
Habitat loss - terrestrial -trampling by humans / livestock, introduced species, invasive weeds, housing developments	1	M
Final risk rating: Online Group: - In -person A: Moderate In- person B: - Group agrees.		
Habitat loss - aquatic	1	M
Final risk rating: Online Group: -		

In -person A: Moderate
In- person B: -

Group agrees.

Predation	1	H

Final risk rating:
Online Group: -
In -person A: Moderate
In- person B: Not discussed but **DR** adds that it would be better to rate as high as needs to be managed

MS:
There may also be possums eating eggs, and echidnas squashing/ eating eggs

Group agrees to list as high.

Reduction in food supplies	1	H

Final risk rating:
Online Group: -
In -person A: High
In- person B: -

Group agrees.

Starvation	1	H

Final risk rating:
Online Group: -
In -person A: High
In- person B: -

Group agrees.

Storm damage	1	? M

Final risk rating:
Online Group: -
In -person A: Low
In- person B: -

DRo: extreme weather events is something they are very concerned by
AC: coastal erosion is a huge threat, dune-cliffing happens every high tide
FS: Cliffing may not be related to storm damage, but rather to high tides
ML: Suggests “coastal erosion”/ “Alteration of habitat by weather”

Coastal erosion		?

Final risk rating: Not properly discussed

Thermal stress	1	M

Final risk rating:
Online Group: -
In -person A: Medium
In- person B: -

Group agrees.

Wildfire	1	H

Final risk rating:
Online Group: -
In -person A: High
In- person B: -

Group agrees.

OTHER		
Cardiac (heart) disease	1	L
Final risk rating: Online Group: - In -person A: Low In- person B: - Group agrees.		
Entanglement	1	L
Final risk rating: Online Group: - In -person A: Low but moderate uncertainty due to the fact that entangled animals in the ocean are likely to be missed In- person B: - **AC:** Probably site-specific depending on fishing nets used in the area **LR:** Not many cases seen on Phillip Island (Millowl) **JHi:** Some cases of entanglement with marram grass seen – invasive species of beach grass		
Gastrointestinal foreign bodies	1	L
Final risk rating: Online Group: - In -person A: Moderate but high uncertainty In- person B: - **JHu:** Did necropsies on 50 animals, fishing line found in one **AC:** Since penguins chase their food, it is unlikely that they take up plastic when they feed **RV:** Has seen that a reasonably high proportion of juveniles (10-40 % but no studies done) might ingest plastic **AC:** Might confuse plastic with jellyfish		
Hepatic (liver) disease	1	L
Final risk rating: Online Group: - In -person A: Low In- person B: - Group agrees.		
Neoplasia	1	L
Final risk rating: Online Group: - In -person A: Low In- person B: - Group agrees.		
Pododermatitis (bumblefoot)	1	L
Final risk rating: Online Group: - In -person A: Low In- person B: - Group agrees.		
Tourism	1	M
Final risk rating: Online Group: - In -person A: Moderate In- person B: - **MS:** Some guides may be taking people away from the boardwalks to look at the penguins **AC:** Recent paper published on the effect of Covid lockdown on PINP penguins, absence of visitors = zero change on penguins https://www.sciencedirect.com/science/article/abs/pii/S000632072300424X **LR:** Moderate risk that is managed in favour of the penguin population; but to look at the effects of tourism on the LPs at PINP overall, not just people attending the penguin parade (**LS**, **DR** and **FS** agree) **MS:** Penguins don't react to people on the boardwalk very much, but as soon as they step off of it, penguins react differently		
Trauma	1	L but monitoring
Final risk rating: Online Group: - In -person A: Low In- person B: -		

JHu: Might want to keep it in mind (boat strikes, more people buying boats), so it's not entirely forgotten about, needs to be monitored.

Group agrees.

Infectious

Disease	Parasite	Hazard population	Final rating
VIRAL			
Arboviruses	BFV, Kunjin RRV, MVEV	1	L
		3	L
	Saumarez reef virus	1	M
Final risk rating BFV, Kunjin RRV, MVEV – Hazard population 1 Online Group: Low In -person A: - In- person B: - **Final risk rating BFV, Kunjin RRV, MVEV – Hazard population 3** Online Group: Low In -person A: - In- person B: - **Final risk rating Saumarez reef virus:** Online Group: Medium, proven pathogenicity, high likelihood it would get in In -person A: - In- person B: - **FS:** Consider WNV, as other exotic viruses are listed by name; huge human problem **LS:** Only in the northern hemisphere so far **RVH:** Little penguins likely susceptible but not within the range à potentially add WNV?			
Avian encephalomyelitis virus 'Pingu virus"	Picornaviridae	1	L
Final risk rating: Online Group: - In -person A: - In- person B: - Group agrees to rate as "low".			
Avian infectious bronchitis virus	Coronaviridae	1	L
Final risk rating: Online Group: Low In -person A: - In- person B: - Group agrees.			
High pathogenicity avian influenza	High pathogenicity avian influenza virus	1	H
		2	H
		3	H
Final risk rating: Online Group: High for all three In -person A: - In- person B: - Group agrees. **RV:** Might be worth to score LPAIV separately as well			
Avian poxvirus	Avipoxvirus	1	L-M
Final risk rating: Online Group: Low In -person A: - In- person B: - **RV:** Would argue for raising it to moderate, has caused mass-mortalities (hundreds to thousands) in rockhopper penguins and gentoos, was never properly published; diphtheroid form seen in these species, potentially respiratory stress; reports available **FS:** There has only been one published case report, but poxviruses in birds are fairly common so risk was evaluated to be "low" in the previous DRA (Scheelings 2020). Infection is self-limiting, doesn't usually impact the birds unless lesions cause mechanical obstructions that impact feeding **RVH:** Uncertainty high, bump up risk rating			
Avian reovirus	Reoviridae	1	L

Final risk rating: Online Group: Low In -person A: - In- person B: - Group agrees.			
Gyrovirus	Gyrovirus, Anelloviridae	1	L-M
Final risk rating: Online Group: Low In -person A: - In- person B: - **LS:** Quite a lot of uncertainty, not sure if it has killed chicks, mild-moderate			
Infectious bursal disease virus	Birnavirus	1	L
Final risk rating: Online Group: Low In -person A: - In- person B: - Group agrees.			
Infectious laryngotracheitis virus Herpes disease	Herpesviridae Avian herpesvirus 1	1	L
Final risk rating: Online Group: Low In -person A: - In- person B: - Group agrees.			
Newcastle disease and Avian paramyxoviruses	Avian paramyxovirus (APMV)	1	?
Final risk rating: Online Group: - In -person A: - In- person B: - **RV:** Depends on strain, if it's the neurotrophic strain it could be quite nasty, probably better to split this. **à Needs to be split further**			
Puffinosis	Suspected viral but unknown	1	L
Final risk rating: Online Group: Low, but high uncertainty In -person A: - In- person B: - Group agrees.			
BACTERIAL			
Avian chlamydia	*Chlamydia psittaci*	1	L
		2	M
		3	M
Final risk rating: *No specific notes from either group.* **MH:** Consequence most likely to be low for penguins, but medium for humans; a bit of uncertainty in the penguins **RV:** Some aggressive cases of chlamydia in penguins in the USA, however, there are very similar pathogens that are non-pathogenic so current serological results are not necessarily reliable, might not circulate in wild penguin populations **LRo**: Based on criteria of the DRA, consequences for humans would be high **à Uncertainty remaining**			
Avian cholera	*Pasteurella multocida*	1	M
Final risk rating: Online Group: M because low likelihood but high consequence In -person A: - In- person B: - Group agrees.			
Avian mycobacteriosis		1	L
		2	L
		3	L
Final risk rating – hazard group 1 Online Group: Low			

In -person A: -
In- person B: -

Final risk rating – hazard group 2
Online Group: Low
In -person A: -
In- person B: -

Final risk rating – hazard group 3
Online Group: Low
In -person A: -
In- person B: -

Group agrees.

E.coli infection	*E.coli*	3	L
		2	L
		3	L

Final risk rating – hazard group 1
Online Group: Low
In -person A: -
In- person B: -

Final risk rating – hazard group 2
Online Group: Low
In -person A: -
In- person B: -

Final risk rating – hazard group 3
Online Group: Low
In -person A: -
In- person B: -

Group agrees.

Erysipelas		1	L
		3	?

Final risk rating – hazard group 1
Online Group: Low likelihood, medium consequences
In -person A: -
In- person B: -

Final risk rating – hazard group 3
Online Group: -
In -person A: -
In- person B:
>> not discussed

RVH and **ML**: Quite sporadic in penguins
LS: Probably more of an issue in captive/ translocated populations

Mycoplasmosis	*Mycoplasma spp.*	1	L

Final risk rating:
Online Group: Low
In -person A: -
In- person B: -

DRo: Some cases diagnosed in cormorants that died in high numbers in captivity, so may not be applicable in wild populations
ML: Will be found in most animals if specifically looked for

Necrotic enteritis	*Clostridium* spp.	1	L

Final risk rating:
Online Group: Low
In -person A: -
In- person B: -

Penguin diptheria	*Corynebacterium* spp.	1	L

Final risk rating:
Online Group: Low, but high uncertainty
In -person A: -
In- person B: -

MH: Great deal of uncertainty here as unsure whether there is an underlying viral pathogenesis
RV: Only been reported in yellow-eyed penguins

Pseudomonas infection	*Pseudomonas* spp.	1	L

Final risk rating:
Online Group: Low

In -person A: - In- person B: - Group agrees.			
Salmonellosis	*Salmonella* spp.	1 2 3	L L L
Final risk rating – hazard group 1 Online Group: Low In -person A: - In- person B: - **Final risk rating – hazard group 2** Online Group: Low In -person A: - In- person B: - **Final risk rating – hazard group 3** Online Group: Low In -person A: - In- person B: - Group agrees.			
FUNGAL			
Aspergillosis	*Aspergillus sp*	1	L
Final risk rating: Online Group: Low to moderate In -person A: - In- person B: - **MH:** Since moderate means that there are "some" population effects, potentially better to change to L-M? **RVH:** Probably more of a concern in captive individuals, **ML** agrees **Changed to "low".**			
Candidiasis	*Candida albicans*	1	L
Final risk rating: Online Group: Low In -person A: - In- person B: - Group agrees.			
ENDOPARASITES			
-CESTODES			
Tetrabothrius infection	*Tetrabothrius sp. and Tetrabothrius lutzi*	1	L
Final risk rating: Online Group: - In -person A: - In- person B: Low **Ian Beverdidge (IB):** Penguins commonly infected with cestodes without any apparent consequences			
-HAEMOPARASITES			
Haemoparasites – Avian malaria	*Plasmodium*	1	M
à Split *Plasmodium* from other Haemoparasites **Final risk rating:** Online Group: - In -person A: High **(FS)** In- person B: - **FS:** Given that the population is about 30.000 penguins strong, the probability that some individuals are infected is high **ML:** Currently looking at blood smears (and will to PCR) on samples taken from the St Kilda colony **FS:** Blood smears have very low sensitivity **ML:** Babesiosis is common but less common to clinically affect the animal, *Plasmodium* exists and impacts the animal **MWe:** *Plasmodium* has been found in little penguins at Taronga, not been found in wild birds in TAZ so far **RV:** We know it's around, outbreaks could be driven by specific weather conditions (e.g. heavy rain)			
	Haemoproteus *Leukocytozoon* *Babesia*	1	?

MWe: *Babesia* found in wild birds in TAZ and may cause disease
FS: *Haemoproteus* probably there, probably does not cause disease, *Leukocytozoon* almost certainly not there

Final risk rating:
Online Group: Low, but high uncertainty
In -person A: -
In- person B: -

à needs to be re-assessed

-PROTOZOANS			
Coccidiosis	*Eimeria, Isospora*	1	L

Final risk rating:
Online Group: -
In -person A: -
In- person B: High likelihood but low consequence at the population level

RV: Separate Crypto from the other two as mortalities have been seen in African penguins (reference available)

Coccidiosis	*Cryptosporidium*	1	L-M

RV: Never been detected in little penguins, but can cause mortalities.

Group agrees to list risk as "low-moderate".

Flagellates	Giardia	1	L
-Giardia		3	M

Final risk rating – hazard group 1
Online Group: -
In -person A: -
In- person B: Low

Final risk rating – hazard group 3
Online Group: -
In -person A: -
In- person B: Moderate for humans

Group agrees.

-Trichomoniasis	*Trichomonas gallinae.*	1	L

Final risk rating:
Online Group: -
In -person A: -
In- person B: Low

Group agrees.

Sarcocystis and	*Sarcocystis spp.*	1	L
Toxoplasmosis	*Toxoplasma gondii*	1	M

Final risk rating:
Online Group: -
In -person A: -
In- person B: Low – medium depending on which one we're talking about; some uncertainty about the potential impacts of Toxoplasmosis

Group agrees.

-NEMATODES			
Capillariasis	*Capillaria spp.*	1	L

Group agrees to list as "low".

Contracaecum - helminths	*C. eudyptulae~~, C. spiculigerum~~*	1	M-H
		3	L

Final risk rating – hazard group 1
Online Group: -
In -person A: -
In- person B: M-H

IB: C. *spiculigerum* apparently misidentified, so only *C. eudyptulae relevant*

Final risk rating – hazard group 3
Online Group: -
In -person A: -
In- person B: Low

Group agrees.			
-TREMATODES			
Trematodiasis	*Cardiocephaloides physalis*	1	L
Trematodiasis	*Galactosomum angelae*	1	L-M
Trematodiasis	*Mawsonotrema eudyptulae*	1	M
Renal fluke	*Renicola*	1	L
Group agrees on the listings as presented by In- person group B.			
ECTOPARASITES			
Fleas	*Parapsyllus* sp	1	L-M
		2	L-M
Final risk rating – hazard group 1 Online Group: - In -person A: - In- person B: L-M **Final risk rating – hazard group 1** Online Group: - In -person A: - In- person B: L-M **JHu:** They're there but prevalence is probably reasonably low, uncertainties remain **JHi:** Single cases years ago of tick paralysis in domestic animals around Phillip Island (Millowl)			
Lice	*Austrogonoides waterstoni*	1	L-M
Final risk rating: Online Group: In -person A: In- person B: High prevalence, low consequence			
Mites	*Ingrassia eudyptula,* & *Veigaia* sp.) and others	1	L-M
Final risk rating: Online Group: - In -person A: - In- person B: High prevalence, low consequence Group agrees.			
Ticks	*Ixodes eudyptidis, Ixodes uriae*	1	M-H
		2	M-H
Final risk rating – hazard group 1 Online Group: - In -person A: - In- person B: M-H **Final risk rating – hazard group 2** Online Group: - In -person A: - In- person B: M-H Group agrees.			

8.3.14 Knowledge gaps

- Ectoparasites
  MS: And what the parasites are carrying
  IB: They are certainly transmitting Babesia
  LS: Probably most efficient to target sick/ dead birds for the efficient use of resources

- Haemoparasites

- Avian influenza surveillance in wild birds in the area (e.g. shearwater)
  RV: Look at the baseline of the occurrence of Avian influenza (LPAIV) in LPs and shearwaters

  RVH: Vaccine?
  RG: No vaccine known to work in penguins, needs to be injected, not practical

LR: Paper released on vaccination trials in African penguins by **DRo and LRo**
https://bvajournals.onlinelibrary.wiley.com/doi/10.1002/vetr.3616
RV: Is widely discussed, has major limitations in terms of feasibility

LRo:
When you got HPAI it's going to impact the animals, there is very little you can do, the only thing that has a chance of working is vaccination, so generally very important to discuss this in advance; Yes, not very practical but LPs are colony-nesting birds so it might be possible to do
Would take a lot of planning, might be expensive, but probably worth it
RV agrees, in some cases it takes only a few weeks for 90 % of the population to die. As soon as the first HPAI cases are reported in Australia, it will likely be too late
Best option is to vaccinate captive LPs now and test efficiency of the vaccine

RG: Probably almost impossible to even import the vaccine
FS: Would penguins even be given priority? Compared to other species that are endangered?
JHi: High economic value and high emotional value

RVH: Vaccine Testing?
RG: Needs to exclusively go through the ACDP
ML: Would probably need to prepare everything in advance, but may not give us enough time, Melbourne Zoo would currently be open to volunteer their penguins for research

- Cryptosporidium
  IB: Gathering further data would be very simple, could do it easily through faecal collections; there is a parasitologist at The University of Melbourne who specialises in Crypto

- Toxoplasmosis
  ML: Could generally investigate more individuals that die in the colony

- Intoxications

- Not a lot of necropsies get done generally
  JHi: Offered to do this regularly for animals that die/ have the be euthanised

8.3.15 Risk management

- Examples on how Critical Control Points can be identified
    - Vitali et al. 2023 – Koala DRA https://wildlifehealthaustralia.com.au/Portals/0/ResourceCentre/BiosecurityMgmt/KDRA%20Report%20v1.2_FINAL.pdf
    - DRA for Eastern barred bandicoots https://www.cpsg.org/sites/cbsg.org/files/documents/EBB%20Disease%20Risk%20Analysis%20Report%20FINAL.pdf
- WHA Avian influenza toolkit https://wildlifehealthaustralia.com.au/Incidents/Incident-Information/high-pathogenicity-avian-influenza-information
- Some protocols already in place at PINP
- Should put more examination protocols in place, can put together a list, e.g. (Western Ground Parrots)
- Protocols can include:
    - Facility recommendations
    - Recommendations for sample collection and analysis
    - Justifications for sample collection

8.3.16 Risk communication

- Engagement today successful
- Written report will be circulated for comment

8.3.17 Implementation and review

- Annual review and management
- Ongoing management informed by annual review which should be formally documented; e.g.

Management Target	Goals	Actions	Frequency	Responsibility	Success measure(s)	Data required
Feral domestic cats of Phillip Island (Millowl) and French Island	Reduced environmental contamination with oocysts	Integrated cat eradication program informed by target density that will achieve goal	Ongoing	Parks Victoria/ Phillip Island (Millowl) Nature Park/ French Island Landcare	Target density met and maintained	Program monitoring data
[…]	[…]	[…]	[…]	[…]	[…]	[…]

- Ongoing surveillance
    - Unknown hazards may not be detected until mortalities are seen

8.3.18 Summary

- DRA is a “living”, “evolving” document reliant on stakeholder collaboration and communication to achieve its purpose

- Tailor recommendations to financial, logistical and practical constraints

8.3.19 Q&A

- **JHu:**
  Where do we go from here? Would be good to get everybody's input a bit more
  - **RVH:** Ideally go through Critical Control Points
  - **LS:** Start off with high risks and then look at moderates etc., also make it dependent on whether actions are required

- **MS:** effectiveness of mitigation measures needs to be evaluated
  - **RVH:** People from the PINP need to be identified who will implement/ support recommendations of the DRA
  - **RV:** Suggests to have specific metrics to quantify success/ benefits

- **IB:** Sometimes historical material is useful, the basement of The University of Melbourne holds a whole bunch of samples (300 tissue samples from LPs from PINP), **Richard Norman's** thesis includes all reports although slides got thrown out
  - **ML:** There should also be PM reports from the Melbourne Zoo penguins
  - **LD:** Should be kept for 20-30 years

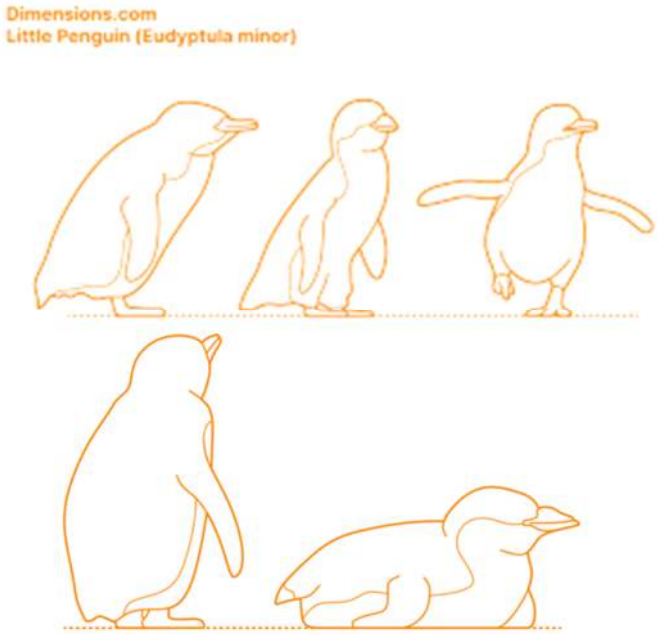
- **AC:** Limited resources but long list of risks, careful prioritisation is necessary
  - **LS:** Threats tend to be heterogeneous; some threats may be extreme at the current colony but may not affect other populations, population needs to grow and spread across the landscape to be resilient; landscape needs to be taken into account

- **Arlene Rutherford (AR):** Assess values of different strategies, what will give you the most "bang for your buck"
  - **MS:** Cost-benefit analysis might really be important

8.3.20 Next steps

- Critical Control Points are being sent out
- All ratings for hazards put together
  - ⇨ Ongoing draft documents are being sent out with timelines

8.4 Necropsy submission form



Individual sampling ID:.....
Location.....
GPS co-ordinates.....
Species.....
Age.....
(estimate: subadult, adult)
Sex.....
Identification.....
.....
.....
Weight.....
Date.....
Capture time .....
Release time.....
Date of death.....
Date of submission.....

*Please note any lesions, masses, trauma and abnormalities on diagrams at point of initial assessment.

Number of animals affected:.....Number sick.....
Population estimate:.....Number dead.....
Sex affected.....Age affected.....
Submitters name.....Contact number.....
Email address.....Department.....
.....

Any abnormal circumstances prior to death, including environmental events, e.g. weather, recent rainfall, sea conditions, recent local use of chemicals, changes in ground water levels, changes in domestic animal management:

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Any abnormal clinical signs noted prior to death?

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8.5 Recommended veterinary field kit equipment list

Equipment	Number
Copan dry swabs – https://www.interpath.com.au/brand/copan&p=9 552C regular FLOQswab in tube (orange cap)	6 / bird
Amie's culture medium swabs	1 / bird
Chlorrhexidine soaked swabs to clean blood collection site	1 / bird
26G and 27G needles	1 / bird
26G butterfly catheters	2 / bird
Flushing needle	7
1ml, 3ml, 5ml, 10ml syringes	2 / bird
Paediatric lithium heparin blood tubes	1 /bird
Styrofoam blood tube holder	1
Microscope glass slides	2 / bird (buy in a pack of 50)
Slide holders	1 large plastic container or multiple single cardboard holders
Micropore tape – in event of excessive bleeding	1 roll
Kaltostat – alginate wound dressing to place over blood collection site with firm digital pressure	1 square pack of dressing material
Gauze swabs	2 packets (200)
Formalin filled small 5ml containers	1 / bird
Ethanol filled small 5ml containers	1 / bird
F10 disinfectant spray	
Alcohol spray or soaked gauze swabs	
Holding bags	
Bluey' absorbent plastic backed towel	
Small esky	
Pesola scales	
Calipers	
Focal light	
Camera	
Stethoscope	
Disposable gloves for handling	1 pair / bird S/M/L
Face masks for handling	P2
Clinical exam sheets	1 / bird
Laboratory submission forms	1 / bird
Pens & pencils for labelling	2 of each
Portable freezer	1
Access to -80'C freezer	
Clavulox injectable antibiotic	1 bottle
Meloxicam 5mg/ml injectable anti-inflammatory	1 bottle
Tramadol injectable 100mg/ml vials	1 box
Tricin eye ointment	1 tube
Pentobarbitone	1 bottle

Wound dressing products including hydrogel paste,iodosorbpaste,melolite,fixomull,fixomull	Multiple tubes and sheets
Fluorescein strips	5
Anaesthetic machine	
Isoflurane	
ET tubes	Size 2,3,4 Coles uncuffed ET tubes
Local anaesthetic spray	1 bottle
Hartmanns compound sodium lactate fluid bag	1L bag
0.9% Sodium chloride fluid bag	1L bag