Disease Risk Analysis for Birds at Ramsar Wetlands in Port Phillip Bay (Western Shoreline) and Bellarine Peninsula

11 - 12 March 2020

Melbourne Water

THE UNIVERSITY OF MELBOURNE

CORANGAMITE CATCHMENT MANAGEMENT AUTHORITY

Disclaimer

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This report can be downloaded from the CPSG website: www.cpsg.org.

Table of Contents

List of Figures

List of Tables

ACRONYMS AND ABBREVIATIONS

ACDP	Australian Centre for Disease Preparedness, formerly known as Australian Animal Health Laboratory (AAHL)
AUSVETPLAN	Australian Veterinary Emergency Plan
Agribio	Centre for AgriBioscience
AGY	Avian gastric yeast
AMR	Antimicrobial resistance
APMV	Avian paramyxovirus
CCMA	Corangamite Management Authority
CCPs	Critical control points
CMA	Catchment Management Authorities
CSIRO	Commonwealth Scientific and Industrial Research Organisation
DAWE	Australian Department of Agriculture, Water and the Environment
DELWP	Victorian Department of Environment, Land, Water and Planning
DRA	Disease risk analysis
EPA	Environment Protection Authority
HPAI	Highly pathogenic avian influenza
IBDV	Infectious bursal disease virus
IUCN	International Union for Conservation of Nature
JEV	Japanese encephalitis virus
LPAI	Low pathogenic avian influenza
MVEV	Murray Valley encephalitis virus
MW	Melbourne Water
NSW	New South Wales
OBP	Orange-bellied parrot
OP	Organophosphate
OIE	World Organisation for Animal Health
PBFD	Psittacine Beak and Feather Disease
PCB	Polychlorinated biphenyl
PFAS	Per- and polyfluoroalkyl substances
POP	Persistent organic pollutant
PPBBP	Port Phillip Bay and Bellarine Peninsula
QLD	Queensland
VIC	Victoria
WA	Western Australia
WHA	Wildlife Health Australia
WTP	Western Treatment Plant

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This disease risk analysis would not have been possible without the significant input and advice from many stakeholders. We are very grateful to Corangamite Catchment Management Authority and Melbourne Water for funding and Melbourne Veterinary School for providing in-kind support. Dr Richard Jakob-Hoff played a key leadership role as facilitator and we were fortunate to have him guide us through this process. The generosity of invited workshop participants and their affiliated organisations (Appendix 4) in providing the time and resources to attend and contribute to this DRA workshop and to review this report is greatly appreciated. We would also like to thank Will Steele, Suelin Haynes, Heather Graham, Robert Bones, Richard Loyn, Keren Cox-Witton, Debbie Eagles, Jemma Bergfeld, Mark Hawes, Falk Wicker and Ian Beveridge, who have provided invaluable technical knowledge, advice and feedback for the development of the workshop briefing notes as well as this final report. And last, but not least a big Thankyou to Rebecca Gang for her research assistance throughout this project and all her work on disease summaries, species and hazards lists, briefing notes and this report. We were looking forward to welcome Dr Robert Dusak from the USGS National Wildlife Health Center (NWHC), Madison, Wisconsin, USA, who kindly agreed to share some North American perspectives and experiences with us. Unfortunately, COVID-19 had other plans and put all international travel on hold. Nevertheless, we look forward to valuable collaborations with the USGS NWHC in the future.

Anke Wiethoelter & Pam Whiteley

Workshop Participants (l. to r.)

Back: Steven Liu, Jemma Bergfeld, Rebecca Gang, Kate Brandis, Jasmin Hufschmid

4th row: Will Steele, Elyse Herrald-Woods, Anna Meredith, Lee Berger, Leanne Wicker

3rd row: Robert Bone, Keren Cox-Witton, Clare Death, Pam Whiteley

2nd row: Naomi Davis, Suelin Haynes, Rachel Pritchard, Yonatan Segal

Front: Anke Wiethoelter, Alistair Legione, Richard Jakob-Hoff

Not depicted: Paul Eden, Simon Firestone, Stacey Lynch, Andrew Peters

EXECUTIVE SUMMARY

Relative to our knowledge of diseases of people and domestic animals, the study of wildlife health is still in its infancy. A disease risk analysis (DRA) provides a systematic, evidence-based process to document and analyse available disease information and develop a risk management plan incorporating the expertise of key stakeholders. The goal of this DRA was (1) to identify and prioritise health hazards for wild birds present at the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula (PPBBP) Ramsar site, (2) to identify mitigation strategies and (3) to inform further research and/or management needs.

In the focus of this DRA were 111 infectious and non-infectious health hazards (Appendix 2) to wild birds (Appendix 1) through their interactions with the wetlands identified in an extensive review of the literature. To facilitate prioritisation of hazards, wild bird species, which formed the populations of interest, were divided into three functional groups: shore and water-based, land-based and predatory birds. Small groups of workshop participants were allocated to one of the three groups to review and prioritize the hazards against the likelihood of exposure and consequence to population criteria (Table 3) recording their results in a matrix (Tables 4-6).

Hazards that were highest-ranked by multiple functional bird groups (Table 8) were assigned a higher weight, resulting in three hazards being selected for a detailed qualitative risk assessment, namely botulism, pasteurellosis, and pollutants and contaminants.

The overall risk of **botulism** to wild birds at the PPBBP Ramsar site was ranked as high with a high confidence in this risk estimation.

The overall risk of **pasteurellosis** to wild birds at the PPBBP Ramsar site was ranked as medium with the potential to move to high risk (based on overseas experience) with a low-medium confidence in the risk estimation.

The overall risk of **pollutants** to wild birds at the PPBBP Ramsar site is ranked as high with a high confidence in this risk estimation.

Risk mitigation actions and research actions were recommended for all three hazards and revealed some similarities (Table 27). The development and implementation of a comprehensive surveillance program to monitor syndromes, diseases and mortality events for early diagnoses and response as well as providing wildlife health baseline data would not only mitigate the risk of botulism, pasteurellosis, pollutants and contaminants, but also address other causes of mortality and morbidity. Furthermore, this DRA highlighted the fact that several aspects of diseases in Australian wild birds are poorly understood. International collaborations could provide strategic support and help to bridge existing knowledge gaps to enhance the understanding of Australian disease ecology and foster wildlife and ecosystem health. Australia is still at the beginning of a significant learning curve on how to improve wildlife health management. However, this DRA with a wide range of stakeholders is a shining example of progress. It applied the process to an ecosystem, which represents a novel and innovative use of this framework.

INTRODUCTION

The Disease Risk Analysis (DRA) Process

Relative to our knowledge of diseases of people and domestic animals, the study of wildlife health is still in its infancy. While the volume of literature on the topic is growing exponentially much valuable information is unpublished but in the heads of individuals. A DRA workshop brings together a group of knowledgeable people to work through a structured, facilitated process. By drawing on their combined knowledge, expertise and understanding of the topic, this provides a basis for reducing the level of uncertainty of the risk analysis, developing stakeholder consensus on the priority health hazards to the populations of interest and the means of managing associated risks.

The DRA framework and tools used in this analysis are described in the *Manual of Procedures for Wildlife Disease Risk Analysis* jointly published by the IUCN-Species Survival Commission and the World Organisation for Animal Health (OIE) (Jakob-Hoff et al., 2014). This is an iterative process that, when fully implemented, generates new information and insights that inform future reviews of the DRA. The overall framework is comprised of six distinct, but interlinked, steps as shown below.

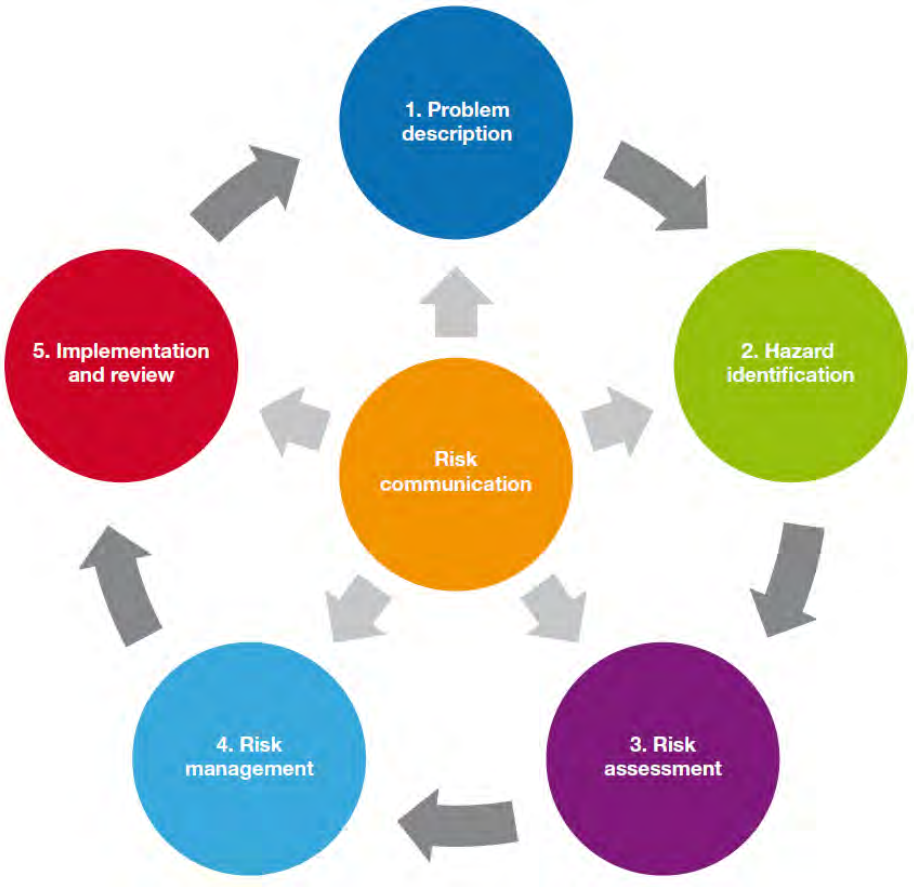


Figure 1. Disease risk analysis process steps (Jakob-Hoff et al., 2014)

PROBLEM DESCRIPTION

Justification and background for this DRA

Wetlands of international significance are listed and protected under the Ramsar Convention (The Ramsar Convention Secretariat, 2014). They are biodiversity hotspots and provide a refugium for seabirds, migratory shorebirds and waterfowl, as well as other native fauna and flora. In Victoria, the Department of Environment, Land, Water & Planning (DELWP), together with Parks Victoria, Melbourne Water and regional catchment management authorities are responsible for conserving and managing 12 Ramsar sites with the aim of maintaining their ecological character. One of these sites is Port Phillip Bay (Western Shoreline) and Bellarine Peninsula (PPBBP) within the local government areas of Greater Geelong, Wyndham, and Melbourne Metropolitan area (Figure 2).

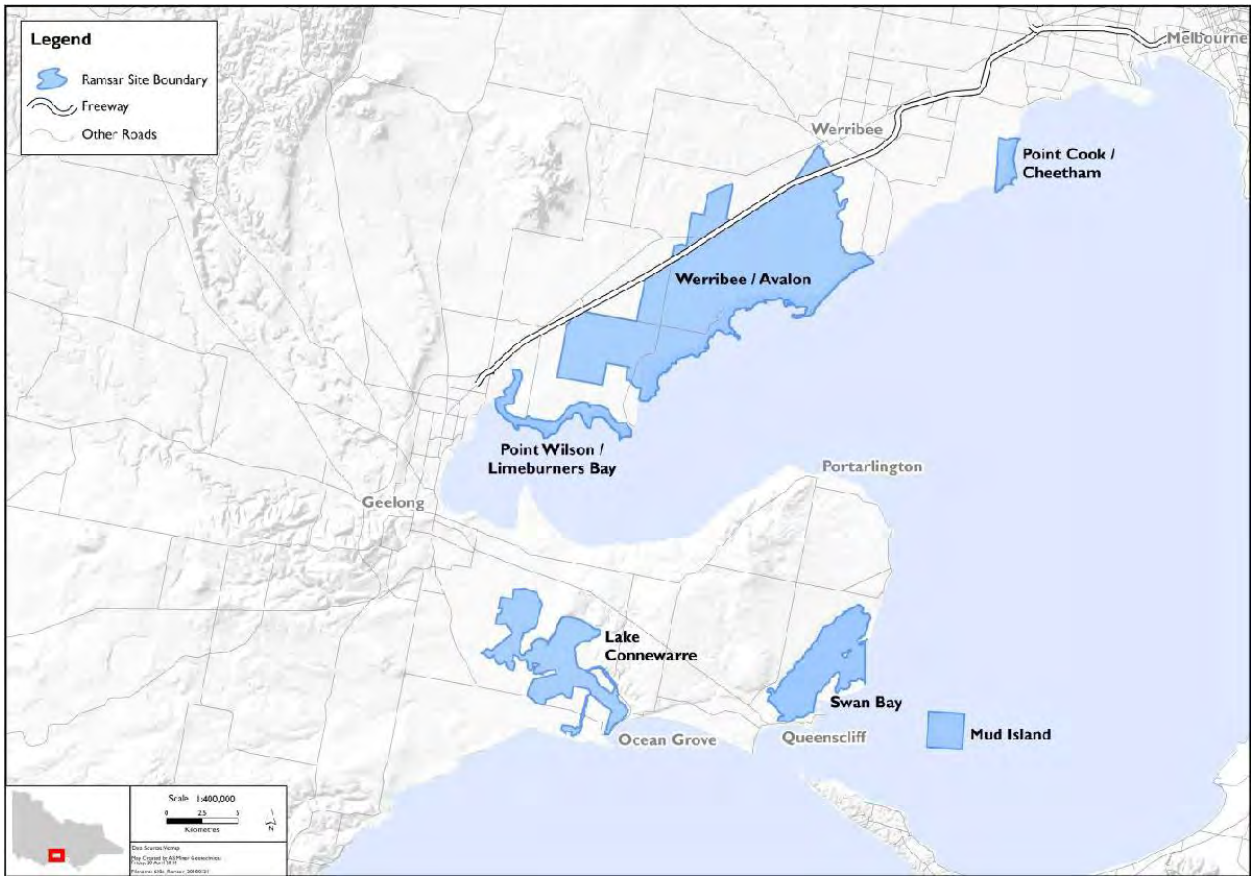


Figure 2. Map of the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar site (DELWP, 2018).

This Ramsar site comprises a high diversity of habitat types, ranging from interconnected freshwater wetlands, ponds, rivers, and lakes to coastal waters including intertidal zones and saltmarsh as well as a wastewater treatment plant. It supports several vulnerable, endangered, or critically endangered wetland-dependent species as well as regularly supports 20,000 or more waterbirds and 1% of the individuals in a population of several species of waterbirds (see APPENDIX 1).

In response to the 2016 Victorian Auditor-General report on 'Meeting obligations to protect Ramsar wetlands' (Frost, 2016), the recent PPBBP Ramsar Site Management Plan was developed and comprised a risk assessment. Priority threats identified back then included climate change; urban development; wastewater, stormwater, and catchment inflows; recreational activities; duck hunting; and invasive

species. However, one of the key knowledge gaps identified in this risk assessment was 'causes and effects of pathogens and disease on waterbirds (e.g. botulism, avian cholera)' (DELWP, 2018). This DRA addresses this gap.

DRA aims

1. Apply a transparent and scientifically sound framework to prioritize Ramsar wetland disease risks for birds, review existing mitigation strategies and identify opportunities and knowledge gaps in managing priority diseases in wild birds.
2. Directly support implementation of the management strategy as per '4.5 Investigate the causes and potential mitigation strategies for avian diseases in the Ramsar site' listed in the PPBBP Ramsar Site Management Plan (DELWP, 2018).
3. Contribute to DELWP's management plans in response to the 2016 Victorian Auditor-General report on 'Meeting obligations to protect Ramsar wetlands' (Frost, 2016).
4. Help to build a good working collaboration between all important stakeholders that may lead to future joint research and innovation partnerships (e.g. Australian Research Council Linkage Projects) and improved wildlife and ecosystem health outcomes.

DRA goal

The goal of this DRA is to identify and prioritise health hazards for wild birds present at this Ramsar site, to identify mitigation strategies and to inform further research and/or management needs.

DRA scope

- Review available data and literature on wild bird species as well as potential pathogens, non-infectious diseases, and other health hazards relevant to the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar wetland site
- Conduct a stakeholder review for expert analysis of disease risks to birds inhabiting this site
- Hold a 2-day disease risk analysis workshop to reach consensus on:
    - prioritization of the specific diseases and bird species that should be targeted for surveillance.
    - cost-effective ways to carry out surveillance for diseases in wild birds in the PPBBP Ramsar site, and
    - strategies to minimise the impact of avian diseases that are known occur in the PPBBP Ramsar site.
- Summarise the findings and outcomes in a written report

DRA focus

Infectious and non-infectious health hazards to wild birds through their interactions at the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar wetlands.

DRA questions

The questions addressed by this DRA are:

1. Which diseases pose the most significant risk to wild bird populations at the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar wetland site?
2. What is the likelihood and consequences of disease to wild birds, resulting from interactions with this Ramsar site?

3. What factors contribute to the risk of disease outbreaks in wild birds using this Ramsar site?
4. How can these risks be effectively managed?
5. Where should research be directed to reduce the level of uncertainty in this risk analysis and further inform disease risk management for this Ramsar site?

Acceptable risk

Life is full of risk and trade-offs must be made between what level of risk is acceptable and what is not. The DRA process recognizes this through a statement, developed by workshop participants, of the level of risk acceptable within the biological system under consideration. The discussion was largely driven by the following questions:

- How does the Ramsar site impact people and how do mortality and morbidity in the wild birds here affect people? The political, biological, personal and economic effects were discussed and explored.
- What situation would trigger a management response? Scenarios of bird mortalities, population monitoring and diseases resulting in significant welfare effects were explored.
- What values are attached to different components of the biological system under consideration by different people and organisations?

The workshop participants decided that acceptable levels of risk are risks that have:

- no long-term impacts on bird abundance and biodiversity
- no health impacts to the wider community
- no significant economic impact
- no significant disruption to recreational use of the Ramsar site
- no sustained reputational damage to organisations responsible for wetland management

This statement informed both the prioritization of hazards and the selection of risk mitigation strategies and actions.

Assumptions and limitations

All decision making involves some assumptions and is limited by various constraints. Making these explicit is an essential part of any wildlife disease risk analysis as information is often scarce and resources limited. This provides a level of transparency that enables conclusions and recommendations arising to be considered within this 'real world' context. The following were identified by the organisers and the subject of further discussion during the workshop.

Assumptions

- Wild birds present at this Ramsar site are susceptible to the full range of health hazards recorded to date in the same species or a closely related species elsewhere.
- Wild birds present at this Ramsar site are susceptible to pathogens that have been demonstrated to have a broad host range across birds.
- Wild birds present at this Ramsar site are mobile and include resident as well as nomadic and migratory species.
- Presence of a pathogen in wild birds elsewhere in Victoria will increase the likelihood that this pathogen is present at this Ramsar site.

Limitations

- Compared to disease knowledge available for domestic animals and humans, the understanding of the range of potential pathogens of wild birds and the epidemiology of these pathogens is poor.

- There have been very few systematic studies that have proactively screened for potential pathogens and provide quantifiable data on morbidity and mortality in wild birds. Most information relates to individual case reports and therefore only provides information on disease presence and/or absence.
- Most diagnostic tests are not validated for wild birds (Gardner et al., 1996).
- Data on mortality and causes of mortality is biased towards larger species (i.e. larger carcasses) and/or species of particular interest which are under increased observation (Ward et al., 2006; Flint et al., 2010).

Populations of interest

The populations of interest identified for this DRA are wild birds inhabiting the Ramsar wetlands located in the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula of Victoria. This Ramsar site supports large numbers of shore- and waterbirds. Maximum annual counts between 2011-2015 resulted in estimates of 22,900 shorebirds and 110,000 non-shorebirds (DELWP, 2018). Additional to these, this analysis includes consideration of wild bird species inhabiting adjacent streams and land.

A list of the bird species recorded at this Ramsar site is included in APPENDIX 1. This list also includes introduced and pest species. While disease effects on these latter species are not of direct concern, they could still play an important role in the disease dynamics for native species. To facilitate prioritisation of hazards, the bird species list was divided into three functional groups (shore and water-based, land-based and predatory birds).

Interaction between avian and in-contact species

Wild birds at this Ramsar site are highly mobile. Some species migrate across Australia or even along international migratory bird flyways (e.g. East Asian Australasian and West Pacific), while interacting with other wildlife (mammals, reptiles, amphibians, fish, and insects), domestic animals, and humans. These interactions provide potential transmission pathways for infectious diseases between and among these populations. Infectious and non-infectious diseases therefore not only represent significant health hazards to birds, but also potentially to other wildlife, domestic animals, humans, and the catchment area ecosystems. The extent and impact of these hazards however is currently unknown; a lack of data as well as several knowledge gaps make it difficult to assess and quantify disease risks present. The primary aim of this DRA is to assess the likelihood of contact between the identified hazards and the avian species of concern, and the consequences to them if contact occurs.

The possible spectrum of interactions between wild birds and in-contact species at the PPBBP Ramsar site is very diverse. Some infectious diseases included in the hazard list are zoonotic, which means they are transmissible between animals and humans. Other diseases (so-called vector-borne diseases) are transmitted by vectors such as mosquitoes, ticks, and fleas.Anthropogenic activities at or near this site include boating, fishing, bait collection, swimming, walking, nature observation, research, birdwatching, (duck) hunting as well as sewerage treatment, agriculture, and salt production. Figure 3 below depicts a simplified and stylized overview.

Importantly, these interactions between species must be considered as bidirectional pathways for health hazards (i.e. wild birds to in-contact species and in-contact species to wild birds). Mitigation strategies for wild bird health at this Ramsar site may therefore require actions off-site (e.g. improved farm biosecurity).

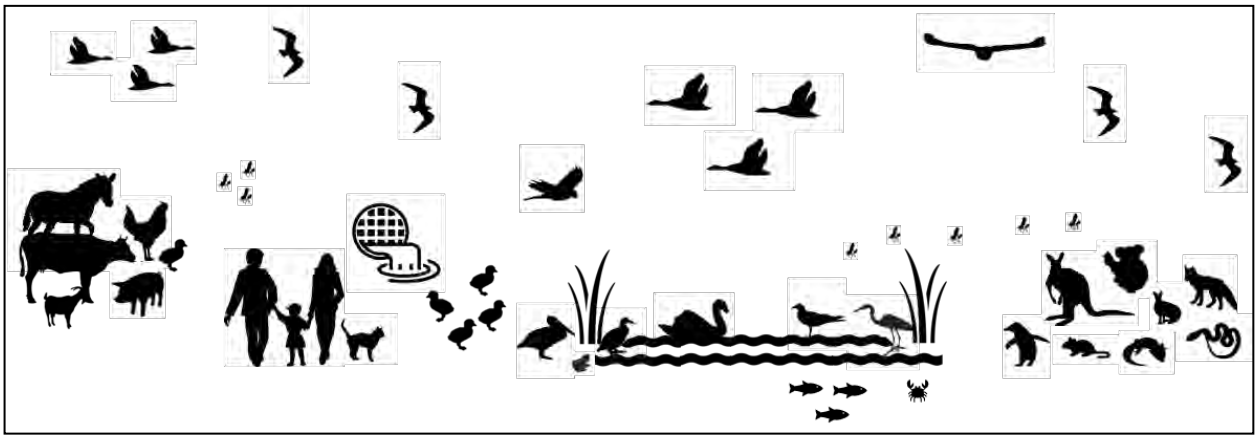


Figure 3. Interactions between resident, nomadic and migratory waterbirds with other wildlife, livestock, domestic animals, and humans.

Diseases recorded at this Ramsar site to date

Due to fragmentation of jurisdictions, it is difficult to gain a complete picture of bird mortality and morbidity at this Ramsar site. Figure 4 presents a summary of data reported by Melbourne Water, Parks Victoria, and Wildlife Health Victoria: Surveillance at the Melbourne Veterinary School. It reflects the discontinuity in reporting and therefore severely underestimates actual annual mortalities at the site.

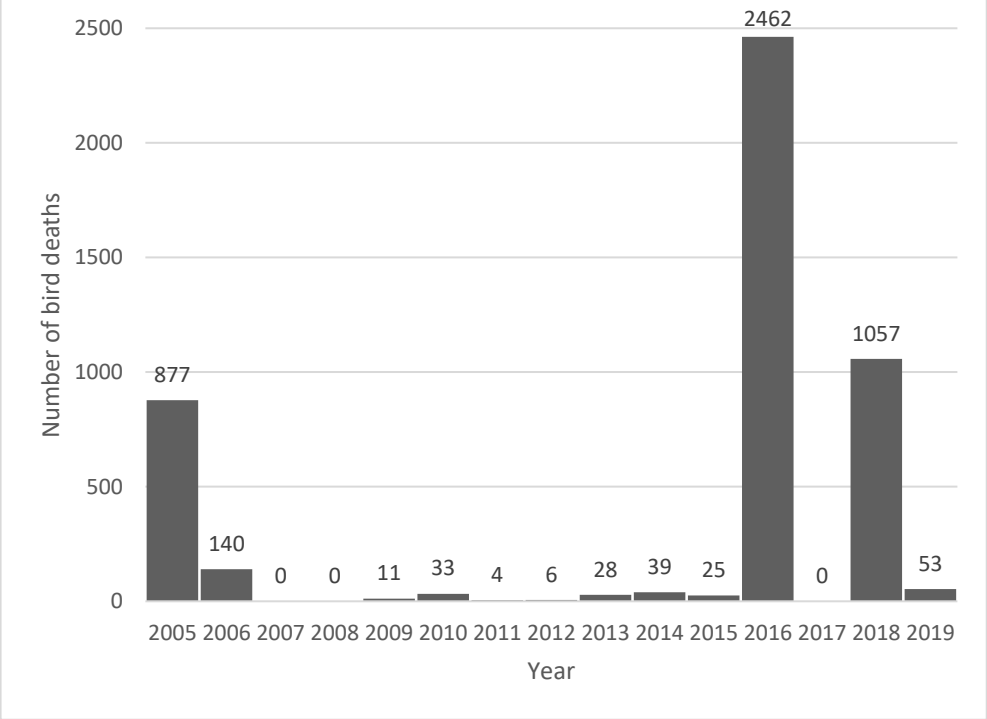


Figure 4. Reported bird deaths from the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar site and immediate surrounding areas, 2005-2019.

Table 1 summarises reported diagnoses from birds at this Ramsar site and immediate surrounding areas. This has been compiled from mortality reports from Melbourne Water and AgriBio, pathology data from Wildlife Health Victoria: Surveillance at the Melbourne Veterinary School and published literature. Many gaps exist due to the incompleteness of databases, difficulties accessing all reported causes of disease and lack of reporting of geographical sources in the literature.

Table 1. Diagnoses in birds at the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar site.

Disease	Year	Postcode	Bird species affected	Comments
VIRAL				
Avian influenza	2007 – 2012	3030	Anseriformes and Charadriiformes	Prevalence of Low Pathogenic Avian Influenza was found to be 3.1% (+/- 0.3%)[1]
	2014 – Feb	3030	Pacific black duck	Not strains H5 or H7
Poxvirus	2016/2017	3220	Australian magpie	
Psittacine beak and feather disease	2016 – Mar	3216	Sulphur crested cockatoo	
BACTERIAL				
Botulism	2008 – Oct	3212	Sacred ibis	Confirmed
	2012 – Dec	3030	Sacred ibis	Suspected, not confirmed*
	2013 – Mar	3030	Australian shelduck Chestnut teal	Suspected, not confirmed*
	2014 – Feb 2014 – Nov	3018 3212	Pacific black duck Sacred ibis	Confirmed Suspected, not confirmed*
	2015 – Feb 2015 – Mar	3212 3030	Magpie goose Australian pelican	Suspected, not confirmed*
	2015 – Dec to 2016 - Feb	3030	Australian shelduck Avocets Silver gulls Pacific black ducks Shoveler Australian pelicans Hardhead Sacred ibis Black swan	Large mortality event of multiple species with almost 3000 deaths. Botulism toxin isolated. Peak in swan deaths lagged behind duck deaths.
	2016 - Mar	3212	Magpie goose	Suspected, not confirmed*

Disease	Year	Postcode	Bird species affected	Comments
	2018	Mud Island	Silver gulls	Large mortality event, approximately 1000 deaths
	2019 – Jan	3030	Australian shelduck Australian pelicans	Large mortality event. Suspected, not confirmed.
Enteric bacteria	2014 – Feb	3030	Australian pelican	Range of pathogens cultured, including *E. coli*
Mycobacteriosis (avian tuberculosis)	2010 – Jan 2005 - 2014	3212	Australian bustard (captive) Brolga (captive)	Confirmed in 7 brolga over 10 years[2]
Pasteurellosis (avian cholera)	2013 – Mar	3030	Chestnut teal	Identified during a mortality event due to suspected botulism
FUNGAL				
Aspergillosis	2016	3212	Magpie goose	
PARASITES				
Cestodiasis (tapeworms)	2010 – Jan 2011 – Mar 2013 - Mar	3227 3226 3018 3227	Little penguins Short-tailed shearwaters Shy albatross Short-tailed shearwater	Clinical significance unknown.
Nematodiasis (roundworms)	2010 – Jan 2010 – Oct 2011 – Jan 2011 – Mar 2014 – Jan 2015 – Mar	3227 3226 3018 3016	Little penguins Short-tailed shearwater Little penguin Australian pelicans Australian pelicans	Clinical significance unknown. Two genera found*: Contracaecum sp., Tetrabothrius sp.*
Trematodiasis (flukes)	2015 – Mar	3016	Australian pelican	Clinical significance unknown
NON-INFECTIOUS				
Algal toxins	2005 - 2006	3030	Australasian shoveler Chestnut teal Pink-eared duck	Over 700 waterfowl were found dead at Melbourne Water Western treatment plant Birds were in full moult of primary feathers, making them flightless. Investigations failed to determine a definitive cause of death, but findings suggested three possible causes: avian botulism, poisoning from toxic algae, and drowning after becoming trapped in drop-inlets to underwater transfer pipes.[3]

Disease	Year	Postcode	Bird species affected	Comments
Anticoagulant toxicity	2018 – Jun to Sep	3227 Bellarine	Nankeen kestrels Barn owls	8 confirmed rodenticide toxicities with a marked increase in mortality across the State thought likely to be the result of a combination of factors including increased breeding and reduced prey availability.[4]
	2019 – Jan 2019 – Mar 2019 – May	3212 3030	Barking owl Whistling kite Raven	
Organophosphate (OP) toxicity	2018 – Mar	3030	House sparrows	Mortality event with 15+ deaths. Confirmed OP toxicity of unknown source.

[1] Grillo VL, Arzey KE, Hansbro PM, Hurt AC, Warner S, Bergfeld J, et al. 2015 Avian influenza in Australia: a summary of 5 years of wild bird surveillance. Australian Veterinary Journal 93(11): 387-93.
[2] Hodge PJ, Sandy JR, Noormohammadi AH, 2019, Avian mycobacteriosis in captive brolgas (*Antigone rubicunda*). Australian Veterinary Journal 97(3): 81-6.
[3] Steele WK, 2008. A cluster of duck deaths at the Western Treatment Plant, Werribee, Victoria, during 2005-06. Australian Field Ornithology 25, 109.
[4] Hawes M, Whiteley P, 2018, Birds of Prey Mortality and Morbidity event 2018 - Victoria. Agriculture Victoria Research Wildlife Victoria.
* Definitive diagnosis is difficult see section 'Botulism – Diagnosis, treatment, control and prevention' for details

Current actions to manage the health of this Ramsar wetland site

Management responsibilities for the PPBBP Ramsar site are distributed across 16 different agencies depending on geographic and/or thematic area (Table 2). This demonstrates the importance of cross-agency collaboration to achieve effective wildlife health outcomes.

Table 2. Managing agencies for the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar wetland site (DELWP, 2018).

Agency	Management area
Avalon Airport	Werribee/Avalon – Avalon airfield
Borough of Queenscliff	Foreshores adjoining urban areas
Central Coastal Board	Coastal action plan and guidelines within the region
City of Greater Geelong	Avalon Beach to Limeburners Bay, Limeburners Lagoon Foreshores adjoining urban areas
City of Wyndham	Foreshores adjoining urban areas
Corangamite Catchment Management Authority (CCMA)	Surrounding catchment, inflowing streams, drainage
Department of Defence	Point Wilson Explosives Area Stingaree Bight (declared naval waters)
Department of Environment, Land, Water and Planning	Unreserved crown lands at: • Point Cook / Cheetham • Werribee / Avalon • Point Wilson / Limeburners Bay
Environment Protection Authority Victoria	Monitor environmental quality at the whole Ramsar site
Melbourne Water	Werribee/Avalon – Western Treatment Plant and adjacent foreshore Water and sewage services
Parks Victoria	National parks, marine parks, and reserves at: • Point Cook / Cheetham • Werribee / Avalon • Point Wilson / Limeburners Bay • Swan Bay • Mud Islands • Lake Connewarre and Reedy Lake
Port Phillip & Westernport CMA	Surrounding catchment, inflowing streams, drainage
Victoria Fisheries Authority	Recreational and commercial fishing at the whole Ramsar site

The Melbourne Water document '*Managing sick, injured or dead wildlife procedure*' outlines actions to be taken when three or more animals are found sick, injured or recently dead during any 24-hour period within areas of less than 1 hectare at a Melbourne Water property. Actions include:

- Notification to the Melbourne Water management and the Emergency Animal Disease Watch Hotline (1800 675 888)
- Collection of carcasses/sick animals
- Documentation of cases and areas
- Further laboratory testing at AgriBio or the Melbourne Veterinary School
- Safe disposal of carcasses

In general, wildlife health incidents (i.e. unusual signs of disease or deaths in wildlife) at this Ramsar site should be reported to the Victorian Wildlife Health Australia Coordinator (see https://wildlifehealthaustralia.com.au/AboutUs/ContactDetails.aspx for up-to-date contact details).

HAZARD IDENTIFICATION

Hazard list – sources of data

The hazard list (APPENDIX 2) was compiled using a combination of published literature, leading textbooks on wild bird and wildlife disease (Friend, 1987, Ladds, 2009) and reports on surveillance and bird health in grey literature. Published literature was identified by searching PubMed, Scopus and Google Scholar with terms: ((("wild birds" OR "avian" OR bird*)) AND ("australia" OR australia*)) AND ("disease" OR "infection" OR infectio*). Papers were excluded if they discussed disease only relating to humans, domestic animals or mammals, focussed on treatment of captive species or laboratory settings or genomics or immunology as well as publications from different climatic zones to Victoria. Hazards that have unknown clinical and epidemiological significance in wild birds but are known to cause disease in domestic or introduced birds, humans or other mammals were included in the list. Parasitic diseases were only included if there are reports of clinically significant infections. Therefore, this is not an exhaustive list of avian parasites.

Hazard prioritization

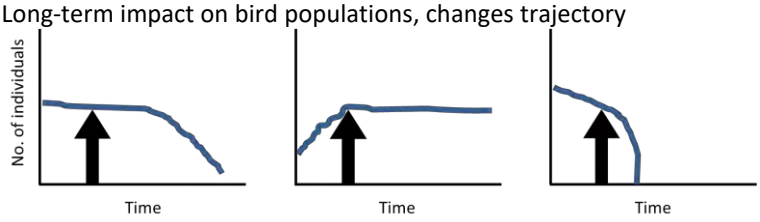
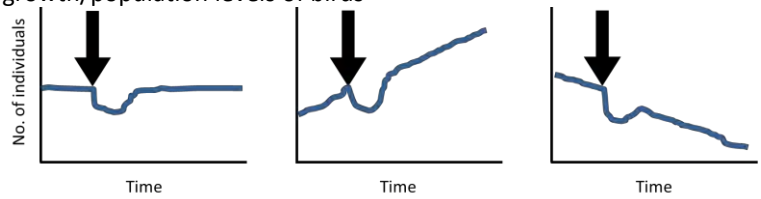
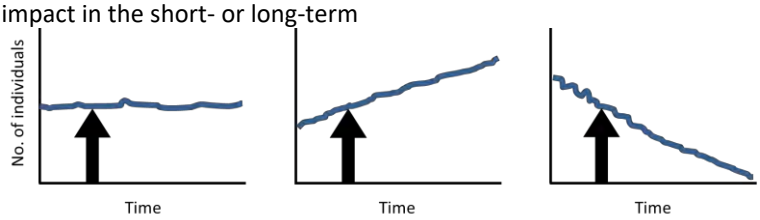
Overall, the hazard list (APPENDIX 2) identified 111 potential hazards to the over 300 bird species reported to use the PPBBP Ramsar wetland site (APPENDIX 1). A specific risk assessment for each hazard and each species was not feasible or necessary. One method of grouping disease hazards is according to their modes of transmission: inhalation (aerosol), ingestion (oral), direct contact, contact with a contaminated inanimate object (fomite) and contact with a contaminated or infected living organism (vector) (Thrusfield and Christley, 2018). In the context of an ecosystem and bearing these modes of transmission in mind, bird species can be grouped according to their habitat and feeding type on the assumption that these behaviours would render some birds more susceptible to some hazards than others. For instance, predatory birds might be more susceptible to ingested toxins that accumulate through the food chain, water-based birds more susceptible to diseases associated with degraded water quality (e.g. botulism) or high concentrations of birds (e.g. pasteurellosis) and land-based birds more susceptible to contact with hazards restricted to non-aquatic species (e.g. parrots exposed to psittacine beak and feather disease virus). Accordingly, the bird species list was divided into these three functional groups to facilitate prioritisation of hazards by small groups during the workshop:

1. Shore and water-based birds
2. Land-based birds (Psittacine, passerine and other ground- and land-dwelling birds)
3. Predators (raptors and piscivores)

It is important to note that all groups comprise migratory (either intra-Australian or internationally along the East Asian-Australasian or West Pacific flyway), nomadic and vagrant species as well as threatened species. This factor influences the exposure, release and consequence assessment for hazards.

Prior to review of the hazard list (APPENDIX 2), workshop participants came to consensus on the rationale to be used in the allocation of prioritization rankings of each hazard using their combined experience and expertise to assess the likelihood that birds would be exposed to each hazard through their interactions with the PPBBP Ramsar sites and the consequence should exposure occur (Table 3).

Table 3. Hazard prioritization criteria for wild birds at PPBBP Ramsar site

LIKELIHOOD OF EXPOSURE	**RATIONALE**
High (3)	Hazard is known or expected to occur/persist at this Ramsar wetland site.
Medium (2)	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 this Ramsar wetland site.
Low (1)	Hazard has been recorded in the geographic range of a species interacting with this Ramsar wetland site, but it is unlikely that the hazard would occur or become established at this Ramsar site.
Negligible (0)	Hazard has not been recorded in the geographic range of the species using this Ramsar site.
CONSEQUENCE TO POPULATION	**RATIONALE**
High (3)	- Long-term impact on bird populations, changes trajectory  - Exceeds defined limits of acceptable change for the Ramsar site in a sustained manner - Changes status listing criteria for bird species or Ramsar wetland site - Impacts multiple bird species - May lead to widespread population impacts offsite - Flow on impacts to ecosystem processes - Has a high impact on welfare – many animals/prolonged suffering
Medium (2)	- No long-term population impact on the expected population growth/population levels of birds  - Detected with some morbidity/mortality; localised – spatial, temporal or species - Does not affect multiple bird species - Medium welfare impact – smaller number of animals affected
Low (1)	- Some individuals are affected, but no discernible bird population impact in the short- or long-term  - Low impact on welfare
Negligible (0)	None of the above

Small groups of 7-8 workshop participants (APPENDIX 4) were allocated to one of the three functional bird groups to review and prioritize the hazard list (APPENDIX 2) against the agreed criteria (Table 3) recording their results in a matrix. The result are shown in Table 4, Table 5 and Table 6.

Table 4. Risk prioritization matrix for shore and water-based birds.

		Consequence to shore and water-based birds			
		High (3)	**Medium (2)**	**Low (1)**	**Negligible (0)**
Likelihood of exposure	**High (3)**	Botulism Mycobacteria Pasteurellosis	*Clostridium perfringens* *Escherichia albertii* Salmonella Algal toxins Medication residues Plastics Human disturbance	Avian paramyxovirus (APMV) Herpesvirus (endemic) Chlamydia *Escherichia coli* Erysipelas Staphylococcus and Streptococcus Yersiniosis Aspergillosis Coccidiosis Giardia	Arbovirus (endemic) Low pathogenic avian influenza (LPAI) Avian poxvirus Coronavirus Psittacine beak and feather disease (PBFD) Rotavirus Campylobacter Candidiasis Cryptococcosis *Macrorhabdus ornithogaster* Cryptosporidia Antimicrobial resistance (AMR)
	Medium (2)	Lead Organophosphates (OPs) Persistent organic pollutants (POPs) Highly pathogenic avian influenza (HPAI)	Quaranjavirus	Arbovirus (exotic) *Edwardsiella tarda*	
	Low (1)	Herpesvirus (exotic) Oil spill (large)		Adenovirus Proventricular disease	Avian polyomavirus Infectious bursal disease virus
	Negligible (0)				Borreliosis

Table 5. Risk prioritization matrix for land-based birds (psittacine, passerine and other ground- and land-dwelling birds).

		Consequence to land-based birds			
		High (3)	**Medium (2)**	**Low (1)**	**Negligible (0)**
Likelihood of Exposure	**High (3)**	Virulent avian paramyxovirus 1 (APMV-1) (pigeon)	PBFD Rotavirus (hepatic) Enteric bacteria Trichomoniasis Persistent organic pollutants (POPs)	Adenovirus Poxvirus Botulism Erysipelas Mycobacteria Pasteurellosis Aspergillosis Cryptococcosis Enteric protozoa Blood parasites Sarcocystis Helminths External parasites Heavy metals Pesticides Algal outbreaks	Low pathogenic avian influenza Avirulent APMV-1 Polyoma virus Coronavirus Staphylococcus and Streptococcus Candidiasis Toxoplasmosis
	Medium (2)	Disease X (newly emerging disease caused by a pathogen currently unknown)	Arboviruses Herpesvirus Chlamydia Spironucleosis Lorikeet paralysis syndrome Disease X	Yersinosis *Macrorhabdus ornithogaster* Black and white bird disease Disease X	Disease X
	Low (1)	Highly pathogenic avian influenza Disease X	Avian bornavirus Disease X	Infectious bursal disease virus (IBDV) Disease X	Disease X
	Negligible (0)				Cyanide

Table 6. Risk prioritization matrix for predators (raptors and piscivorous birds).

		Consequence to predatory birds			
		High (3)	**Medium (2)**	**Low (1)**	**Negligible (0)**
Likelihood of Exposure	**High (3)**	Botulism Pasteurellosis Heavy metals / pesticides & other bioaccumulative toxins	Enteric bacteria Yersinosis Anticoagulant rodenticides Trichomoniasis Intestinal helminths	Low pathogenic avian influenza (LPAI) Avian poxvirus Coronavirus Herpesvirus Psittacine beak and feather disease (PBFD) Rotavirus Erysipelas Mycobacteria Staphylococcus and Streptococcus Aspergillosis Candidiasis Enteric protozoa Haemosporidia Sarcocystis Toxoplasmosis	Murray Valley Encephalitis Virus (MVEV) Ross River virus Avirulent Avian paramyxovirus (APMV) Virulent APMV-1 (pigeon) Avian polyomavirus Spironucleosis
	Medium (2)	West Nile Virus (WNV) Highly pathogenic avian influenza (HPAI) Virulent APMV		Adenovirus Quaranjavirus Cryptococcosis	Infectious bursal disease *Macrorhabdus ornithogaster*
	Low (1)			Japanese Encephalitis Virus (JEV) Kunjin virus Avian bornavirus Chlamydia	Borreliosis
	Negligible (0)	Usutu virus			Bagaza virus

Brief rationales for the prioritisation by each group were recorded and are summarised in Table 7 below. Small groups presented their prioritisation of hazards back to the workshop plenum. Where necessary, clarification was sought, and categorisations were discussed.

Table 7. Comparison of hazard prioritisation rationales across the three functional bird groups (Rank: E = Exposure; C = Consequence; H = High; M = Medium; L = Low; N = Negligible).

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
VIRAL HAZARDS			
Adenovirus	EL, CL Not known in water birds, but some uncertainty due to a possible outbreak in long-tailed ducks in Alaska.	EH, CL Has been detected in birds within the region. Has been detected in association with mortalities, but role in mortalities was thought to be minimal.	EH, CL Known in species which occur in the area, known in captive birds in the area; can be asymptomatic, unlikely that multiple species would be impacted over a sustained period
Arboviruses	EM, CL Exotic arboviruses (WNV & Usutu): Could be introduced by migrating birds or vectors on aircraft/boat, lower susceptibility in water birds than land-based species (some uncertainty here). EH, CN Endemic arboviruses (MVE, RRV, Kunjin): Present with waterbird reservoirs, unlikely to cause more than mild disease in birds.	EM, CM Flaviviruses may not have been detected much lower than the Murray River. However, there have been some un-speciated flaviviruses detected further south. Ross River cases have been detected though. The vectors for arboviruses are present in the area in significant numbers. There have been significant outbreaks worldwide in wild birds due to arboviruses, pathogenicity is difficult to determine within Australia	EH, CN Murray Valley encephalitis virus (MVEV) – endemic in Victoria, no pathology known in birds EL, CL Kunjin virus – occurs in Australia, but not at this Ramsar site; not known to cause clinical disease in birds EM, CH West Nile Virus (WNV) – widely distributed through Africa/Asia/Middle East, cyclical EN, CH Usutu – not present in the geographic range, has caused mas mortalities overseas EN, CN Bagaza – not in the geographic range, not known in our species EH, CN

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
			RRV – present in the area, not known to cause disease in birds
Avian influenza	EM, CH Highly pathogenic avian influenza (HPAI): Highly infectious, could be introduced by internationally migratory birds from Asia or North America; high mortality seen in several events overseas. [*The plenum discussed whether consequence should be reduced to medium, but decided to keep it at high due to events overseas.*] EH, CN Low pathogenic avian influenza (LPA): Already present, has not caused mass mortalities in wild birds to date.	EL, CH HPAI does not occur at the site. EH, CN LPAI exists at the site, but does not cause mortalities.	EM, CH HPAI - occurs in similar sites, if established would have high consequences EH, CL LPAI - present, sometimes causes lesions
Avian paramyxovirus (APMV)	EH, CL APMV: Strains present, unlikely to cause significant mortalities in the species in our group.	EH, CH Virulent APMV strains Pigeon paramyxovirus is present in Victoria, but has not been formally detected at this Ramsar site. EH, CN Avirulent APMV strains	EH, CN Avirulent – occurs, no pathology for our species EM, CH Virulent – not found at this site, but occurs in similar ecosystems, high pathogenicity, mortalities in cormorants in North America EH, CN Pigeon paramyxovirus – known to occur in Victoria, no evidence of population impact on predatory bird species
Polyoma virus	EL, CN Reports only in captive birds, may be present.	EH, CN Present but unlikely to clinical disease	EH, CN Present, not known in these species

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
Poxvirus	EH, CN Present, usually self-limiting.	EH, CL Present, but low likelihood of disease	EH, CL Present, self-limiting disease
Coronavirus	EH, CN Present, but disease unlikely.	EH, CN Low levels of coronavirus in recent studies in passerines, but detected in waterfowl.	EH, CL Exposure is likely to be high as many coronaviruses are circulating, however no evidence of disease
Herpesvirus	EH, CL Endemic herpesvirus: Strains present, new strain introduction from elsewhere in Australia could cause mortality. EL, CH Exotic herpesvirus (DVE/Duck Plague): Novel strain so could have significant disease.	EM, CM Detected in numerous species in Victoria, including pigeons No evidence of herpesvirus disease in Victoria in passerines at Ramsar wetlands Exotic psittacine herpesvirus presents an introduction risk for endemic parrots, Psittacid herpesvirus-1 is included on the Australian priority list of exotic diseases	EH, CL Again, exposure likely to be high as many herpesviruses circulating, no evidence of disease
Infectious bursal disease virus (IBDV)	EL, CN Clinical disease not recorded in wild birds.	EL, CL Not recorded in wild birds, exposure in wild birds in other parts of the country (WA).	EM, CN Occurs in a wide range of sites around Australia, no evidence of disease
Proventricular dilatation disease (PDD)/ bornavirus	EL, CL Low concern for water-based birds.	EL, CM Not recorded in wild birds, but in captive birds. If it entered the country the consequences could be significant for some native species. Parrot bornavirus is included on the Australian priority list of exotic diseases.	EL, CL Most birds show no clinical signs.
Psittacine beak and feather disease (PBFD) circovirus	EH, CN Psittacine disease that has been detected in a wide range of species.	EH, CM This virus has only once in 10 years caused significant mortalities in nestlings of the endangered orange-bellied parrots (OBPs).	EH, CL Some reports of disease in raptors, laughing kookaburra, corvids, powerful owl. Present at the site.

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
		PBFD is listed as a Key Threatening Process for OBPs) Impact is focussed in breeding grounds, rather than this Ramsar site. Causes disease in columbids/pigeons, finches.	
Pigeon rotavirus	EH, CN Not important for shore and water-based bird species.	EH, CM Widespread. Pathogenicity depends on the form. Hepatic form may be quite pathogenic, but populations would likely recover.	EH, CL Is present everywhere, unlikely to be clinically significant
Quranjavirus	EM, CM Newly reported, unknown epidemiology. Caused mortality in ducks in SA.	Not prioritised.	EM, CL Clinical significance unknown.
BACTERIAL HAZARDS			
Borrelia	EN, CN Not present in Australia, not important for water birds	Not prioritised.	EL, CN Found in similar geographical places, but unlikely to became established, unlikely to be clinically significant
Botulism	EH, CH Endemic at the Ramsar site, caused mass mortalities previously. Site is a drought refuge due to tidal waters and water influx from the Western Treatment Plant, causes an increase in bird numbers in dry conditions, including animals stressed and already in poor condition. Some areas of the site are prone to stagnant water and conditions favouring bacteria growth in summer.	EH, CL Present at this site, land-based species are less susceptible and less likely to be exposed	EH, CH Present, some species known to be affected
Chlamydia	EH, CL	EM, CM	EL, CL

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
	Present, generally low level of disease in these bird species.	Disease is present, around 30% prevalence in the Brisbane ranges; at population level, outbreaks occur every now and again.	Present, unlikely to cause disease in these bird species group
Enteric bacteria (*Campylobacter* spp., *Clostridium* spp., *E. coli*, *Salmonella* spp.)	EH, CN Campylobacter: Present in sewage, but disease not known in wild waterbirds. EH, CM *C. perfringens*: Present; disease in Australia, but not in waterbirds; disease in waterbirds overseas. EH, CM *E. albertii*: Present; disease in Australia but not in waterbirds; disease in waterbirds overseas. EH, CL *E. coli*: Present in sewerage, low level of disease. EM, CL *Edwarsiella*: Assumed, but not known if present; low level of disease. EH, CM Salmonella: Present at high prevalence; high density of birds.	EH, CM All bacteria are present at site. Salmonella outbreaks in house sparrows and doves caused mortalities.	EH, EM Occur, can cause mortality events
Erysipelas	EH, CL Present, multiple species are susceptible; not common, but could cause mortality.	EH, CL Birds more likely to be reservoirs. Other livestock may cause exposure at Ramsar sites	EH, CL Occurs, incidental disease in birds
Mycobacteria / Avian tuberculosis	EH, CH Present in environment; possible chronic effects	EH, CL Likely to be present. Individual birds affected	EH, CL Present, likely an individual event

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
	with potential long-term population impacts.		
Pasteurellosis / Avian cholera	EH, CH Present at this site, significant disease effects.	EH, CL Likely to be present. Unlikely to cause significant disease in passerines and other land-based birds	EH, CH Occurs, has been associated with mortality events in predatory bird species, and mass mortalities have been seen.
Staphylococcus / *Streptococcus* spp.	EH, CL Opportunistic infections, with stress/competition.	EH, CN Likely to be present, individual birds	EH, CL Ubiquitous, individual impact
Yersinosis	EH, CL Present, low/medium consequences.	EM, CL Likely to be present, no mass mortalities in Australia	EH, CM Occurs, can cause sporadic disease and larger mortality events, but is unlikely to be sustained
FUNGAL HAZARDS			
Aspergillosis	EH, CL Present; individual cases.	EH, CL Likely to be present, likely to affect only small numbers	EH, CL Ubiquitous; unlikely to have a massive population impact
Candidiasis	EH, CN Present; opportunistic.	EH, CL Likely to be present, likely to affect only small numbers	EH, CL Ubiquitous; unlikely to be massive population impact,
Cryptococcosis	EH, CN Present; no disease in wild birds.	EH, CL River red gums are present at the site, low numbers affected	EM, CL Occurs in the region, can cause disease in individuals, has been seen causing disease in European vultures feeding of carcasses
Macrorhabdus ornithogaster / Avian gastric yeast (AGY)	EH, CN Present; no disease reported in waterbirds.	EM, CL Lots of birds carry AGY. Individual effect.	EM, CN Commonly found in species, not known to cause disease in these species
PARASITIC HAZARDS			
Enteric protozoa	EH, CN	EH, CL	EH, CL

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
(*Coccidia*, *Giardia*, *Cryptosporidia* spp.)	Cryptosporidia: Present in sewerage; disease can occur, but not common. EH, CL Giardia: Present in sewerage; clinical disease.	High exposure, individual effects.	Likely to occur, some individuals may show clinical signs
Blood parasites (*Haemoproteus*, *Leucocytozoon*, *Plasmodium* spp.)	Not of highest priority* *[*To expedite the hazard prioritisation given the long list of hazards and limited time available at the workshop, all infectious hazards hereafter were compared to hazards already rated as the highest priority (high likelihood of exposure & high consequence to water-based bird populations such as botulism, mycobacteria and pasteurellosis]*	EH, CL Flies, mosquitoes are present. Unlikely to cause significant disease in these species	EH, CL Haemosporidia are present, but clinical significance normally low
Sarcocystosis	Not of highest priority*	EH, CL Would be present but have a low effect on population.	EH, CL Likely to be present, but not known in wild birds in Australia, but known to cause pathology in some birds
Spironucleosis	Not of highest priority*	EM, CM Impact could be species dependent	EH, CN Likely to be present, not known in these species
Toxoplasmosis	Not of highest priority*	EM, CN Likely to be present, impact on species low	EH, CL Likely to be present, but only causes clinical signs in individuals, unlikely to have a population effect
Trichomoniasis	Not of highest priority*	EH, CM High likelihood and medium impact in columbids and passerines	EH, CM Likely to be present given the prevalence in feral pigeons, will cause some morbidity/mortality in raptors

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
Helminths	Not of highest priority*	EH, CL Present, but variable knowledge around the impact depending on the species. Mortalities in Orange-bellied parrots with ascarids have been reported, although on the whole, minimal impact on population	EH, CM Very likely to be present, unlikely to cause long-term changes on a population level
External parasites	Not of highest priority*	EH, CL Present, but low impact	Not prioritised
HAZARDS OF UNKNOWN ORIGIN			
Lorikeet paralysis	Not of highest priority*	EM, CM Occurs in clusters, specifically lorikeets	Not prioritised
Black and white bird syndrome	Not of highest priority*	EM, CL Mainly in NSW, limited to individual birds	Not prioritised
Disease X	Difficult to determine unknowns Not of highest priority*	EL-M, CN-H Hard to classify as it is unknown; potentially high consequences once it emerges, but high uncertainty	Not prioritised
NON-INFECCTIOUS HAZARDS			
Heavy metals	EM, CH Lead: Present (e.g. hunting and fishing), but not at all areas; long-term population impacts possible due to bioaccumulation	EH, CL Hunting allowed near Werribee and on Lake Connewarre. Definitely present, although unclear impact on these species. Not commonly documented to affect this bird group.	EH, CH Bioaccumulation along the food chain
Insecticides / herbicides and other pesticides	EM, CH Agriculture and water treatment nearby, thus exposure to organophosphates (OPs) and persistent organic pollutants (POPs) likely; possible long-term	EH, CL Pesticides exposure is high through the environment, but less so directly, e.g. 1080 (sodium fluoroacetate) exposure to this group is	EH, CH Bioaccumulation of pesticides and other toxins along the food chain EH, CM

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
	impacts from sublethal doses.	low compared with raptors Limited knowledge on exposure and effects in these species. EH, CM POPs – persistent in soil, therefore insects likely infected. Limited knowledge on exposure and effects in these species.	Anticoagulant rodenticides, localised mortalities upon exposure.
Other toxins (Algal toxins, cyanide, crude oil toxicosis)	EH, CM Algal toxins: Present; mass mortalities at the Western Treatment plant have been observed with microcystins present, but direct causation is still unproven EL, CH Oil spills: Some areas of the site (e.g. Mud Island, Swan Bay) are at risk for large spills, small spills occur regularly, effects are not well documented	EH, CL Algal toxins: Outbreaks of blue-green algae have occurred at this Ramsar site. Land-based species are unlikely to be affected though. EN, CN Cyanide: low effect	Not prioritised
Antimicrobial resistance	EH, CN Exposed, but no impact on birds, mainly a human health issue.	Only an impact if trying to treat the birds with antibiotics.	Not prioritised
Medication residues	EH, CM Present in sewerage; impact is difficult to assess, evidence of an impact on ecosystems is starting to build (oestrogens, antidepressants).	Not prioritised	Not prioritised
Plastic and rubbish	EH, CM Present in sewage, tidal waters, negative impact of microplastics on	Not prioritised	Not prioritised

Hazard	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
	individual, long-term impact on populations not specified.		

Selection of hazards for detailed risk assessment

Paired Ranking

Following completion of the risk prioritisation matrix for each of the bird groups, a paired ranking process was applied to further prioritize the highest-ranking hazards in each matrix (high likelihood of exposure and high consequences to populations). In this process each hazard is compared, in turn, to each other hazard in the list asking the question, which hazard is likely to have the greatest negative impact on the population of interest. As only one hazard was categorised as the highest priority for land-based birds, hazards ranked as the second highest priority (high likelihood of exposure and medium consequences) were included in the paired ranking process by this group. Two hazards, namely pigeon paramyxovirus 1 and psittacine beak and feather disease, were excluded from the paired ranking exercise as they are already managed under specific programs (Notifiable animal diseases in Victoria (Agriculture Victoria, 2020a); Threat abatement advice (DAWE, 2020b)).

The results of each group's paired ranking were collated for discussion by the whole group (Table 8).

Table 8. Highest-ranking hazards identified by each functional bird group.

Hazard rank	Group		
	Shore and water-based birds	Land-based birds	Raptors and piscivorous birds
1	Botulism	Enteric bacteria	Heavy metals / pesticides & other bioaccumulative toxins
2	Pasteurellosis	Persistent organic pollutants (POPs)	Botulism
3	Mycobacteria	Trichomonas	Pasteurellosis

Within the time available, it was agreed that only three hazards could be subjected to a detailed risk assessment during the workshop. A whole group discussion considered the criteria to be used for selection of these hazards from among those identified by the paired ranking exercise. The group came to the consensus that hazards that had been prioritised by multiple functional bird groups should carry a higher weight. It also emerged that contaminants were considered differently between groups. Some were assessing individual contaminants while others were using groupings. If viewed as one large group (including heavy metals, persistent organic pollutants and other residues), these hazards would have ranked higher in all functional bird groups. Thus, the three hazards selected for detailed risk assessment were:

- Botulism
- Pasteurellosis
- Pollutants and contaminants

Hazard Transmission Pathways and Critical Control Points (CCPs)

The final step in the Hazard Identification involved the creation of a graphical representation of the transmission pathways for each of the selected hazards. Participants were again grouped ensuring equal numbers, diverse areas of expertise and broad representation of stakeholders in each group.

Once constructed, all figures were scrutinised to identify critical control points, which are those positions on the pathway where mitigating actions could be taken to prevent or minimise the risk of transmission to birds. Results are shown in Figures 5-7 and corresponding CCPs are described in Table 9-11.

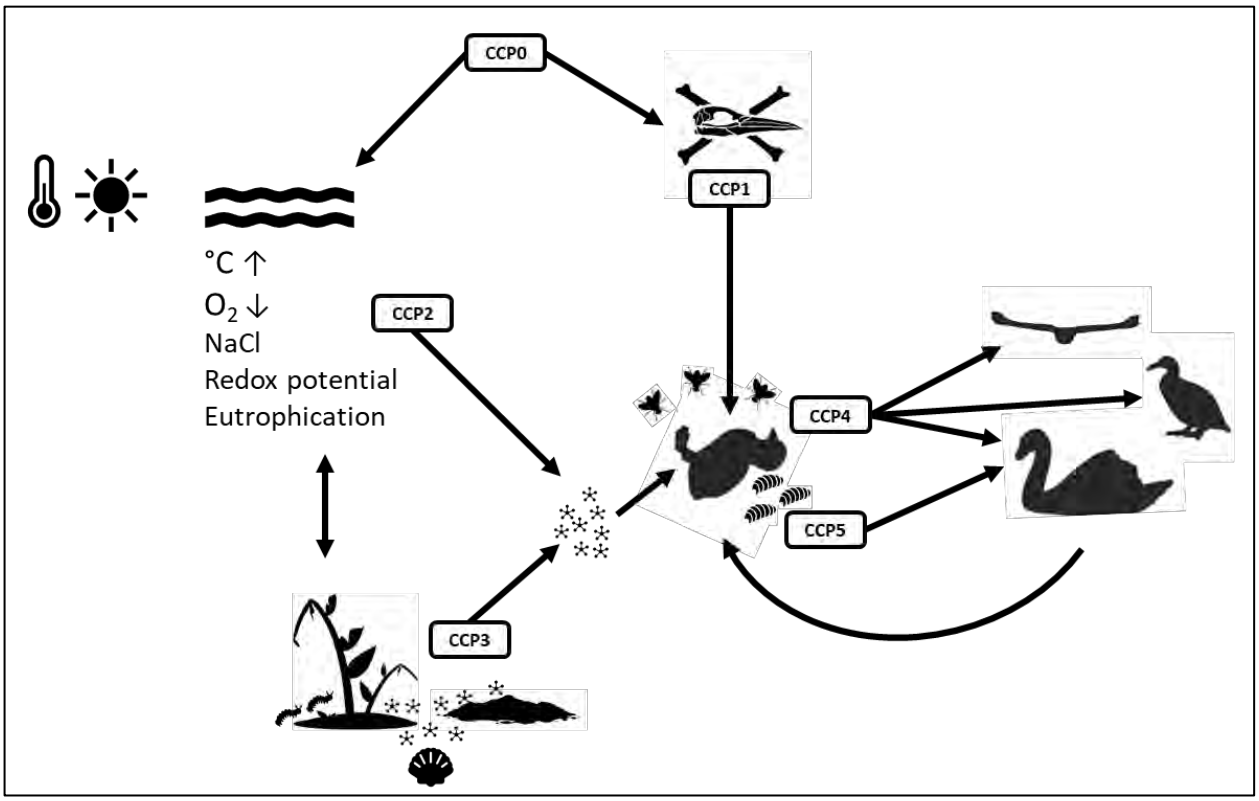


Figure 5. Hazard transmission pathways and critical control points (CCPs) for botulism.

Table 9. Critical control points (CCPs) in the hazard transmission pathways of botulism.

CCP#	CCP Target	Description
0	Prevention of outbreaks through monitoring and surveillance	Monitoring of environmental conditions that favour spore germination and growth of *Clostridium botulinum* and toxin production (e.g. weather, water, time of year) to increase prevention and preparedness. Early detection of potential causes of mortalities as well as rapid reporting of occurring bird mortalities through higher intensity of disease surveillance.
1	Mortalities in birds	Birds dying of other causes such as starvation, shooting, trauma, disease, old age, severe weather events or contaminants may trigger an outbreak by creating favourable conditions for bacterial replication and toxin production leading to accumulation of botulinum toxin within the carcass, invertebrates, and the environment.
2	Water conditions	High water temperature, low oxygenation, high pH level, low salinity, eutrophication and redox potential affect the spore germination and growth of *C. botulinum* as well as toxin production.
3	Vegetation, invertebrates and soil	Clostridia are bacteria commonly present in soil and mud that can produce resistant spores, which survive long-term. Dropping water levels leaving edges exposed or other factors may result in decaying vegetation or other organic matter (e.g. dead invertebrates), thereby providing favourable conditions for spore germination, bacterial growth and toxin production. Invertebrates and soil can become contaminated with toxin. Invertebrates may accumulate toxin.
4	Removal and appropriate disposal of dead birds.	Birds that have died of botulism act as a source of toxin to other birds either directly by predation or indirectly via contamination of the environment or toxin accumulating invertebrates.
5	Maggots	Maggots in carcasses can accumulate botulism toxins and may be consumed in high numbers by other birds, then leading to further mortalities. Further, maggots may play a role in cooler weather conditions by warming carcasses and thus increasing bacterial growth and toxin production. Removal of maggots or preventing conditions that are favourable for maggots would break this transmission cycle.

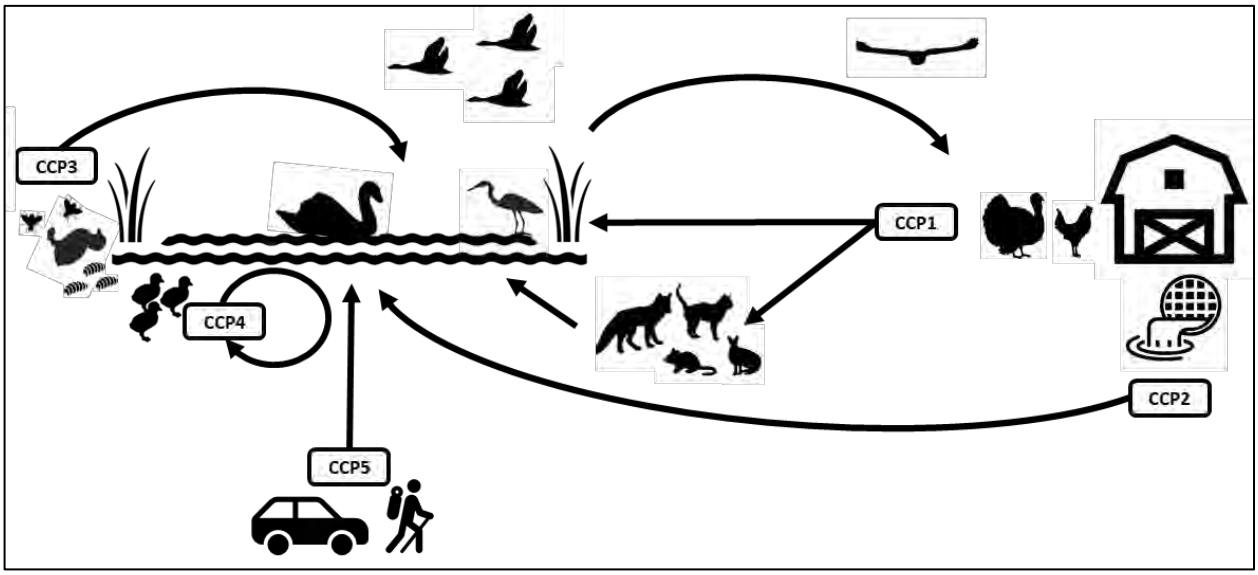


Figure 6. Hazard transmission pathways and critical control points (CCPs) for pasteurellosis.

Table 10. Critical control points (CCPs) in the hazard transmission pathway of pasteurellosis.

CCP#	CCP Target	Description
1	Contact between wild and farmed birds.	Bidirectional transmission of *Pasteurella multocida* between wild birds entering poultry farms, particularly free-range farms is possible unless direct and indirect contact between wild and farmed birds is minimised and/or poultry is vaccinated for pasteurellosis. Other wildlife and feral animals (e.g. cats, foxes, rodents, rabbits) may play a role in spreading pasteurellosis.
2	Contact between wild birds and poultry farm effluent	Wild birds can become infected by having contact with poultry effluent either on farm or from effluent run off into the wetlands.
3	Removal and appropriate disposal of dead birds.	Birds that have died of pasteurellosis act as a source to other birds either directly by predation or indirectly via contamination of the environment.
4	Infected wild birds	Infected wild birds contaminate the environment and water, so that healthy birds coming in contact may become infected leading to a transmission cycle within the wetlands.
5	Human and vehicle movement, fomites	Movement of contaminated water and soil may spread disease.

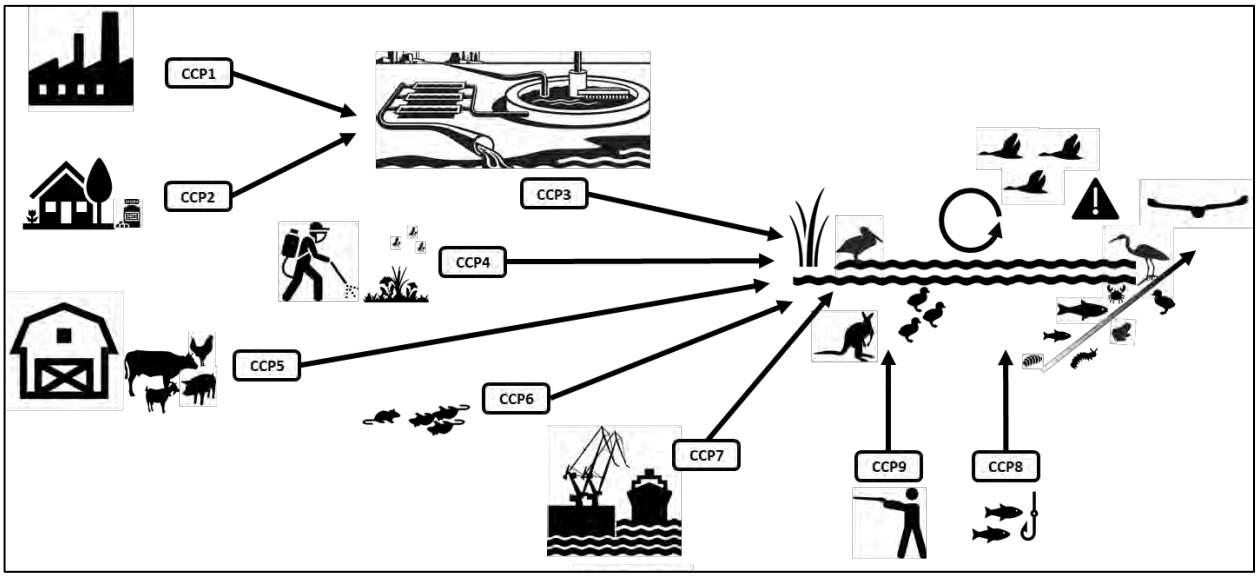


Figure 7. Hazard transmission pathways and critical control points (CCPs) for pollutants.

Table 11. Critical control points (CCPs) in the hazard transmission pathways of pollutants.

CCP#	CCP Target	Description
1	Use and discharge of chemicals policy	Chemical use in industrial and domestic settings are controlled by the Environment Protection Act 1970 and subordinate legislation.
2	Domestic use and disposal of chemicals and pharmaceuticals	Chemical and medications use in domestic situations allow these compounds to enter the sewage system and contaminate water.
3	Sewage from the Melbourne Water Western Treatment Plant	Contaminants enter the sewage system through domestic, agricultural and industrial use of chemicals and pharmaceuticals (e.g. persistent organic pollutants, fertilizers, per-and polyfluorinated alkyl substances, heavy metals, antibiotics, narcotics, hormones).
4	Domestic and industrial use and disposal of herbicides and pesticides	Herbicides and pesticides can contaminate the environment through run off and inappropriate disposal.
5	Agricultural chemicals use	Agricultural chemical use such as herbicides, pesticides fungicides and fertilizers can cause pollution of the environment through run off and effluent as well as direct bird contact with treated areas.
6	Rodent control and anticoagulant use	Rodent and other pest control relies heavily on anticoagulant use which can cause toxicity to birds either from direct ingestion of baits or secondary toxicity from ingestion of poisoned pest species.
7	Shipping and port industry	Ships and port industry discharge a range of pollutants (e.g. oil spills, antifouling chemicals).

CCP#	CCP Target	Description
8	Lead sinkers	Lead sinkers used for fishing cause contamination of waterways and marine ecosystems. These sinkers may be from ongoing use or from historical fishing activity.
9	Lead ammunition	Lead ammunition is used for duck and terrestrial hunting and causes contamination of the environment and carcasses.
⚠	Bioaccumulation and biomagnification	Many transmission pathways for this hazard involve biomagnification (up trophic levels) and bioaccumulation (at a trophic level) and can affect e.g. immune function, disease resistance, reproduction, having chronic and acute health impacts on wild birds.

RISK ASSESSMENTS

Based on the hazard prioritisation, detailed risk assessments were conducted for (1) botulism, (2) pasteurellosis and (3) pollutants and contaminants.

Risk assessment – Botulism

Workshop group participants: Lee Berger, Kate Brandis, Robert Bone, Keren Cox-Witton, Steven Liu, Will Steele, Anke Wiethoelter

Botulism is caused by a toxin produced by *Clostridium botulinum* and causes neurological disease in a range of species. Several bacterial strains producing different types of neurotoxins (A-G) have been identified. In particular, toxin types C and E are known to affect birds and fish (Rocke and Bollinger, 2007). Invertebrates such as maggots can act as accumulators from carcasses. *Clostridium botulinum* spores are resistant in the environment and common in soil and aquatic environments (Rocke and Bollinger, 2007, Soos and Wobeser, 2006). Avian botulism is present throughout the world and regularly causes bird mortality events (Le Marechal et al., 2016b). While all birds may be affected by it, waterfowl are the most predisposed. In Australia, botulism is seen mainly in Anseriformes and Pelecaniformes during the warmer months (Nov-April)(Grillo et al., 2013, Brandis et al., 2019).

Justification for hazard status (i.e. priority for this DRA)

Botulism was chosen for detailed risk assessment for the following reasons:

- Endemic disease known to have caused mass mortalities at the site (see Table 1)
- High likelihood of exposure due to persistent spores in the environment (Rocke and Bollinger, 2007)
- Multiple species affected (Le Marechal et al., 2016b, Grillo et al., 2013, Brandis et al., 2019)
- Assumption that population effects are based on extent of recorded mortalities (Rocke, 2006)
- Increased risk due to climate change influencing predisposing environmental and water changes (e.g. increased temperature, decreased oxygen) and parts of this Ramsar site being a critical drought refuge for birds due to constant water influx from the Western Treatment Plant

Release assessment (likelihood of hazard entering or being present in the sites of interest)

Already present:

- *Clostridium botulinum* found in sediment samples at Melbourne Water Western Treatment Plant (pers. comm. W. Steele 28/05/2020)
- Outbreak in silver gulls and terns at Mud Island in 2018 – diagnosed based on clinical signs and environmental conditions (pers. comm. Parks Victoria and Mark Hawes 22/01/2020).
- Large outbreak at the WTP in 2015-16 with over 3,000 reported mortalities affecting multiple species (e.g. shelduck, silver gull, pelican, sacred ibis, black swans) and botulism toxin isolated (Whiteley, 2020).

On this basis the release assessment is ranked as HIGH.

Exposure assessment (likelihood that, if present, populations of interest will be exposed to the hazard)

Hazard exposure is not continuous, it is affected by seasonal, environmental factors, and can follow other causes of mortality resulting in decaying carcases that provide additional sites for clostridial growth and toxin production (Anza et al., 2014, Soos and Wobeser, 2006, Vidal et al., 2013). The exposure is likely to increase due to climate change increasing predisposing environmental factors and, at the same time, leading to higher bird densities at the site due to its nature as a draught refuge for birds due to tidal zones and constant water influx from the Western Treatment Plant. Some areas of this Ramsar site (e.g. Point Cook and Swan Bay) are potentially of lower risk due to their intertidal ecology and the high salinity of the water.

Although all birds are susceptible to botulinum toxins, feeding ecology seems to be the main risk factor with omnivorous waterbirds feeding on eutrophic water ponds at highest risk (Rocke and Friend, 1999). Nevertheless, exposure risk also exists for predatory birds and to a lesser extent passerines and psittacines (Rocke, 2006).

On this basis the exposure assessment is ranked as HIGH.

Consequence assessment

This hazard is known to have caused mass mortalities and high welfare impacts (sick birds drowning slowly) at the site in the past. For example, more than 3,000 waterbird mortalities reported in 2015-16 were associated with this hazard (see Table 1). Botulism affects a wide range of species although the long-term effect on populations overseas and in Australia is currently poorly understood (Brandis et al., 2019, Rocke, 2006). Endangered species might not be able to withstand sporadic, but high losses, if their distribution and/or numbers are limited (Rocke, 2006), whereas common and widely distributed species are more likely to recover from mass mortality events.

On this basis the consequence assessment is ranked as HIGH, in particular for bird species already recognized as endangered (e.g. Blue-billed duck, Freckled duck).

Risk estimation

Based on the above and the risk matrix below (Figure 8) the overall risk of this hazard to wild birds at the PPBBP Ramsar site is ranked as **HIGH** and risk mitigation actions are recommended.

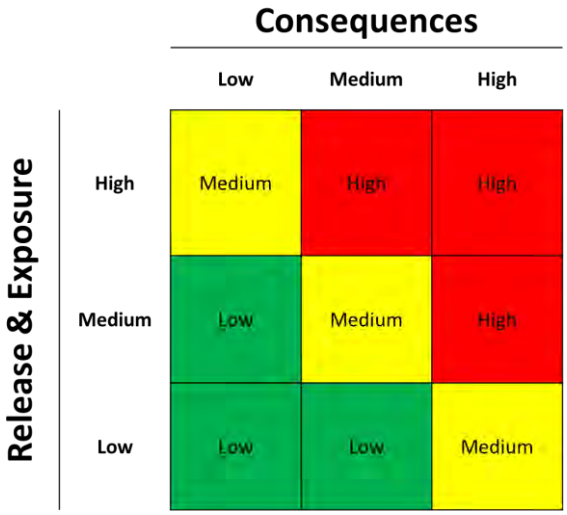


Figure 8. Risk assessment matrix combining release, exposure and consequence assessment.

Level of confidence in this risk estimation

There is high confidence in this risk estimation due to:

- Observation of large mortalities at this site and significant impacts reported overseas (Rocke and Friend, 1999, Rocke and Bollinger, 2007)
- Presence of vulnerable species at the site (e.g. Blue-billed duck, Musk duck, Freckled duck) where mass mortalities (~100-10,000 birds) could be significant
- Nature of the site as a critical drought refuge, potentially increasing bird densities
- Increasing risk associated with climate change exacerbating predisposing environmental factors.

However, there is still uncertainty in relation to long-term population level impacts of botulism in Australian waterbirds (Brandis et al., 2019).

Knowledge gaps

Table 12. Knowledge gaps and measures to reduce uncertainty in this risk assessment for botulism.

Knowledge gap	Measures needed to reduce uncertainty	Research priority
Specific environmental triggers in the Australian context and identification of high-risk areas	Disease ecology research	High
	Predictive model of predisposing environmental factors including climate change impacts; spatial risk map	High
Lack of good diagnostic tests	Development of better laboratory tests (reliable, accessible, timely, financially viable) and understanding of optimal sampling protocols	High
	Capacity for testing in local laboratories close to the site (currently only available in WA and QLD)	High
Optimal prevention and response strategies	Research and adaptive management including evaluation of interventions	High
Individual species susceptibility especially smaller ducks and waders	Surveillance and population monitoring before and during outbreak, focused on threatened or endangered species	Medium
Extent of *C. botulinum* (incl. spores) in environment and bird populations at site	Environmental monitoring for *C. botulinum* and targeted surveillance program in birds	Medium
Botulinum toxin types present in Australia	Research and accurate laboratory tests	Low

Risk management – Botulism

Predisposing factors

Environmental, agent and host factors that may predispose wild birds to botulism were reviewed (Table 13) as a basis for developing the hazard transmission pathway diagram (Figure 5) and identifying critical control points (Table 9). Consideration of these factors informed the identification and evaluation of risk mitigation options (Table 14).

Table 13. Environment, agent and host factors for botulism.

Environment factors influencing transmission	Agent factors influencing negative consequences to host	Host factors influencing susceptibility to disease
Water factors: temperature, water level, pH, redox potential, salinity, eutrophication	Persistence of spores in environment	Bird density
Invertebrate abundance	Toxin concentrated in maggots	Omnivorous and carnivorous diet
Causes of mortality e.g. food abundance, weather events, contaminants	Transmission via carcasses	Habitat preference
Season / air temperature	High pathogenicity	Species susceptibility
Drought		Summer breeding

Diagnosis, treatment, control and prevention

Diagnosis

Definitive diagnosis is difficult and often a presumptive diagnosis is made based on the species, clinical signs (progressive weakness, birds being unable to lift their necks, inability to fly, third eyelid protrusion, paralysis), ruling out other diseases on necropsy (where possible) as well as taking environmental factors such as current conditions and previous outbreaks into consideration. Non-infectious disease causes such as blue-green algae toxicity as well as other contaminants are common differential diagnoses.

Specimens and samples:

- Blood and tissues (e.g. liver) samples
- Full post-mortem
- Invertebrates (e.g. maggots) collected from dead birds or gut content of affected birds

Diagnostic tests:

Presence of *C. botulinum* in the gut alone does not provide a definitive diagnosis as the bacterium can be part of a healthy avian gut flora (Vidal et al., 2013, Le Marechal et al., 2016b). Detection of botulism toxin in invertebrates such as maggots on dead animals or in the gut contents of affected birds might indicate botulism (Rocke and Bollinger, 2007). However, a definite diagnosis requires detection of botulism toxin in serum or tissue samples. As the toxin binds to nerve cells, level of circulating toxin in the blood might be low, resulting in false negative test results (Rocke and Bollinger, 2007). High quality samples are required to ensure accurate test results.

- Capture ELISA (cELISA) detecting type C and D toxin, currently only available at laboratories in QLD and WA (Wildlife Health Australia, 2019)

- PCR for Type C/D (Le Marechal et al., 2016a), currently only available at a laboratory in WA (Wildlife Health Australia, 2019)

Treatment

Not practical at population level, but individual animals may recover with supportive treatment (e.g. fluids and feeding) over weeks.

Control

Control of botulism outbreaks is mainly focused on carcass collection and disposal as infected carcasses can increase environmental contamination with spores and thereby increase the number of contaminated vertebrates leading to further intoxications (Soos and Wobeser, 2006). Water management such as flushing with excess aerated water can further reduce bacterial growth and toxin production, but might not be feasible in drought conditions. Once an outbreak has been detected, moving birds off the affected area (through hazing etc.) may help to reduce exposure and the number of cases (as long as it is not a breeding ground).

Toxoid vaccination commercially available for mink has been trialled in ducks (Martinez and Wobeser, 1999, Rocke et al., 2000). It provides protection; however, it requires manual handling of individual birds (injection) and is therefore not feasible as a large-scale field application.

Prevention

- Water management – preventing stagnant warm water bodies, excess organic matter and warmer water temperatures may reduce the risk of a botulism outbreak but may not be possible in natural water bodies and coastlines. Reducing organic inputs into wetland.
- Artificial pond design – allow for appropriate water and vegetation management along the edges to prevent predisposing environmental factors for *C. botulinum* growth.
- Reduce other causes of mortality.
- Biosecurity – prevent soil spread from infected to uninfected sites by people and vehicles.

Risk management option evaluation

Based on the transmission pathways and CCPs identified for botulism (Figure 5 and Table 9), risk mitigation options were qualitatively assessed by workshop participants according to their likely feasibility and effectiveness (Table 14). Although several options have been recommended, each option should be assessed before implementation in terms of site variables, cost and feasibility. There may be different triggers and thresholds for some actions (e.g. number of bird mortalities or birds at risk).

Table 14. Risk management option evaluation for botulism to wild birds at the PPBBP Ramsar site.

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
0	Passive surveillance for sick and dead birds	High	Medium	Early detection of outbreaks to allow more effective response	Yes
	Water quality monitoring	High	Variable depending on area	Prediction of outbreak to allow prevention strategies	Yes
1 Bird mortalities	Ban shooting	Medium	Medium	There may be political hurdles. Mortality of birds is only one factor in the possible transmission pathways.	Yes
	Move powerlines, flight diverters on powerlines	Medium	Medium	Mortality of birds is only one factor in the possible transmission pathways.	Yes (site specific only for high risk areas)
	Disease surveillance Biosecurity	Medium	Medium	Surveillance and biosecurity reduce other hazards such as infectious disease	Yes
2 Water conditions	Flushing or draining of water – water levels	High	High	Depends on infrastructure and site.	Yes
	Circulation of water – aeration	High	High	Reduces other hazards such as algae and contaminants	Yes

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
	Intercept drains/filters, change adjacent land use, input flows, reducing plant material, reducing nutrient inputs	High	Low	Depends on infrastructure and site.	Yes
3 Vegetation, invertebrates, soil	Vegetation removal	High	High	Already current practice, however, is site dependant and expensive.	Yes
4 Dead birds	Carcass collection and disposal	High	Medium	Current practice where feasible, however labour intensive, difficult for some species and sites, resources not always available. Reduces other hazards by removal of infectious agents and contaminants.	Yes
	Collection of sick birds for treatment or euthanasia	Medium	Low	Is site dependant and labour intensive.	Yes
	Using hazing methods (actions aimed at dispersing birds or deterring them from entering an area) or physical exclusion from an affected area	Medium	Low	Hazing is labour intensive and birds become desensitised to it. Exclusion may be more feasible. Some methods e.g. shading could also have positive impact on water temperature (CCP2).	Yes (site specific, only for high risk areas)
5 Maggots	Carcass collection and disposal	High	Medium	As above Removes or reduces number of maggots present	Yes
	Fly control – e.g. insecticide spraying of dead carcasses	High	Low	Unknown – not trialled. Risk of contamination of environment.	No (more research needed)

*All mitigation techniques should use current methodologies and best practices

Implementation and review – Botulism

Risk management action plan

An action plan for recommendations arising from this risk management option evaluation for botulism was developed and is presented in Table 15.

Table 15. Risk management action plan for botulism to wild birds at the PPBBP Ramsar site.

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators of success
Disease surveillance CCP0	Development and implementation of a comprehensive general surveillance program to detect and collect sick and dead birds in Ramsar wetlands for early detection of a botulism outbreak	Pam Whiteley Lee Berger	University of Melbourne Parks Victoria Melbourne Water CMAs Agriculture Victoria Australian Centre for Disease Preparedness (ACDP) DELWP Zoos Victoria, Leanne Wicker, Healesville, Paul Eden, Werribee Wildlife Health Australia Community groups, citizen science reporting International partners (e.g. USGS National Wildlife Health Center,	Seek funds from DAWE in 2020 Implementation of the program by start of 2021.	- Funds secured - Surveillance program developed for Victorian Ramsar sites - Increased reporting of sick and dead birds - Investigation of disease events, diagnosis, identification of agents - Implementation of a systematic ongoing data collection process and reporting on pathogens and diseases at the Victorian Ramsar sites

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators of success
			Canadian Wildlife Health Cooperative)		
Monitoring of environmental conditions CCP0	Development and implementation of a monitoring program for environmental conditions to predict high risk periods for botulism.	Robert Bone with Ramsar coordinating committee	Parks Victoria Melbourne Water CCMA Port Philip CMA Wyndham City Council EPA	Implementation of the program by end of 2021.	- Monitoring program developed - Systematic ongoing data collection on environmental conditions relevant to botulism - Regular risk reporting
Water management CCP2	Water management to reduce environmental conditions that result in high risk of botulism including: flushing or draining of water (water levels); circulation of water (aeration); intercept drains/filters, change adjacent land use, input flows, reducing plant material or harvesting (reducing nutrient inputs). Trigger for this action includes environmental monitoring and disease surveillance.	Will Steele Robert Bone as liaison to Parks Victoria and other land managers	Disease ecologists Bird ecologists CCMA ARC Linkage researchers (for botulism and algae risk modelling)	2023 Upon completion of the ongoing projects around cyanobacteria and toxins with the Water and Eliza Hall Institute (WEHI) and the Nuisance and Harmful Algae Science-Practice Partnership (NHASP)	- Response plan developed - Water characteristics are maintained within the range considered to reduce risk of botulism - Long-term reduction in frequency and severity of botulism outbreaks
Collection of sick and dead birds CCP2 & 4	Collection of sick and dead birds during a botulism outbreak to remove them as a source of further botulism cases	Will Steele Robert Bone as liaison to DELWP and other land managers	Parks Victoria Melbourne Water CCMA Zoos Victoria University of Melbourne	Response plan developed by the end of 2020.	- Trigger point for collection of dead birds defined. - Response plan developed (if not already available) for collection of dead birds, and management of sick birds during an outbreak.

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators of success
					- Sick and dead birds collected according to the policy. - Long-term reduction in severity of botulism outbreaks.
Removal of vegetation CCP3	Reduction in growth of *C. botulinum* through removal of organic material such as vegetation from the water edges and decaying matter from the water.	Will Steele Robert Bone	Bird ecologist CCMA Universities DEWLP	End of 2021	- Response plan developed - Vegetation levels are maintained within the range considered to reduce risk of botulism - Long-term reduction in frequency and severity of botulism outbreaks

Research plan

In addition to the management action plan (Table 15), a research plan based on the knowledge gaps identified (Table 12) was developed and is presented in Table 16 below.

Table 16. Action plan addressing knowledge gaps for botulism to wild birds at the PPBBP Ramsar site.

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators
Understanding the specific environmental triggers for botulism in wetlands in the Australian risk areas and context.	Disease ecology research Predictive modelling including climate change impacts Spatial risk map	Based on knowledge gained from WEHI and NHASP projects currently underway Will Steele	MW WTP CCMA	3 years	- Robust predictive capacity for botulism outbreak

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators
More accurate diagnostic tests for botulism in wild birds	Development of better laboratory tests and better understanding of optimal sampling protocols for diagnosis of botulism. Development of capacity for botulism testing in local laboratories in Victoria.	Yonatan Segal Alistair Legione	Other universities Murdoch University laboratory CSIRO ACDP Mark Hawes, Christina McCowan AgriBio Melbourne Veterinary School, Prof Glenn Browning Australian Department of Defence	3-5 years	- Validated accurate diagnostic test available for botulism in a Victorian laboratory - Agreed optimal sampling protocols for diagnosis of botulism
Better understanding of optimal prevention and response strategies for botulism in wild birds	Research to develop and assess current and novel prevention and response strategies. To follow WEHI and NHASP research (see above)	Kate Brandis	Water managers CMAs Ramsar wetland managers State/federal governments Melbourne Veterinary School International partners (e.g. USGS NWHC and CWHC) Wildlife Health Australia	5 years	- Funding secured - Research completed - Recommendations for management based on research findings.

Risk assessment – Pasteurellosis

Workshop group participants: Jemma Bergfeld, Rebecca Gang, Alistair Legione, Rachel Pritchard, Yonatan Segal, Pam Whiteley

Pasteurellosis, also known as avian cholera or fowl cholera, is caused by the bacterium *Pasteurella multocida.* Different strains of *P. multocida* cause disease in many vertebrate species, including humans. Although classed as a zoonotic disease, avian pasteurellosis rarely causes human disease other than wound infections. Pasteurellosis is transmitted via close contact, inhalation or ingestion. The bacteria are shed in all bodily secretions, especially in faeces. Birds that recover are considered to have life-long infections and continue to shed (Botzler, 1991). An outbreak is most likely triggered via introduction of the pathogen through carrier birds or potentially other animals (Botzler, 1991, Samuel et al., 2007, Singh et al., 2014). Ongoing transmission is driven by environmental contamination of soil and water caused by contaminated carcasses (Blanchong et al., 2006).

Justification for hazard status

Avian cholera was chosen for detailed risk assessment for the following reasons:

- Endemic disease in poultry (chickens, ducks, turkeys) in Australia (Singh et al., 2013) and Victoria (Agriculture Victoria, 2020b), but has rarely been diagnosed in waterbirds in Australia, probably due to inadequate surveillance.
- Has been detected at Melbourne Water Western treatment plant and elsewhere in Victoria in a chestnut teal, a black swan and Eurasian coots. See Table 1 for details and APPENDIX 2.
- In North America and other countries *P. multocida* has been a major cause of mass mortality events in wild waterfowl for approximately 50 years (Friend, 1987, Samuel et al., 2007, Wang et al., 2009, Pedersen et al., 2003).
- Molecular diagnostics has linked *P. multocida* from wild birds to poultry (Whiteley, 2020).
- Based on the above, this disease was identified as a significant hazard in the hazard prioritisation step for shore and water-based birds and predators.

Release assessment

Possibly already present:

- *Pasteurella multocida* is present in poultry farms (Agriculture Victoria, 2020b), especially free-range farms, where wild birds (waterbirds, raptors, etc.) visit and then return to wetlands, making contamination of wetlands possible (Singh et al., 2014, Singh et al., 2013). The organism can also originate in wetlands and wild birds and be transferred to poultry farms (Christensen and Bisgaard, 2000, Samuel et al., 2004).
- There may also be a significant role for other species (e.g. predators/scavengers like foxes, cats, rodents) in the transmission of *P. multocida* between animals in both directions(Singh et al., 2014).
- Mortalities in Victoria appear to only occur sporadically, indicating that either the pathogen is only entering the system sporadically or that other factors are required to cause disease. There has been limited surveillance and testing for Pasteurellosis in dead birds at this Ramsar wetland site and in Victoria and Australia in general. Consequently, the low number of reported outbreaks might present a considerable underestimation of the disease prevalence.

On this basis the release assessment is ranked as MEDIUM.

Exposure assessment

There are multiple pathways and interfaces through which wild birds may be exposed to *P. multocida* (see Figure 6). This is an important disease in poultry (chickens, turkeys, ducks etc.), including free-range poultry production, in Australia.

Once in the transmission pathway *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 (Blanchong et al., 2006) and, as a result, birds may be persistently exposed (Samuel et al., 2007).

On this basis the exposure assessment is ranked as HIGH.

Consequence assessment

There is the potential for the bacteria to cause mass mortalities, particularly given the experience in North America and Asia (Samuel et al., 2007, Wang et al., 2009, Friend, 1987). However, it has only been identified sporadically in Australia in small numbers of birds, apart from being identified in conjunction with a suspected botulism mortality event at the Melbourne Water Western Treatment Plant in 2013 and as part of a mortality event at Lake Tooliorook (Lismore) in country Victoria (Whiteley, 2020). It is possible that strain differences in the bacteria, differences in waterbird densities or other unknown factors may contribute to the different epidemiological picture in Australia as compared with other countries in North America, Asia and Europe.

On this basis the consequence assessment is currently ranked as MEDIUM, however this may increase pending the results of more targeted surveillance for this organism.

Risk estimation

Based on the above and the risk matrix (Figure 8) the overall risk of pasteurellosis to wild birds at the PPBBP Ramsar site is ranked as **MEDIUM with the potential to move to high risk** (based on overseas experience) and risk mitigation actions are recommended.

Level of confidence in this risk estimation

There is low-medium confidence in the risk estimation given that there are significant knowledge gaps (see Table 17), particularly around the epidemiology of the disease and the gaps in surveillance of wild birds in the Australian context.

Knowledge gaps

Table 17. Knowledge gaps and measures to reduce uncertainty in this risk assessment for pasteurellosis.

Knowledge gap	Measures needed to reduce uncertainty	Research priority
Why do other countries have large outbreaks of disease compared with Australia?	Strain comparison of *P. multocida* isolates from wild birds and poultry within Australia and overseas using genomics, pathogenicity trials, etc.	High
	Density of birds and species	High

	Understand the role of predators and scavengers (cats, foxes, rodents, rabbits, raptors) in spread of *P. multocida* in Australia	High
How often do cases/outbreaks occur within Australia?	Surveillance within wild birds to determine prevalence	High
What is the prevalence of *P. multocida* in all types of poultry production?	Capture existing data from Agriculture Victoria	High
Is there persistence of *P. multocida* in the environment or within carrier birds without clinical disease?	Active surveillance and environmental sampling	Medium
Are there higher risk periods for infection in Australia (time of year, breeding, environmental conditions)?	Surveillance within wild birds to determine seasonality and correlations between environmental and species conditions.	Low

Risk management – Pasteurellosis

Predisposing factors

Environmental, agent and host factors that may predispose wild birds to pasteurellosis were reviewed (Table 18) as a basis for developing the hazard transmission pathway diagram (Figure 6) and identifying critical control points (Table 10). Consideration of these factors informed the identification and evaluation of risk mitigation options (Table 19).

Table 18. Environment, agent and host factors for pasteurellosis.

Environment factors influencing transmission	Agent factors influencing negative consequences to host	Host factors influencing susceptibility to disease
Density of scavengers and predators	Strains and potential variation in pathogenicity	Feeding behaviour (e.g. water feeding vs seed)
Environmental temperature, UV.	Infectious dose	Density of birds
Proximity of production animals	Persistence in the environment	Co-morbidities (infections and contaminants)
Wild bird access to poultry facilities and effluent	Characteristics of survival and transmission in different host species	Mobility of bird species (nomadic, migratory)
Human movement pathways (roads, paths)	Persistence in the environment	Immune status/stress
Climate change and associated increases of bird concentrations at this wetland site as some areas acts as a drought refugium (MW Western Treatment Plant, intertidal zones).	Persistence in the environment	Carrier status, could vary with species resistance to disease

Diagnosis, treatment, control and prevention

Diagnosis

Specimens and samples

- Whole carcasses
- Tissue samples, especially liver, intestine and heart – fresh and fixed
- Bone marrow from wings or other bones of decomposed carcasses
- Oropharyngeal swabs in live birds

Testing

- Bacterial culture
- PCR

Treatment

Not applicable in this context.

Control

During a confirmed or suspected outbreak, carcasses should be removed to avoid increasing environmental contamination. A variety of vaccines are available for poultry in Australia and overseas, ranging from commercially available live attenuated and live avirulent vaccines to autogenous vaccines (Glisson et al., 2003).

Vaccination has been used in the US in wild birds, however, this is not generally practical for large wild bird populations. Vaccination of poultry is widespread.

Prevention

Surveillance to identify outbreaks early in the disease course and to further describe the epidemiology in wild birds in Australia is recommended. Prompt carcass removal during a disease outbreak is recommended to reduce the accumulation of *Pasteurella multocida* in the environment. Dispersal of birds has been used in North America during large outbreaks (Botzler, 1991).

Risk management option evaluation

Based on the transmission pathways and CCPs identified for avian pasteurellosis (Figure 6 and Table 10), risk mitigation options were qualitatively assessed by workshop participants according to their likely feasibility and effectiveness (Table 19).

Table 19. Risk management option evaluation for pasteurellosis to wild birds at the PPBBP Ramsar site.

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
1 Interaction between wild animals & poultry farms	Fencing including overhead netting/fencing	High	Medium	Excluding wild birds and predator/scavenger access to poultry would reduce transmission. Limits to what you can implement in free range farms (cultural farming practices and settings).	Yes
	Vaccination of poultry	High	High	Vaccination already recommended. Increase awareness within poultry industry Would reduce poultry disease burden and potential transmission	Yes
	Scare tactics (e.g. lasers, dogs)	High	High	Scare tactics are already used and effective. May be cheaper than fencing.	Yes
	Keeping feed indoors	Medium	Medium	Food that attracts waterfowl may not be able to be moved indoors (grass) but other food should already be indoors. Stops scavengers (rodents, passerines etc).	Yes

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
				Farmers may feed outside to encourage poultry to be outside.	
	Responsible carcass disposal	High	High	Poultry carcass disposal stops contact of wild birds with dead poultry. Already occurring as per industry best practice.	Yes
	Best practice control of predators and scavengers including exclusion fencing	Medium	Medium	Baiting may have other considerations and consequences. May not be effective.	Yes
2 Effluent	Excluding wild birds from dams and effluent ponds	High	Medium	It may be difficult to remove dams or stop wild birds accessing them. Effluents ponds don't necessarily exist on free-range farms (sell manure as fertiliser). Water areas attract wild birds.	Yes
	Compost manure before use as fertiliser	High	Medium	Composting manure is effective in reducing bacteria which reduces wild bird contact with *P. multocida*.	Yes
3 Dead bird contamination of environment	Early detection of outbreaks by disease surveillance	High	High	To allow implementation of other mitigation strategies. Need funding for testing etc.	Yes
	Rapid collection and disposal of dead wild birds	High	Low	Collection of all carcasses is unrealistic due to landscape and water bodies as well as resources but would be effective.	Yes

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
				Removing carcasses as soon as possible would help and have cross over benefits for other diseases such as botulism.	
4 Infection of wild birds from other wild birds	Scare tactics	Low	Low	Scaring birds away from an outbreak may spread disease. May be useful if there was a spatial focus of outbreak. Site specific as to whether feasible.	No
	Drain water from site of outbreak	Medium	Low	Most natural sites cannot be drained. 3-4 sites can have water levels manipulated	No
5 Fomite transmission	Ongoing biosecurity (signage, communication, decontamination)	High	Medium	Many groups of people that would need to follow biosecurity.	Yes
	Early notification of outbreaks	Medium	High	Effective communication between organisations and people using the site would allow fast biosecurity interventions to be implemented.	Yes

*All mitigation techniques should use current methodologies and best practices

Implementation and review – Pasteurellosis

Risk management action plan

An action plan for recommendations arising from this risk management option evaluation for pasteurellosis was developed and is presented in Table 20.

Table 20. Management action plan for pasteurellosis to wild birds at the PPBBP Ramsar site.

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators
Vaccination of poultry, scare tactics and responsible carcass disposal	Many of these actions have already been implemented by Agriculture Victoria and the poultry industry as mitigation strategies for a number of other infectious disease and as part of good husbandry practices.	Yonatan Segal	Poultry industry	N/A	N/A
Rapid identification of outbreaks	To develop syndromic and disease surveillance by encouraging the rapid reporting and communication of morbidity and mortalities in birds by visitors or staff at wetlands and by developing reporting guidelines and communication tools for diagnosis, along with communication pathways for response options.	Yonatan Segal Lee Berger Pam Whiteley as Coordinator, Wildlife Health Victoria: Surveillance at the Melbourne Veterinary School	- Implementation - Mark Hawes as Wildlife Health Australia (WHA) Victoria Coordinator - Notification – Member of public, Melbourne Water - Initial Collection –MW, CCMA, Parks Vic, DELWP & others - Diagnosis – AgriBio (using Special Disease Investigation), University of Melbourne, Melbourne Veterinary School and Wildlife Health Victoria: Surveillance	2020/21 for funding and planning 2021/22 for implementation	- Guidelines are developed - Guidelines are widely available - All key players understand their role
Biosecurity during an outbreak	Prepare guidelines and communication tools for the application of biosecurity principles by wetlands users and managers to reduce the potential spread of *P. multocida* (and other	Robert Bone CCMA	- Melbourne Water - Agriculture Victoria	12 months	- Availability of guidelines

	infectious agents) through fomite transmission during an outbreak.		- Use of Wildlife Health Australia's emergency animal disease preparedness plans - Melbourne Veterinary School		

Research plan

In addition to the management action plan (Table 20), a research plan based on the knowledge gaps identified (Table 17) was developed and is presented in Table 21 below.

Table 21. Action plan addressing knowledge gaps for pasteurellosis to wild birds at the PPBBP Ramsar site.

Targeted knowledge gap	Description	Lead	Collaborators/ Partners	Timeline	Indicators
Why do other countries have large outbreaks of disease compared with Australia?	To secure funding to analyse the differences in epidemiology of *P. multocida* strains found in North America and Australia, including consideration of: A. Density of birds and other species B. The role of predators and scavengers (cats, foxes, rodents, rabbits, raptors) in the spread of *P. multocida* C. Pathogenicity comparison of different isolates	Pam Whiteley	- USGS National Wildlife Health Center - Canadian Wildlife Health Cooperative - DELWP - Parks Victoria - Agriculture Victoria	2 years	- Funding secured - Clarification of the epidemiology in Australia
How often and where do cases/outbreaks occur within Australia?	To increase surveillance of wild birds at wetlands for *P. multocida* in Victoria by encouraging increased diagnostic testing of all dead water birds over a period of 2 years. This will inform when, where and in what species the disease is occurring.	Pam Whiteley	Agriculture Victoria DELWP Parks Victoria Melbourne Water CCMA	3 years	- Funding obtained for the project - Development of submission guidelines - Increased submission of dead wild birds from

	Improved information about prevalence by increasing awareness between collaborating partners and providing submission guidelines.				wetlands to diagnostic laboratories - Better estimates of *P. multocida* prevalence and risk across wetlands.
What is the prevalence of *P. multocida* in all types of poultry production?	To collate reporting data to determine the prevalence of *P. multocida* in poultry and the potential risk of *P. multocida* to wild birds through spatial analysis.	Yonatan Segal Pam Whiteley	University of Melbourne Scolexia ACE Laboratory Services	12 months	- Map of *P. multocida* distribution and wetland bird distribution - Prevalence estimates over time

Risk assessment – Pollutants

Workshop group participants: Naomi Davis, Clare Death, Suelin Haynes, Elyse Herrald-Woods, Jasmin Hufschmid, Anna Meredith, Leanne Wicker

Pollutants encompass a wide variety of chemicals that may contaminate wetland environments from many different sources including water run-off, sewage and direct contamination of waterways and land environments. The broad groups that were considered in this risk assessment include:

- Heavy metals such as lead, mercury, cadmium and arsenic
- Pesticides including insecticides, herbicides and fungicides. This group also includes persistent organic pollutants (POPs) such as dieldrin, DDT, DDE, polychlorinated biphenyls (PCBs) and other dioxins
- Fertilisers, trace elements and other agricultural chemicals
- Per-and polyfluoroalkyl substances (PFAS)
- Pharmaceuticals
- Anticoagulants

Many of these chemical compounds are stable in the environment and cause persistent contamination leading to biomagnification and bioaccumulation through food chains.

Lead can cause acute and chronic toxicity of wild birds and has been responsible for mass mortality events in Victoria, other Australian states and other countries (Wickson et al., 1992, Sweet and Phalen, 2014).

Multiple classes of chemicals are used in the control of vertebrate, invertebrate, plant and fungal species deemed pests. Wild birds may be affected through direct poisoning during a pest control program, secondary toxicity from eating poisoned animals or plants or environmental contamination through the use, misuse and disposal of the chemicals (Grillo, 2011, Grillo et al., 2014a, Grillo and Post, 2010).

Organophosphates (OPs) can cause acute toxicity in vertebrates and invertebrates but degrade rapidly in the environment (Story and Cox, 2001, Grillo et al., 2014b). Carbamates are a similar class but are less commonly used. Toxicity with POPs is seen in all vertebrates and may have chronic health effects at low levels (McKenzie et al., 1982, Wildlife Health Australia, 2017b).

Anticoagulants are generally used for rodent control. First generation anticoagulants require multiple feeds to cause death and are not likely to cause disease when the carcass is eaten by another animal. Second generation toxins have a much more toxic impact and can cause secondary toxicity (Grillo, 2011).

Justification for hazard status

Pollutants were chosen for detailed risk assessment for the following reasons:

- High likelihood of contamination by a range of pollutants.
    - 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.
    - Proximity to Avalon Airport and Point Cook RAAF base as well as major freeways resulting in contamination of environment.
    - Port Phillip Bay and Corio Bay with associated port, industry and recreational uses are assumed to be contributing to pollutants.
    - The Western Treatment Plant processes wastewater that contains pollutants (Tjandraatmadja and Diaper, 2006).

- Presumed lead presence due to legacy and continuing sinker and ammunition pollution due to fishing and hunting activity at the site. A study of lead levels in wetlands in Victoria's Western District did not find elevated levels (Environment Protection Authority Victoria, 2017), however similar studies at the PPBBP site have not been conducted. Lead pollution secondary to hunting and fishing has been identified as a concern for human and animal health (Hampton et al., 2018).
- Two episodes of anticoagulant toxicity in birds of prey (Whiteley and Hawes, 2018) and one of organophosphate toxicity in sparrows (Whiteley, 2020) have been recorded in the nearby areas.

Release 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 (Tjandraatmadja and Diaper, 2006, DAWE, 2020a). Domestic pharmaceutical use and disposal has resulted in Victorian waterways being contaminated with medications (Richmond et al., 2018). Heavy metals have been shown to be present in Victorian wetlands (Sharley et al., 2017, Ficken and Byrne, 2013) and ammunition, sinkers as well as industrial run-off are known to cause environmental contamination with lead (Haig et al., 2014, Hampton et al., 2018).

On this basis the release assessment is ranked as HIGH.

Exposure assessment

Exposure to pollutants will vary temporally and spatially, affected by:

- Host factors: species (affects feeding ecology, trophic position, and other behaviours), age, sex, and reproductive status
- Compound factors: concentration, stability, bioavailability
- Environmental factors: origin of contamination (e.g. industry vs lead shot), source of exposure (e.g. stormwater vs deposit on vegetation), weather conditions, season

On this basis the exposure assessment is ranked as HIGH.

Consequence assessment

Consequences are likely to vary depending on species affected, life stage, level of concentration and type of pollutant. There is clear evidence for acute severe impacts for some compounds in individuals (e.g. lead (Haig et al., 2014, Whitehead and Tschirner, 1991, Degernes, 1995)) and anticoagulants (Whiteley and Hawes, 2018, Lohr and Davis, 2018). However, there may be significant chronic effects of lower level exposure at both individual and population level (Hunt, 2012, Degernes, 1995, Whiteley and Yuill, 1990, Ohlendorf et al., 1988). There are large knowledge gaps about the population level effects of chronic exposure to a large range of pollutants globally (Hunt, 2012).

On this basis the consequence assessment is ranked as HIGH.

Risk estimation

Based on the above and the risk matrix (Figure 8) the overall risk of pollutants to wild birds at the PPBBP Ramsar site is ranked as **HIGH** and risk mitigation actions are recommended.

Level of confidence in this risk estimation

Despite significant knowledge gaps (see Table 22), there was a high level of confidence in the risk estimation based on the knowledge available.

Knowledge gaps

Table 22. Knowledge gaps and measures to reduce uncertainty in this risk assessment for pollutants

Knowledge gap	Measures needed to reduce uncertainty	Research priority
Chronic health, ecological and behavioural effects of various pollutants and interactions between different pollutants within individuals as well as co-morbidities seen with exposure (including potential additive impacts of climate change)	- Systematic literature review - Surveillance of birds for concentrations of various pollutants - Investigations of health effects, including subclinical and chronic effects (e.g. behaviour, immune response, reproductive effects) - Better test methods to detect environmental levels, and clear guidelines on acceptable levels	High
Lack of knowledge of baseline concentrations in "normal" populations to assess role of pollutants in mortality or morbidity events	- Baseline surveillance data of bird populations - Investigation of apparently healthy birds for various pollutants - Environmental study to assess pollutant levels	High
The role of other pollutants (e.g. microplastics)	- Systematic literature review - Surveillance of birds for concentrations of various pollutants	Medium
Lack of knowledge of which sources are responsible for which pollutants and at which rates	- Systematic literature review - Environmental sampling to assess pollutant levels	Medium
Sources of antimicrobial resistance	- Surveillance of resistant genes and bacteria in different sources relevant to Ramsar wetlands - More relevant to public than wildlife health	Low

Risk management – Pollutants

Predisposing factors

Environmental, agent and host factors that may predispose wild birds to pollutants were reviewed (Table 23) as a basis for developing the hazard transmission pathway diagram (Figure 7) and identifying critical control points (Table 11). Consideration of these factors informed the identification and evaluation of risk mitigation options (Table 24).

Table 23. Environment, agent and host factors for pollutants and contaminants.

Environment factors influencing transmission	Agent factors influencing negative consequences to host	Host factors influencing susceptibility to disease
Weather, including rainfall, wind speed and direction for airborne pollutants	Type of contaminant	Species
Catchment drainage for water-based pollutants	Concentration / dose	Physiological or behavioural characteristics that increase or decrease risk (e.g. foraging or scavenging behaviour)
Season	Bioavailability	Reproductive status including eggs
Climate	Bioaccumulation	Age
Topography and hydrology	Biomagnification	Behaviour
Geographic location	Stability / persistence	Co-morbidities/immune status
Species assemblage (e.g. food sources)		Feeding ecology including trophic level
Vegetation		Distribution/home range
Anthropogenic activity (including land and water use, hunting activity, domestic use of chemicals, agricultural and industrial use of chemicals, compliance with legislation)		Migratory patterns

Diagnosis, treatment, control and prevention

Diagnosis

Environmental detection

- Water samples
- Soil samples
- Vegetation samples
- Sediment samples

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.

Treatment

Not practical at population level, but individual animals may be treated. For example, raptors with lead toxicity may be captured and treated with calcium EDTA and hospitalisation. Treatment varies with type of pollutant.

Control

- Active and passive surveillance of hosts and environment
- Trade waste controls for industry

Prevention

- Legislation for environmental health
- Treatment of water upstream of treatment plan

Risk management option evaluation

Based on the transmission pathways and CCPs identified for pollutants (Figure 7 and Table 11), risk mitigation options were qualitatively assessed by workshop participants according to their likely feasibility and effectiveness (Table 24).

Table 24. Risk management option evaluation for pollutants to wild birds at the PPBBP Ramsar site.

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
1	Improving and enforcing 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.	Yes
	Increase standards for on-site management of waste (e.g. decrease threshold levels)	High	Medium	Would require legislative change and therefore very strong political will and evidence base.	Yes
2	Better education on disposal of chemicals, medication etc in domestic households	Medium	High	Requires behaviour change, which can be difficult to affect through education alone. Programs would need appropriate reach to result in education and action.	Yes
	Ban domestic use of certain chemicals	High	Low/medium	Would require legislative change and therefore very strong political will and evidence base. There may be significant community and political opposition.	Yes
3	Increase capacity to keep up with rate of growth of Melbourne	High	High	Already happening – further communication/action not necessary	No
	Ensure current best practice and standards	High	High	Already happening – further communication/action not necessary	No

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
4	Educate public and industry on appropriate use and disposal of pesticides, herbicides and other contaminants	Medium	High	Pollutants would be reduced by appropriate disposal. Requires behaviour change, which can be difficult to affect through education alone. Programs would need appropriate reach to result in education and action.	Yes
	Integrated pest management that reduces use of high-risk chemicals	Medium	Medium	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.	Yes
	Ban domestic sale of high-risk chemicals	High	Low/Medium	Would require legislative change and therefore very strong political will and evidence base.	Yes
5	Ban and regulate use of certain agricultural chemicals	High	Medium?	Would require legislative change and therefore very strong political will and evidence base. Alternatives to currently used chemicals would need to be available and not present significant barriers (cost, availability, safety)	Yes
	Educate farmers on regulations and appropriate use depending on conditions	Medium	High	Requires behaviour change, which can be difficult to affect through education alone.	Yes
	Review use of agricultural/industrial chemicals in surrounding catchments that may affect Ramsar site	High	High	Understanding of local chemical use would enable more specific risk characterisation for the wetland sites by identifying the types of products used and disposed of.	Yes
6	Control use of anticoagulants	High	High		Yes
	Education on use of anticoagulants	Medium	High		Yes

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
	Educate public on non-chemical rodent control	Medium	High		Yes
7	Stop new port being built near Ramsar site	High	Low	Process is already under way so stopping existing plans would require very strong political will and evidence base. Environmental conditions required for ports are limiting	Yes
	Enforce and improve legislation on ballast water discharge/biofouling prevention chemicals	High	Medium	Limited resources for policing and logistically difficult to enforce. Contamination often only identified retrospectively	Yes
	Improve oil spill prevention and response	High	High	There are already extensive arrangements in place, however, these can be reviewed. Not an urgent action as current arrangements are strong.	No
8	Education of anglers on use of existing sinkers	Medium	High	Can't force people to listen to education; but very possible through various agencies	Yes
	Ban sale of lead sinkers and shift to alternative sinkers	High	Medium	Changing practice can be very difficult Cost may be prohibitive Uncertainty about details of alternative options and supply. Concern that alternatives may also contaminate environment. Implementation requires strong evidence base to guide legislative change and political will.	Yes
	Removal of existing/legacy sinkers	High	Low	Access to sinkers is difficult as they are likely to be widely distributed.	No

CCP#	Mitigation options*	Effectiveness	Feasibility	Explanation	Recommendation (Y/N)
				Disturbance of sediment during removal may cause further environmental and ecological problems.	
9	Ban lead ammunition for terrestrial hunting and enforce compliance with current legislation around duck hunting	High	Medium/High	Alternatives to lead shot already exist, however, they are more expensive. There is existing public awareness around hunting practices and problems with lead ammunition. May be politically unpalatable.	Yes
	Ban duck hunting at Ramsar site	High	Medium/high	May be politically unpalatable.	Yes
	Educate hunters in carcass removal and on impacts of using lead ammunition	Medium	High	Requires behaviour change, which can be difficult to affect. Would require communication with hunting groups.	Yes
	Removal of historical lead ammunition in environment	High	Low	Logistically unfeasible	No
Overall prevention	Active and passive surveillance of birds outside of and during mortality/morbidity events, using most up to date methodology	High	High	Surveillance would provide information about pollutant background levels, identify pollutants present in bird species, as well as identifying disease events correlated to elevated levels.	Yes

*All mitigation techniques should use current methodologies and best practices

Implementation and review – Pollutants

Risk management action plan

An action plan for recommendations arising from this risk management option evaluation for pollutants was developed and is presented in Table 25.

Table 25. Management action plan for pollutants to wild birds at the PPBBP Ramsar site.

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators of success
Disease detection and response preparedness plan	Developing a surveillance system for monitoring syndromes and mortality events to detect pollutants and the emergence of disease in birds in the Ramsar site. This plan will include the practical aspects of how morbidity/mortality events are detected over the sites, sample and carcass collection, submission, and funding. A clear outline of diagnostic inclusions (e.g. molecular diagnostics, whole genome sequencing) and allowances for the investigation of novel pathogen significance for bird health and populations will be included.	University of Melbourne Veterinary School - Lee Berger, Alistair Legione, Jasmin Hufschmid, Pam Whitely, Anke Wiethoelter Zoos Victoria - Leanne Wicker	WHA - Keren Cox-Witton ACDP - Jemma Bergfeld Department of Agriculture, Water and Environment - Elyse Herrald-Woods Agriculture Victoria - Mark Hawes EPA	Plan submitted for funding by end 2020, surveillance ongoing once plan is designed and funding approved	Preparedness plan for investigation of the emergence of an unexpected disease, including triggers for investigation, sampling/submission plans, type of investigations to be undertaken, and proposal to fund the investigation of unexpected disease events is submitted to Department of Agriculture Environmental Biosecurity Project Fund or other appropriate funding source.

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators of success
	The plan will outline how land managers should respond to disease events in the site.				
Educate hunters in carcass removal and on impacts of using lead ammunition	An education campaign and materials are designed to target duck and terrestrial hunters on the potential health and environmental impacts of using lead ammunition and the importance of carcass removal. This information could be incorporated into gun licencing, social media campaigns, and sporting shooters association of Australia (SSAA).	University of Melbourne Veterinary School - Jasmin Hufschmid (post-doctoral research on impacts of lead)	Communications department from Parks Victoria Game management authority Bird watching community Animal welfare organisations	12 months following completion of impact review (from University of Melbourne post-doctoral research)	- Materials for education campaign designed - Materials distributed and available
Better education on disposal of chemicals, medication etc in domestic households	Materials developed for school groups visiting Melbourne Water Western treatment plant and digital materials for Melbourne Water website and social media channels with the aim of reducing inappropriate disposal of chemicals into the sewerage system. This will link into the existing Sustainability Victoria	Melbourne Water - Suelin Haynes	EPA CMA Sustainability Victoria www.returnmed.com.au	Materials are completed by June 2021	- Materials incorporated into Melbourne Water Western treatment plant education program - Materials have been shared on MW social media channels and website

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators of success
	program 'Detox your home: Household chemical disposal program' and will include appropriate disposal of used medicines.				
Review use of agricultural/industrial chemicals in surrounding catchments that may affect Ramsar site	Desk based and survey of local stakeholders, agriculture industry and other industry to develop an understanding of the pollutants and practices (with a potential impact on bird health in the Ramsar site) used in the surrounding catchment	CCMA - Robert Bone	Melbourne Water Parks Victoria	6 months to complete	- Report is completed - Guidance provided on which pollutants are likely to be commonly found in the Ramsar site and a priority for surveillance (in both birds and environment).
Investigate pollutants as a contributor to death in mortality events	Investigate pollutants as a contributor to death in mortality events	University of Melbourne Veterinary School - Jasmin Hufschmid	EPA	12 months to completion following completion of impact review (from University of Melbourne post-doctoral research)	- Materials for education campaign designed - Materials distributed and available

Research plan

In addition to the management action plan (Table 25), a research plan based on the knowledge gaps identified (Table 22) was developed and is presented in Table 26 below.

Table 26. Action plan addressing knowledge gaps for pollutants to wild birds at the PPBBP Ramsar site.

Action target	Description	Lead	Collaborators/ Partners	Timeline	Indicators of success
Current lack of a baseline understanding of the type and level of pollutants in the environment and in birds, and the impact of these pollutants on bird health and populations	Undertake a study to understand the background/baseline presence/level and impact of pollutants in birds and the environment at the Ramsar sites to inform ongoing management recommendations for ecosystem monitoring. A project team collaborates on the design and funding of a project which will provide baseline information on which pollutants are present in the birds at the Ramsar site, and at what levels these occur. The health and population level impacts of significant pollutants on birds is investigated including literature reviews to identify global and local knowledge gaps on health and population impacts.	The University of Melbourne Veterinary School - Jasmin Hufschmid	CCMA RMIT University EPA Melbourne Water Parks Victoria DELWP USGS National Wildlife Health Center Canadian Wildlife Health Cooperative	Funding secured mid 2021 Study is completed within 3 years of funding	- Study designed and funded - Baseline information on pollutants in birds and environment is obtained - Ongoing monitoring program is designed.
Lack of information as to whether lead due to fishing is a significant contributor to environmental lead levels, and therefore contributing to population effects and bird health issues.	Use a community survey of anglers and local suppliers to understand current practices within the local angling community, gauge the importance of this activity, its potential contribution to environmental lead levels and quantify the use of lead in angling in the Ramsar site.	The University of Melbourne Veterinary School - Jasmin Hufschmid	Parks Victoria	12 months following completion of impact review (from University of Melbourne post-doctoral research)	

RECOMMENDATIONS AND CONCLUSIONS

The timeline below (Figure 9) summarises the recommended management action and research plans for botulism, pasteurellosis and pollutants.

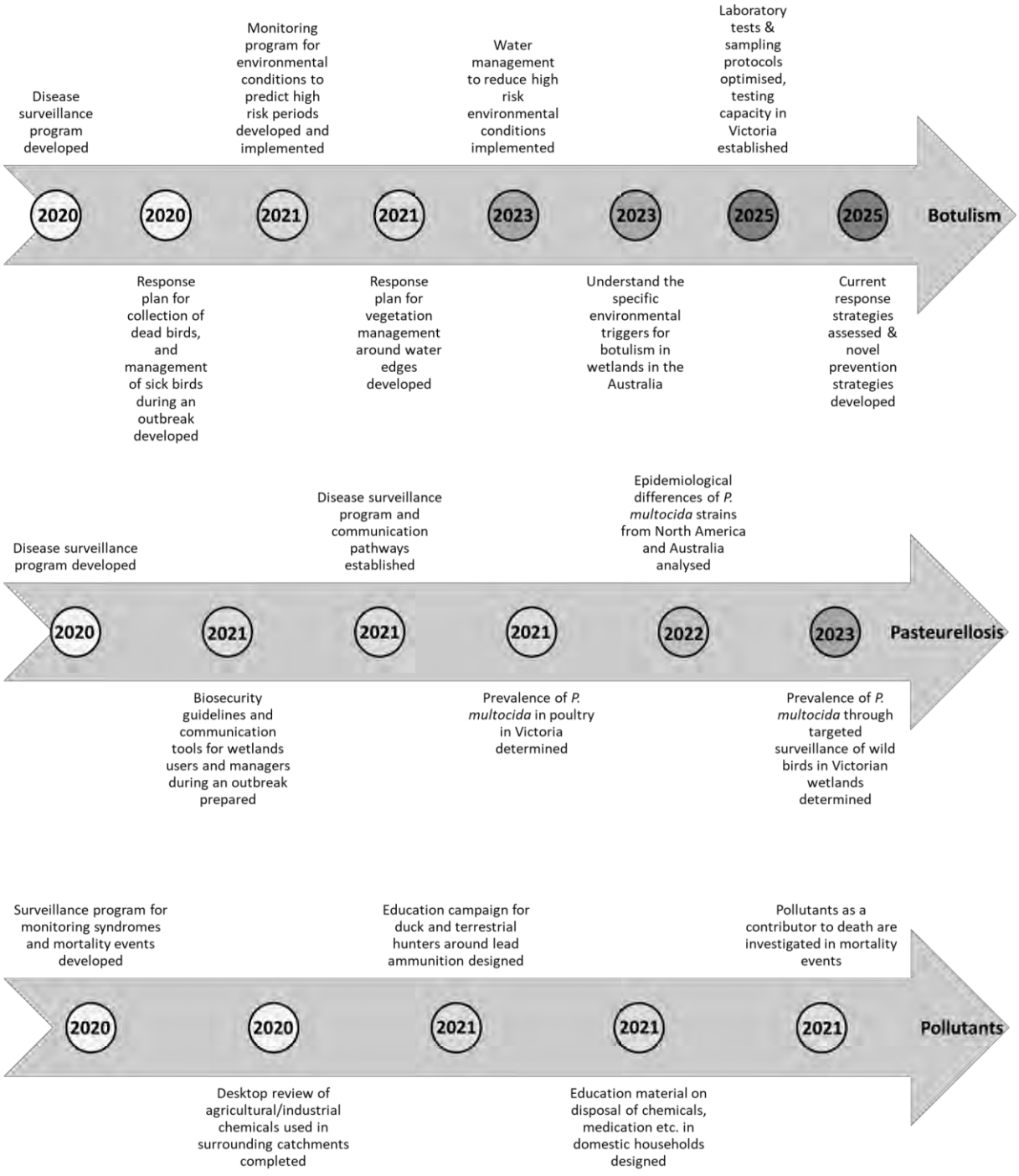


Figure 9. Timelines for management action and research plans for botulism, pasteurellosis and pollutants.

Some recurrent themes became evident and as some management actions address several diseases at once, those should be prioritised (Table 27).

Combined priority risk management action plan

Table 27. Priority management action plan for wild birds at the PPBBP Ramsar site.

Action target	Disease targets	Description	Lead	Collaborators/ Partners	Timeline	Indicators
Disease surveillance program for early detection and response	All causes of mortality and morbidity including but not limited to botulism, pasteurellosis, pollutants	Development and implementation of a comprehensive surveillance program to monitor syndromes, diseases and mortality events for early diagnoses and response as well as providing wildlife health baseline data. Rapid reporting of morbidity and mortalities in birds by visitors (citizen science) or staff at wetlands will be encouraged by developing reporting guidelines and communication pathways for response. Program will detail the practical aspects such as locations, sample and carcass collection, submission, and diagnostic techniques as well as include a preparedness and response plan and required training.	University of Melbourne Veterinary School - Lee Berger, Jasmin Hufschmid, Pam Whiteley, Anke Wiethoelter	- Parks Victoria - Melbourne Water - CMAs - Agriculture Victoria - Yonatan Segal; Agribio - Mark Hawes - DELWP - University of Melbourne – Pathology - ACDP - Jemma Bergfeld - WHA - Keren Cox-Witton - Zoos Victoria - Leanne Wicker Healesville Sanctuary, Paul Eden Werribee Zoo - DAWE - Elyse Herrald-Woods - EPA	Proposal for funding drafted and submitted by mid-2020. Implementation of the program by start of 2021.	- Funds secured - Collaboration with international partners established - Surveillance program (=systematic ongoing data collection on pathogens and diseases) designed and implemented - Reporting guidelines are developed and available; key players understand their role - Preparedness plan for investigation of an emerging disease is developed, including triggers for investigation, sampling/ submission plans, and type of investigations to be undertaken - Increased reporting of sick and dead birds - Investigation of disease events

Action target	Disease targets	Description	Lead	Collaborators/ Partners	Timeline	Indicators
				- Community groups, citizen science reporting - International partners (e.g. USGS National Wildlife Health Center, Canadian Wildlife Health Cooperative)		
Collection of sick and dead birds	Botulism, pasteurellosis, lead toxicity, other infectious diseases	Collection of sick and dead birds during disease outbreaks to remove them as a source for further cases. Inform duck and terrestrial hunters of the importance of carcass removal when using lead ammunition. Submission of suitable samples (fresh dead) for diagnosis, feeding into the surveillance program described above. Improve animal welfare.	William Steele, Suelin Haynes Robert Bone Jasmin Hufschmid (hunters & lead)	- Parks Victoria - Melbourne Water - DELWP - CMA - Zoos Victoria - Bird watching community - University of Melbourne - Animal welfare organisations - Game management authority	Response plan developed by the end of 2020.	- Response plan developed (if not already available) for collection of dead birds, and management of sick birds during an outbreak - Definition of trigger point for collection of sick and dead birds - Sick and dead birds collected according to the policy - Long-term reduction in severity of disease outbreaks - Materials for hunter education campaign designed and distributed
Biosecurity during an outbreak	Pasteurellosis, botulism and any mortality/ morbidity event	To prepare guidelines and communication tools on the awareness and application of biosecurity principles by wetlands users and managers to reduce the potential spread of infectious	Robert Bone	- Melbourne Water - Agriculture Victoria - University of Melbourne	Mid-2021	- Guidelines developed and available

Action target	Disease targets	Description	Lead	Collaborators/ Partners	Timeline	Indicators
		agents through fomite transmission during an outbreak. Based on WHA's emergency animal disease preparedness plans and AUSVETPLAN		- WHA		
Education campaigns	Pollutants, lead toxicity	Education campaign and materials are designed to target users of potential pollutants such as duck and terrestrial hunters (lead ammunition), domestic households (disposal of chemicals, medication), agricultural industry (chemicals used)	The University of Melbourne - Jasmin Hufschmid Melbourne Water - Suelin Haynes	- Communications department from Parks Victoria - Bird watching community - Animal welfare organisations - Sporting shooters association of Australia - EPA - CMA - Sustainability Victoria - returnmed.com.au	2021/22	- Materials for education campaigns designed - Materials distributed and available, where applicable incorporated into ongoing education programs (WTP, gun licencing), shared on social media channels and appropriate website

Combined research plans

Like the management actions, the research plans addressing knowledge gaps also revealed similarities across botulism, pasteurellosis and pollutants. The following aspects of diseases in wild birds are in general poorly understood and warrant further research:

- baseline data on prevalence,
- ecological factors and environmental triggers in the Australian context, including risk maps,
- causality and interaction of host, pathogen and environmental factors,
- long-term effects on populations,
- available, accurate diagnostic tests,
- assessment of response strategies and move towards novel preparedness and prevention strategies.

International collaborations with long-standing institutions that have decades of experience in these research areas such as the USGS National Wildlife Health Center and Canadian Wildlife Health Cooperative could help to rapidly build research capacity in Australia, enhance the understanding of Australian disease ecology and foster wildlife and ecosystem health in Australia. This could provide a strategic, efficient, and wise method of bridging existing knowledge gaps as key approaches and learnings from North America could be transferred to Australia and then validated in the Australian setting, rather than starting from scratch and repeating research.

Disease has been widely recognised as an important cause of wildlife population declines (McCallum, 2012). An accepted approach for identifying and minimising the negative effects of disease in wildlife populations is collection of data through surveillance, i.e. the ongoing systematic collection and analysis of health information and the timely dissemination of that information so that appropriate action can be taken (Salman, 2003). This includes, for example, monitoring the frequency of disease events and prompt implementation of management and mitigation strategies when the frequency exceeds expected levels. The 2012 Ramsar Wetland Disease Manual states that disease prevention and control need to be integrated into wetland management plans. The detection of new emerging diseases, robust risk assessments, and effective disease control in and around wetlands all rely on data generated through effective disease surveillance and monitoring (Cromie et al., 2012).

The planned disease surveillance program will fulfil these demands. It will provide required baseline data and allow early diagnosis and detection of disease as well as rapid responses. In the long-term, the program will contribute to closing the knowledge gaps and thereby support a shift in disease management from reaction to prevention. Australia is still at the beginning of a significant learning curve on how to improve wildlife health management. However, this DRA with a wide range of stakeholders is a shining example of progress.

RISK COMMUNICATION

As shown in Figure 1, effective communication with all relevant stakeholders is central to the success of a wildlife DRA. During the DRA workshop, participants began the process by identifying relevant stakeholders and potential communication challenges.

Stakeholders and stakeholder groups relevant to this DRA

The following authorities, institutions, organisations, industry and user groups were identified to either be involved in managing aspects of this Ramsar site or have an interest in the health of wild birds at this site.

Table 28. Stakeholders relevant to the outcomes of the DRA for birds at Ramsar wetlands in PPBBP.

Category	Stakeholder
Ramsar site management authorities	Avalon Airport
	Central Coastal Board
	Catchment Management Authorities (Corangamite CMA, Port Phillip & Westernport CMA)
	Department of Defence
	Environment Protection Authority Victoria
	Local councils (Borough of Queenscliff, City of Greater Geelong, City of Wyndham)
	Melbourne Water
	Parks Victoria
	Victorian Department of Environment, Land, Water and Planning
	Victoria Fisheries Authority
Other government	Agriculture Victoria
	Department of Agriculture, Water and the Environment
	Game Management Authority
	Members of Parliament
	MW WTP Biodiversity Conservation Advisory group
	Ramsar Secretariat
	Victorian Department of Health and Human Services
Communities	Field naturalists
	Friends of groups (e.g. Mud Island, Point Cook)
	Indigenous communities
	Local communities
	Local veterinarians
	Wildlife carer network
Users	Anglers and hunters
	Birdlife Australia

	Bird watchers
	Victorian National Parks Association
	Victorian Waders Study Group
Surrounding industry	Commercial companies that use the bay such as fisheries, tourism operators, shipping companies
	Farmers (Victorian farmers' federation, Australian Chicken Grower's Council, Australian Chicken Meat Federation, Egg Farmers of Australia)
	Geelong Port
	Local industries such as refineries
	Port of Melbourne Authority
Other institutions	Australian Centre for Disease Preparedness (formerly AAHL)
	Commonwealth Scientific and Industrial Research Organisation
	Countries in migration flyways
	Universities
	Wildlife Health Australia
	Zoos Victoria

Communication challenges

The following challenges around effective communication and potential areas of resistance were identified at the workshop:

- Shared management of this site adds complexity and can lead to a lack of clarity around roles and responsibilities, lack of coordination and collaboration, lack of regulations, legislation and enforcement as well as mixed messages
- Money, time and other resources available
- Difficulties around exerting authority and influence without resources
- High diversity of stakeholders with different reputations, vested interests, level of education and understanding needs to be taken into account when addressing knowledge gaps and fears
- Impacts of COVID-19

A range of actions involving communications with a wide range of stakeholders was identified in the Implementation and Review stage of the DRA process. Excluding the complications associated with the Covid-19 pandemic, the challenges identified are common to all multi-stakeholder collaborations. These will need to be kept under consideration during the development of the communications plan to ensure it effectively supports the needs of – and encourages contributions from - the new and established alliances that will be needed to realise the benefits to the wetlands, birds and other stakeholders identified by workshop participants.

Communications etiquette

As noted by Jakob-Hoff et al. (2014), once relevant experts and stakeholders have been identified, a communications plan should be **jointly** developed and include what information these individuals can supply, what information they need in order to play their part. The frequency and form of communication delivery

should be specified. Importantly, "*Communication etiquette should include appropriate acknowledgement of contributors and sources of information and respect of issues of confidentiality and intellectual property. … Where individuals from different disciplines or cultures are involved the use of technical terms should be avoided wherever possible. Where such terms must be used for clarity their meaning should also be explained in non-technical language*".

Further plan development and implementation

The responsibility for leading the development of a detailed communication plan was assigned to Robert Bone during the Implementation and Review stage of the DRA workshop. A draft of this final report has been circulated to all workshop participants for their consideration and input. Upon finalisation and in consultation with the funding organisations Corangamite Catchment Management Authority and Melbourne Water this report will be made publicly available through websites and circulated to selected stakeholders. It is also envisaged that this report will be added to the IUCN-SSC Conservation Planning Specialist Group document library (https://www.cpsg.org/document-repository) to serve as a blueprint for other disease risk analyses at ecosystem level.

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APPENDICES

APPENDIX 1. SPECIES LIST

The list below details bird species recorded at this Ramsar site, their conservation status in Victoria and Australia, whether the species contributes to the Ramsar wetland criteria, their migratory status, their abundancy at the Melbourne Water Western Treatment Plant (WTP) and their local habitat preference. Species are ordered according to taxon based on (Christidis and Boles, 2008). The table was compiled from bird species data provided by Melbourne Water, Corangamite Catchment Authority, BirdLife Australia, the Victorian Wader Study group, Maarten Hulzebosch, Richard Loyn, Fred Smith, Falk Wicker, the Information Sheet on Ramsar Wetlands (RIS) – Port Phillip Bay (Western Shoreline) and Bellarine Peninsula, and the Port Phillip Bay (Western Shoreline) and Bellarine Peninsula Ramsar Management plan (DELWP, 2018).

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
LAND-BASED BIRDS (Psittacine, passerine and other ground- and land-dwelling birds)						
Stubble Quail	*Coturnix pectoralis*				Common	O
Brown Quail	*Synoicus ypsilophora*				Uncommon	F
Rock Dove*	*Columba livia*				Common	U
Spotted Turtle-Dove*	*Streptopelia chinensis*				Common	U
Common Bronzewing	*Phaps chalcoptera*				Rare	F

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Brush Bronzewing	*Phaps elegans*				Rare	F
Crested Pigeon	*Ocyphaps lophotes*				Common	O
Diamond Dove	*Geopelia cuneata*	Near threatened (VIC)			Rare	F
Peaceful Dove	*Geopelia placida*				Rare	F
White-throated Needletail	*Hirundapus caudacutus*	Vulnerable (VIC)		Migratory	Uncommon	F
Fork-tailed Swift	*Apus pacificus*			Migratory	Uncommon	O
Plains-wanderer	*Pedionomus torquatus*	Critically endangered (VIC); Vulnerable (Aus)			Rare	O
Painted Button-quail	*Turnix varius*				Rare	F
Little Button-quail	*Turnix velox*	Near threatened (VIC)			Rare	O
Yellow-tailed Black-Cockatoo	*Zanda funereus*				Rare	F
Gang-gang cockatoo	*Callocephalon fimbriatum*				Rare	F

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Galah	*Eolophus roseicapillus*				Common	O
Long-billed Corella	*Cacatua tenuirostris*				Uncommon	O
Little Corella	*Cacatua sanguinea*				Uncommon	O
Sulphur-crested Cockatoo	*Cacatua galerita*				Uncommon	O
Cockatiel	*Nymphicus hollandicus*				Rare	O
Rainbow Lorikeet	*Trichoglossus moluccanus*				Common	UF
Scaly-breasted Lorikeet	*Trichoglossus chlorolepidotus*				Rare	F
Musk Lorikeet	*Glossopsitta concinna*				Uncommon	F
Little Lorikeet	*Glossopsitta pusilla*				Uncommon	F
Purple-crowned Lorikeet	*Glossopsitta porphyrocephala*				Uncommon	F
Australian King-Parrot	*Alisterus scapularis scapularis*				Rare	F

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Crimson Rosella	*Platycercus elegans elegans*				Uncommon	F
Eastern Rosella	*Platycercus eximius*				Uncommon	O
Australian Ringneck	*Barnardius zonarius*				Rare	F
Swift Parrot	*Lathamus discolor*	Critically Endangered (Aus)		Intra-Australian migratory	Rare	F
Red-rumped Parrot	*Psephotus haematonotus*				Common	O
Budgerigar	*Melopsittacus undulatus*				Rare	F
Blue-winged Parrot	*Neophema chrysostoma*				Uncommon	S
Elegant Parrot	*Neophema elegans*	Vulnerable (VIC)			Rare	S
Orange-bellied Parrot	*Neophema chrysogaster*	Critically endangered (VIC and Aus)	Supports a critically endangered species; >1% of the population	Intra-Australian migratory	Uncommon	S

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Eastern Ground Parrot	*Pezoporus wallicus*	Vulnerable (VIC)				S
Horsfield's Bronze-Cuckoo	*Chalcites basalis*			Migratory	Common	F
Black-eared Cuckoo	*Chalcites osculans*	Near threatened (VIC)		Migratory	Rare	F
Shining Bronze-Cuckoo	*Chalcites lucidus*			Migratory	Uncommon	F
Pallid Cuckoo	*Heteroscenes pallidus*			Migratory	Rare	F
Fan-tailed Cuckoo	*Cacomantis flabelliformis*			Migratory	Uncommon	F
Brush Cuckoo	*Cacomantis variolosus*			Migratory	Rare	F
Rainbow Bee-eater	*Merops ornatus*				Uncommon	F
Dollarbird	*Eurystomus orientalis*			Migratory	Rare	F
White-throated Treecreeper	*Corombates leucophaeus*				Rare	F

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Brown Treecreeper (south-eastern)	*Climacteris picumnus*	Near threatened (VIC)			Rare	F
Superb Fairy-wren	*Malurus cyaneus*				Common	FS
White-browed Scrubwren	*Sericornis frontalis*				Common	FS
Striated Fieldwren	*Calamanthus fuliginosus*				Common	S
Speckled Warbler	*Pyrrholaemus sagittatus*	Vulnerable (VIC)			Rare	F
Weebill	*Smicrornis brevirostris*				Rare	F
White-throated Gerygone	*Gerygone olivacea*				Rare	F
Striated Thornbill	*Acanthiza lineata*				Rare	F
Yellow Thornbill	*Acanthiza nana*				Rare	F
Yellow-rumped Thornbill	*Acanthiza chrysorrhoa*				Common	O

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Buff-rumped Thornbill	*Acanthiza reguloides*				Rare	F
Brown Thornbill	*Acanthiza pusilla*				Common	FS
Southern Whiteface	*Aphelocephala leucopsis*				Rare	O
Spotted Pardalote	*Pardalotus punctatus*				Uncommon	F
Striated Pardalote	*Pardalotus striatus*				Uncommon	F
Eastern Spinebill	*Acanthorynchus tenuirostris*				Uncommon	F
Yellow-faced Honeyeater	*Caligavis chrysops*				Uncommon	F
Singing Honeyeater	*Gavicalis virescens*				Uncommon	O
White-eared Honeyeater	*Nesoptilus leucotis*				Rare	F
Yellow-tufted Honeyeater	*Lichenostomus melanops*				Rare	F
Fuscous Honeyeater	*Ptilotula fusca*				Rare	F

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
White-plumed Honeyeater	*Ptilotula penicillatus*				Common	F
White-fronted Honeyeater	*Purnella albifrons*				Rare	F
Noisy Miner	*Manorina melanocephala*				Uncommon	O
Spiny-cheeked Honeyeater	*Acanthagenys rufogularis*				Uncommon	F
Little Wattlebird	*Anthochaera chrysoptera*				Uncommon	F
Red Wattlebird	*Anthochaera carunculata*				Common	F
Orange Chat	*Epthianura aurifrons*				Rare	S
White-fronted Chat	*Epthianura albifrons*				Common	SO
Tawny-crowned Honeyeater	*Glyciphila melanops*				Rare	F
Crescent Honeyeater	*Phylidonyris pyrrhopterus*				Rare	F

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
New Holland Honeyeater	*Phylidonyris novaehollandiae*				Common	F
Brown-headed Honeyeater	*Melithreptus brevirostris*				Rare	F
White-naped Honeyeater	*Melithreptus lunatus*				Uncommon	F
Varied Sittella	*Daphoenositta chrysoptera*				Rare	F
Black-faced Cuckoo-shrike	*Coracina novaehollandiae*				Uncommon	F
White-bellied Cuckoo-shrike	*Coracina papuensis*				Rare	F
White-winged Triller	*Lalage tricolor*				Uncommon	F
Crested Shrike-tit	*Falcunculus frontatus*				Uncommon	F
Golden Whistler	*Pachycephala pectoralis*				Uncommon	F
Rufous Whistler	*Pachycephala rufiventris*				Uncommon	F

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Grey Shrike-thrush	*Colluricincla harmonica*				Uncommon	F
Olive-backed Oriole	*Oriolus sagittatus*			Intra-Australian migratory	Uncommon	F
White-breasted Woodswallow	*Artamus leucorynchus*				Rare	F
Masked Woodswallow	*Artamus personatus*				Rare	F
White-browed Woodswallow	*Artamus superciliosus*				Rare	F
Dusky Woodswallow	*Artamus cyanopterus*				Uncommon	F
Grey Butcherbird	*Cracticus torquatus*				Rare	O
Pied Butcherbird	*Cracticus nigrogularis*				Rare	F
Australian Magpie	*Gymnorhina tibicen*				Common	O
Pied Currawong	*Strepera graculina*				Rare	F
Grey Currawong	*Strepera versicolor*				Rare	F
Rufous Fantail	*Rhipidura rufifrons*			Migratory	Rare	F

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Grey Fantail	*Rhipidura fuliginosa*				Uncommon	FS
Willie Wagtail	*Rhipidura leucophrys*				Common	O
Australian Raven	*Corvus coronoides*				Rare	F
Forest Raven	*Corvus tasmanicus*				Rare	F
Little Raven	*Corvus mellori*				Common	O
House Crow*[1]	*Corvus splendens*			Vagrant	Rare	T
Leaden Flycatcher	*Myiagra rubecula*			Migratory	Rare	F
Satin Flycatcher	*Myiagra cyanoleuca*			Migratory	Rare	F
Restless Flycatcher	*Myiagra inquieta*				Rare	F
Magpie-lark	*Grallina cyanoleuca*				Common	O
White-winged Chough	*Corcorax melanorhamphos*				Rare	F
Jacky Winter	*Microeca fascinans*				Uncommon	F
Scarlet Robin	*Petroica multicolor boodang*				Rare	F
Red-capped Robin	*Petroica goodenovii*				Rare	F

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Flame Robin	*Petroica phoenicea*				Common	F
Rose Robin	*Petroica rosea*				Rare	F
Pink Robin	*Petroica rodinogaster*				Rare	F
Eastern Yellow Robin	*Eopsaltria australis*				Rare	F
Horsfield's Bushlark	*Mirafra javanica*				Uncommon	O
Eurasian Skylark*	*Alauda arvensis*				Common	O
Golden-headed Cisticola	*Cisticola exilis*				Common	OS
Australian Reed-warbler	*Acrocephalus australis*				Common	W
Little Grassbird	*Megalurus gramineus*				Common	WS
Rufous Songlark	*Cincloramphus mathewsi*				Uncommon	F
Brown Songlark	*Cincloramphus cruralis*				Rare	O
Silvereye	*Zosterops lateralis*				Common	FS

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White-backed Swallow	*Cheramoeca leucosterna*				Rare	O
Barn Swallow	*Hirundo rustica*			Migratory	Rare	O
Welcome Swallow	*Hirundo neoxena*				Common	OW
Fairy Martin	*Petrochelidon ariel*			Intra-Australian migratory	Common	OW
Tree Martin	*Petrochelidon nigricans*			Intra-Australian migratory	Uncommon	FW
Bassian Thrush	*Zoothera lunulata*				Rare	F
Common Blackbird*	*Turdus merula*				Common	UF
Song Thrush	*Turdus philomelos*				Uncommon	U
Common Starling*	*Sturnus vulgaris*				Common	OU
Common Myna*	*Acridotheres tristis*				Common	UO
Mistletoebird	*Dicaeum hirundinaceum*				Uncommon	F
Zebra Finch	*Taeniopygia guttata*				Uncommon	O

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Red-browed Finch	*Neochmia temporalis*				Common	F
Diamond Firetail	*Stagonopleura guttata*	Near threatened (VIC)			Rare	F
House Sparrow*	*Passer domesticus*				Common	UOS
Eurasian Tree Sparrow*	*Passer montanus*				Uncommon	U
Australasian Pipit	*Anthus novaeseelandiae australis*				Common	O
Eastern Yellow Wagtail	*Motacilla tschutschensis*				Rare	O
Common Greenfinch*	*Chloris chloris*				Common	OU
European Goldfinch*	*Carduelis carduelis*				Common	OSU
PREDATORS (raptors and piscivores)						
Tawny Frogmouth	*Podargus strigoides*				Uncommon	F
Australian Owlet-nightjar	*Aegotheles cristatus*				Rare	F

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Wilson's Storm-Petrel	*Oceanites oceanicus*			Migratory		M
White-faced Storm-Petrel	*Pelagodroma marina*	Vulnerable (VIC)	Supports critical life stage of breeding (~1,000 nests on Mud Island)	Migratory	Rare	M
Black-browed Albatross	*Thalassarche melanophris*			Migratory		M
Shy Albatross	*Thalassarche cauta*	Vulnerable (VIC and Aus)		Migratory		M
Southern Giant-Petrel	*Macronectes giganteus*	Vulnerable (VIC); Endangered (Aus)		Migratory	Rare	M
Northern Giant-Petrel	*Macronectes halli*	Vulnerable (VIC); Vulnerable (Aus)		Migratory	Rare	M
Fairy Prion (southern)	*Pachyptila turtur*	Vulnerable (VIC and Aus)			Rare	M
Sooty Shearwater	*Ardenna grisea*			Migratory		M
Short-tailed Shearwater	*Ardenna tenuirostris*			Migratory	Rare	M

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Fluttering Shearwater	*Puffinus gavia*			Migratory	Uncommon	M
Hutton's Shearwater	*Puffinus huttoni*			Migratory	Rare	M
Common Diving-Petrel	*Pelecanoides urinatrix*	Near threatened (VIC)			Rare	M
Little Penguin	*Eudyptula minor*				Uncommon	BM
Lesser Frigatebird	*Fregata ariel ariel*				Rare	M
Cape Gannet	*Morus capensis*			Vagrant		B
Australasian Gannet	*Morus serrator*				Uncommon	B
Brown Booby	*Sula leucogaster plotus*				Rare	B
Australasian Darter	*Anhinga novaehollandiae*				Rare	W
Little Pied Cormorant	*Microcarbo melanoleucos*				Uncommon	WB
Great Cormorant	*Phalacrocorax carbo*				Uncommon	WB

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Little Black Cormorant	*Phalacrocorax sulcirostris*				Common	WB
Pied Cormorant	*Phalacrocorax varius*	Near threatened (VIC)	Supports critical life stage of breeding (WTP colony)	Common	WB	Pied Cormorant
Black-faced Cormorant	*Phalacrocorax fuscescens*	Near threatened (VIC)			Rare	B
Australian Pelican	*Pelecanus conspicillatus*				Common	WB
Australasian Bittern	*Botaurus poiciloptilus*	Endangered (VIC and Aus)	Supports an endangered species	Intra-Australian migratory	Rare	W
Australian Little Bittern	*Ixobrychus dubius*	Endangered (VIC)			Rare	W
White-necked Heron	*Ardea pacifica*				Rare	W
Eastern Great Egret	*Ardea alba modesta*	Vulnerable (VIC)			Rare	WT
Intermediate Egret	*Ardea intermedia plumifera*	Endangered (VIC)			Rare	W

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Eastern Cattle Egret	*Bubulcus ibis coromanda*				Rare	OW
White-faced Heron	*Egretta novaehollandiae*				Uncommon	WT
Little Egret	*Egretta garzetta nigripes*	Endangered (VIC)			Rare	WT
Nankeen Night-Heron	*Nycticorax caledonicus*	Near threatened (VIC)			Uncommon	W
Glossy Ibis	*Plegadis falcinellus*	Near threatened (VIC)			Uncommon	W
Australian White Ibis	*Threskiornis moluccus*		Supports critical life stage of breeding (~7,500 nests on Mud Island)		Common	WT
Straw-necked Ibis	*Threskiornis spinicollis*		11,000 (1.1% of population)		Common	OW
Royal Spoonbill	*Platalea regia*	Near threatened (VIC)			Uncommon	WT
Yellow-billed Spoonbill	*Platalea flavipes*				Uncommon	W

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Eastern Osprey	*Pandion haliaetus cristatus*				Rare	B
Black-shouldered Kite	*Elanus axillaris*				Common	O
Letter-winged Kite	*Elanus scriptus*				Rare	O
Black-breasted Buzzard	*Hamirostra melanosternon*				Rare	O
White-bellied Sea-Eagle	*Haliaeetus leucogaster*	Vulnerable (VIC)			Uncommon	WB
Whistling Kite	*Haliastur sphenurus*				Common	W
Black Kite	*Milvus migrans*				Common	O
Brown Goshawk	*Accipiter fasciatus*				Uncommon	F
Collared Sparrowhawk	*Accipiter cirrhocephalus*				Uncommon	F
Grey Goshawk	*Accipiter novaehollandiae*	Vulnerable (VIC)			Uncommon	F
Spotted Harrier	*Circus assimilis*	Near threatened (VIC)			Uncommon	O

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Swamp Harrier	*Circus approximans*				Abundant	W
Wedge-tailed Eagle	*Aquila audax*				Uncommon	F
Little Eagle	*Hieraaetus morphnoides*				Uncommon	F
Nankeen Kestrel	*Falco cenchroides*				Uncommon	O
Brown Falcon	*Falco berigora berigora*				Common	O
Australian Hobby	*Falco longipennis*				Uncommon	O
Black Falcon	*Falco subniger*	Vulnerable (VIC)			Uncommon	O
Peregrine Falcon	*Falco peregrinus*				Uncommon	F
Brown Skua	*Catharacta antarcticus*				Rare	B
Pomarine Jaeger	*Stercorarius pomarinus*				Rare	B
Arctic Jaeger	*Stercorarius parasiticus*			Migratory	Uncommon	B
Black Noddy	*Anous minutus*			Vagrant	Rare	M

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Little Tern	*Sternula albifrons*	Vulnerable (VIC)		Migratory	Rare	B
Fairy Tern	*Sternula nereis*	Endangered (VIC); Vulnerable (Aus)	Supports a vulnerable species; 59 (3.4% of population);		Uncommon	B
Australian Gull-billed Tern	*Gelochelidon nilotica macrotarsa*				Rare	WTB
Caspian Tern	*Hydroprogne caspia*	Near threatened (VIC)			Rare	WB
Whiskered Tern	*Chlidonias hybridus*	Near threatened (VIC)		Migratory	Abundant	W
White-winged Black Tern	*Chlidonias leucopterus*	Near threatened (VIC)		Migratory	Uncommon	W
White-fronted Tern	*Sterna striata*	Near threatened (VIC)		Migratory		BM
Common Tern	*Sterna hirundo*			Migratory	Uncommon	B
Arctic Tern	*Sterna paradisaea*			Migratory	Rare	M

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Crested Tern	*Thalasseus bergii*		Supports critical life stage of breeding (~1,300 nests on Mud Island)		Uncommon	B
Pacific Gull	*Larus pacificus*	Near threatened (VIC)			Uncommon	TB
Kelp Gull	*Larus dominicanus*				Rare	T
Franklin's Gull	*Leucophaeus pipixcan*			Vagrant	Rare	WTB
Silver Gull	*Chroicocephalus novaehollandiae*		41,000 (2% of population)		Abundant	WTB
Barking Owl	*Ninox connivens*	Endangered (VIC)			Rare	F
Southern Boobook	*Ninox boobook*				Uncommon	F
Eastern Barn Owl	*Tyto alba delicatula*				Uncommon	O
Eastern Grass Owl	*Tyto longimembris*				Rare	O
Azure Kingfisher	*Ceyx azureus*	Near threatened (VIC)			Rare	W

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Laughing Kookaburra	*Dacelo novaeguineae*				Uncommon	F
Sacred Kingfisher	*Todiramphus sanctus*				Uncommon	F
SHORE AND WATER-BASED BIRDS						
Magpie Goose	*Anseranas semipalmata*	Near threatened (VIC)			Rare	W
Plumed Whistling-Duck	*Dendrocygna eytoni*				Rare	W
Musk Duck	*Biziura lobata*	Vulnerable (VIC)	1020 (4% of population)		Abundant	WB
Freckled Duck	*Stictonetta naevosa*	Endangered (VIC)			Common	W
Cape Barren Goose	*Cereopsis novaehollandiae*				Uncommon	WO
Black Swan	*Cygnus atratus*				Abundant	WB
Australian Shelduck	*Tadorna tadornoides*		11,000 (1.1% of population)		Abundant	WO
Muscovy Duck*	*Cairina moschata*					UW

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Australian Wood Duck	*Chenonetta jubata*				Uncommon	WO
Pink-eared Duck	*Malacorhynchus membranaceus*		17,100 (1.7% of population)		Abundant	W
Australasian Shoveler	*Spatula rhynchotis*	Vulnerable (VIC)	5,900 (5.9% of population)		Abundant	W
Northern Shoveler	*Spatula clypeata*			Vagrant	Rare	W
Grey Teal	*Anas gracilis*				Abundant	WB
Chestnut Teal	*Anas castanea*		7,000 (7% of population)		Abundant	WB
Northern Mallard*	*Anas platyrhynchos*				Rare	UW
Pacific Black Duck	*Anas superciliosa*				Abundant	WU
Hardhead	*Aythya australis*	Vulnerable (VIC)			Abundant	W
Tufted Duck	*Aythya fuligula*			Vagrant	Rare	W
Blue-billed Duck	*Oxyura australis*	Endangered (VIC)	3,100 (3.1% of population)		Abundant	W

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Australasian Grebe	*Tachybaptus novaehollandiae*				Common	W
Hoary-headed Grebe	*Poliocephalus poliocephalus*		15,600 (1.6% of population)		Abundant	WB
Great Crested Grebe	*Podiceps cristatus*				Common	WB
Brolga	*Antigone rubicunda*	Vulnerable (VIC)			Rare	W
Purple Swamphen (Australasian Swamphen)	*Porphyrio porphyrio melanotus*				Common	W
Lewin's Rail	*Lewinia pectoralis*	Vulnerable (VIC)			Uncommon	W
Buff-banded Rail	*Hypotaenidia philippensis*				Common	W
Baillon's Crake	*Zapornia pusilla*	Vulnerable (VIC)			Uncommon	W
Australian Spotted Crake	*Porzana fluminea*				Common	W
Spotless Crake	*Zapornia tabuensis*				Uncommon	W
Black-tailed Native-hen	*Tribonyx ventralis*				Uncommon	W

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Dusky Moorhen	*Gallinula tenebrosa*				Rare	W
Eurasian Coot	*Fulica atra australis*		15,800 (>1% of population)		Abundant	W
Bush Stone-curlew	*Burhinus grallarius*	Endangered (VIC)			Rare	F
Australian Pied Oystercatcher	*Haematopus longirostris*		158 (1.3% of population)		Uncommon	T
Sooty Oystercatcher	*Haematopus fuliginosus*	Near threatened (VIC)			Rare	T
Black-winged Stilt (Pied Stilt)	*Himantopus leucocephalus*				Common	W
Red-necked Avocet	*Recurvirostra novaehollandiae*		2,000 (>1% of population)		Common	WT
Banded Stilt	*Cladorhynchus leucocephalus*				Common	W
Pacific Golden Plover	*Pluvialis fulva*	Vulnerable (VIC)	Regularly supports critical life stage of migration	Migratory	Uncommon	T

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Grey Plover	*Pluvialis squatarola*	Endangered (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	T
Red-capped Plover	*Charadrius ruficapillus*				Common	WT
Double-banded Plover	*Charadrius bicinctus*		690 (1.4% of population)	Migratory	Common	WT
Lesser Sand Plover	*Charadrius mongolus*	Critically endangered (VIC); Endangered (Aus)	Supports an endangered species; regularly supports critical life stage of migration	Migratory	Rare	T
Greater Sand Plover	*Charadrius leschenaultii*	Critically endangered (VIC); Vulnerable (Aus)	Supports a vulnerable species; regularly supports critical life stage of migration	Migratory	Rare	T
Oriental Plover	*Charadrius veredus*			Migratory	Rare	W
Black-fronted Dotterel	*Elseyornis melanops*				Uncommon	W

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Hooded Plover	*Thinornis cucullatus*	Vulnerable (VIC and Aus)	Supports a vulnerable species		Rare	T
Red-kneed Dotterel	*Erythrogonys cinctus*				Uncommon	W
Banded Lapwing	*Vanellus tricolor*				Uncommon	O
Masked Lapwing	*Vanellus miles*				Common	OW
Australian Painted Snipe	*Rostratula australis*	Critically endangered (VIC); Endangered (Aus)			Rare	W
Latham's Snipe	*Gallinago hardwickii*	Near threatened (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	W
Black-tailed Godwit	*Limosa limosa*	Vulnerable (VIC)	Supports critical life stage of migration	Migratory	Rare	WT
Hudsonian Godwit	*Limosa haemastica*			Vagrant	Rare	WT

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Bar-tailed Godwit	*Limosa lapponica*	Vulnerable (Aus)	Supports a vulnerable species; supports critical life stage of migration	Migratory	Uncommon	T
Little Curlew	*Numenius minutus*			Migratory	Rare	OW
Whimbrel	*Numenius phaeopus*	Vulnerable (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	T
Eastern Curlew	*Numenius madagascariensis*	Vulnerable (VIC); Critically endangered (Aus)	Supports a critically endangered species; regularly supports critical life stage of migration	Migratory	Rare	T
Terek Sandpiper	*Xenus cinereus*	Endangered (VIC)		Migratory	Rare	WT
Common Sandpiper	*Actitis hypoleucos*	Vulnerable (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	WT

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Grey-tailed Tattler	*Tringa brevipes*	Critically endangered (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	T
Common Greenshank	*Tringa nebularia*	Vulnerable (VIC)	Regularly supports critical life stage of migration	Migratory	Uncommon	WT
Lesser Yellowlegs	*Tringa flavipes*			Vagrant	Rare	W
Marsh Sandpiper	*Tringa stagnatilis*	Vulnerable (VIC)	Regularly supports critical life stage of migration	Migratory	Uncommon	W
Wood Sandpiper	*Tringa glareola*	Vulnerable (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	W
Ruddy Turnstone	*Arenaria interpres interpres*	Vulnerable (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	T
Asian Dowitcher	*Limnodromus semipalmatus*			Migratory	Rare	T

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Great Knot	*Calidris tenuirostris*	Endangered (VIC); Critically endangered (Aus)	Supports a critically endangered species; regularly supports critical life stage of migration	Migratory	Rare	T
Red Knot	*Calidris canutus*	Endangered (VIC and Aus)	Supports an endangered species; regularly supports critical life stage of migration	Migratory	Uncommon	T
Sanderling	*Calidris alba*	Near threatened (VIC)		Migratory	Rare	T
Little Stint	*Calidris minuta*			Vagrant	Rare	WT
Red-necked Stint	*Calidris ruficollis*		12,600 (3.9% of population); regularly supports critical life stage of migration	Migratory	Abundant	WT
Long-toed Stint	*Calidris subminuta*	Near threatened (VIC)		Migratory	Rare	W

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
White-rumped Sandpiper	*Calidris fuscicollis*			Vagrant	Rare	W
Pectoral Sandpiper	*Calidris melanotos*	Near threatened (VIC)	Regularly supports critical life stage of migration	Migratory	Rare	W
Sharp-tailed Sandpiper	*Calidris acuminata*		4,800 (3% of population); regularly supports critical life stage of migration		Abundant	WT
Curlew Sandpiper	*Calidris ferruginea*	Endangered (VIC); Critically endangered (Aus)	Supports a critically endangered species; 5,700 (5.7% of population); regularly supports critical life stage of migration	Migratory	Common	WT
Stilt Sandpiper	*Calidris himantopus*			Vagrant	Rare	W
Buff-breasted Sandpiper	*Calidris subruficollis*			Vagrant	Rare	W

Common Name	**Scientific Name**	**Conservation status** in Victoria or Australia	**Ramsar criteria**	**Migratory status** Intra-Australian migratory Migratory Vagrant	**WTP status** 1-10 rare, 11-100 uncommon, 101-1000 common, >1,000 abundant	**Main local habitats** B - sheltered bay waters F - forests, woodlands M - marine waters O - open country, farmland S - saltmarsh T - tidal mudflats U - urban, suburban W - land-locked wetlands
Broad-billed Sandpiper	*Calidris falcinellus*			Migratory	Rare	WT
Ruff	*Calidris pugnax*		Regularly supports critical life stage of migration	Migratory	Rare	W
Red-necked Phalarope	*Phalaropus lobatus*			Vagrant	Rare	W
Oriental Pratincole	*Glareola maldivarum*			Migratory	Rare	W
Australian Pratincole	*Stiltia isabella*	Near threatened (VIC)			Rare	W

Migratory status – Intra-Australian migratory = seasonally migrates away from breeding grounds, but remains within Australia, Migratory = regular, seasonal movement along a flyway, Vagrant = outside its expected breeding, overwintering or migrating range; WTP status - abundancy at the Melbourne Water Western treatment plant: Rare = 1 - 10 individuals, Uncommon = 11 - 100 individuals, Common = 101 - 1,000 individuals, Abundant = >1,000 individuals; * introduced species; [1] usually not present in Australia, occasional reports, believed to be brought in on ships

APPENDIX 2. HAZARD LIST

The hazard list was compiled using a combination of published literature from Australia and overseas, leading textbooks on wild bird and wildlife disease (Friend, 1987, Ladds, 2009) and reports on surveillance and bird health in grey literature. Published literature was identified by searching PubMed, Scopus and Google Scholar with terms: ((("wild birds" OR "avian" OR bird*)) AND ("australia" OR australia*)) AND ("disease" OR "infection" OR infectio*). Papers that discussed disease only related to humans, domestic animals or mammals, focussed on treatment of captive species or laboratory settings or genomics or immunology as well as publications from different climatic zones to Victoria were excluded. Diseases were included if they occur in humans, mammals or domestic birds and the significance of wild birds in the epidemiology is currently unknown. Similarly, where the clinical significance of the disease in wild birds is unknown or it currently only occurs in introduced species, the disease was included in the list. Parasitic diseases were only included if there are reports of clinically significant infections. Therefore, this should not be seen as an exhaustive list of avian parasites.

Potential infectious hazards to avian populations of interest

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
VIRAL						
Adenovirus	Psittacine adenovirus Adenovirus spp. Egg-drop syndrome-76 virus	Psittacine adenovirus has been reported in captive psittacine on Port Phillip Bay and in captive orange bellied parrots in Tasmania and South Australia. Adenoviruses have been identified in a range of captive psittacine species and a tawny frogmouth. There is a record of adenovirus identification in captive birds in the Port Phillip bay area. Antibodies to egg-drop syndrome-76 virus have been detected in wild birds in WA.		Yes		(Hulbert et al., 2015, Yang et al., 2019, Ladds, 2009, Wilcox et al., 1983a)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Arboviruses	Genus *Flavivirus:* Japanese encephalitis (JEV) Murray Valley encephalitis virus (MVEV) Kunjin virus (KUN) West Nile virus (WNV) Usutu virus (USUV) Bagaza virus (BAGV) Genus *Alphavirus:* Ross River virus (RRV)	JEV, WNV, USUV and BAGV are exotic to Australia. JEV is present in south east Asia and has been moving closer towards Australia. The reservoirs hosts are thought to be herons and egrets. MVEV is endemic in Australia and has a wild bird reservoir, thought to be herons and cormorants. There have been seropositive birds found in Victoria. It is asymptomatic in birds. KUN is closely related to WNV, also referred to as WNV Kunjin subtype. KUN is endemic in Australia where the main reservoir is the Nankeen night heron and potentially other waders. WNV is present throughout North America, Africa and Asia. In wild birds it previously caused little clinical disease, however a more recently emerged virulent North American strain causes mass mortalities in water and migratory birds. It has caused disease in humans and a range of other mammals. USUV is present in South Africa and Europe and causes mass mortalities in	Yes	Yes	Yes	(Marshall et al., 1982, Anderson, 1953, Whiteley, 2020, Fereidouni et al., 2011) Cox-Witton, K., pers. comm.

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
		passerines and birds of prey and rarely disease in mammals and humans. BAGV is present in the Middle East and Europe, causes clinical disease in Galliformes and pigeons and is not known to be zoonotic. Unidentified flavivirus serum positives have been reported in long-billed corellas in Kaniva, VIC and shearwaters in Warrnambool, VIC. RRV is endemic to Australia. The reservoir is likely to be marsupials, but a full epidemiological picture does not exist yet. Birds do not show clinical disease.				

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Avian influenza	Avian influenza virus	HPAI is widespread in wild birds internationally, with mortalities reported in China and Europe. The first major reported outbreak of HPAI in wild birds occurred in Qinghai, China in 2005 (H5N1) with subsequent disease events in China, other parts of Asia and Europe. Active surveillance and disease outbreak investigations identified HPAI as the causes of the mortality events. No virus, but antibodies detected in migratory wild birds in Australia. Previous outbreaks in poultry. LPAI exposure detected in Australian and Victorian wild birds with LPAI H5 predominant. Influenza infection and disease has been reported in a range of mammalian species overseas and may be a risk to Australian wildlife such as pinnipeds, domestic poultry (chickens, ducks, turkeys), pigs and possibly humans.	Yes	Yes	Yes (potentially)	(Ferenczi et al., 2016, Grillo et al., 2015, Peroulis and O'Riley, 2004, Tracey, 2010, White, 2013, Wang et al., 2008, Chen et al., 2005, Kleyheeg et al., 2017, Wille et al., 2019a)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Avian paramyxovirus	APMV-1, 5 & 6 PPMV-1	Widespread presence of avirulent APMV-1 in duck and waterbird species in Australia, including in Victoria. Novel APMV virus detected in musk lorikeet. Not reported to cause disease in wild birds in Australia, however virulent APMV-1 has caused mass mortality events in cormorants in North America. PPMV-1 outbreak in pigeons with some spill over into other species.	Yes (rare and mild)	Yes		(Peroulis and O'Riley, 2004, Hoque et al., 2012, Hore et al., 1973, Ladds, 2009, Australian Wildlife Health Network, 2012, Amery-Gale et al., 2018, Pedersen et al., 2016, Bergfeld et al., 2016, Wille et al., 2019b)
Avian polyomavirus		Unlikely to cause disease in wild birds, however significant disease seen in captive birds in Australia. Captive psittacine case reported in Port Phillip Bay. Seropositives in wild sulphur-crested cockatoos in NSW.				(Hulbert et al., 2015, Raidal et al., 1998)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Avian poxvirus		Avipoxviruses are endemic to Australia, except for psittacinepox. Poxviruses have been diagnosed histologically in Mornington Peninsula, VIC in a raven, in Geelong from a sulphur-crested cockatoo and a novel virus from a crimson rosella in Healesville, VIC. In their host species and an endemic area, clinical disease is minimal. Severe disease may occur when introduced into a naïve population.				(Sarker et al., 2017, Slocombe et al., 2013, Friend, 1987, Whiteley, 2020)
Coronavirus		Unlikely to be causing disease in wild birds. Coronaviruses (CoV) have been detected in wild birds on every continent. An average prevalence of 15% was found in a study from Victoria with mainly Charadriiforms and Anseriformes sampled. Another study (Victoria) did not detect CoV. CoV strains cause diseases of human and livestock production significance. There may be a role of migratory shorebirds as vectors.	Yes	Yes	Yes	(Chamings et al., 2018, Amery-Gale et al., 2018)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Herpesvirus	Avian herpesvirus spp. Columbid herpesvirus Duck virus enteritis (DVE) Goose herpesvirus Psittacid herpesvirus (PsHV)	Herpesvirus has been identified in various species in Victoria and across Australia. Clinical disease outbreaks of columbid herpesvirus have been seen in geese, swans, raptors and pigeons in Victoria. There is no known presence of PsHV in wild birds in Australia. However, it has been identified in captive eclectus parrots (PsHV-3) in NSW and green-winged macaws (PsHV-1) in NSW and Qld. An outbreak of goose herpesvirus was reported in Queensland in domestic geese. No outbreaks have been recorded in Victoria. DVE is exotic to Australia, but has caused significant disease in wild birds overseas.		Yes		(Amery-Gale et al., 2018, Phalen et al., 2017, Phalen et al., 2011, Reece et al., 1987, Gough and Hansen, 2000, Ketterer et al., 1990, Gabor et al., 2013)
Infectious bursal disease	Infectious bursal disease virus	Significant clinical disease in domestic chickens and turkeys. No clinical disease recorded in wild birds. Virus and exposure have been detected in wild birds in various countries including Australia (WA). Very virulent strains are not present in Australia.		Yes		(Kasanga et al., 2008, Wilcox et al., 1983a, Wilcox et al., 1983b, Ogawa et al., 1998, Jeon et al., 2008)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Proventricular dilatation disease	Avian bornavirus	Infects psittacines, passerines, waterfowl, gulls and raptors. There are no reports in wild birds in Australia, however there are cases from captive birds and wild birds overseas have been found to carry it.		Yes		(Weissenbock et al., 2009, Doneley et al., 2007, Doneley and Phalen, 2013)
Psittacine beak and feather disease	Psittacine beak and feather disease virus (PBFD)	PBFD is endemic in Australia and clinical disease mainly occurs in psittacines. Many other species may be infected, however and passerines and columbids may show similar pathology. A survey of captive psittacines around Port Phillip Bay show a prevalence of 30%.				(Hulbert et al., 2015, Ladds, 2009, Amery-Gale et al., 2017)
Quaranjavirus	Cygnet River virus Wellfleet Bay virus Johnston Atoll virus	Recently described genus of viruses causing disease in wild and domestic ducks and found in arthropod vectors. Closely related virus has been reported to cause human disease. Cygnet River virus was detected in mortality events in captive Muscovy ducks in South Australia. Prevalence and significance in Australian wild birds is unknown.		Yes		(Allison et al., 2015, Kessell et al., 2012, Ballard et al., 2017)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Rotavirus	Pigeon rotavirus	Likely widespread low-level rotavirus infection throughout world with minimal clinical disease. An outbreak of pigeon rotavirus in captive and feral pigeons occurred in all states of Australia. Unknown significance and impact on native species.		Yes		(McCowan et al., 2018, Ursu et al., 2011)
BACTERIAL						
Borreliosis	*Borrelia* spp.	Unknown significance and presence in Australian birds. *Borrelia burgdorferi* causes Lyme disease in humans in the Northern Hemisphere.	Yes	Yes	Yes	(Chalada et al., 2016, Wildlife Health Australia, 2016)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Botulism	*Clostridium botulinum* toxin Type C, C/D mosaic, E	Diagnosis of botulism is difficult and toxin testing is currently not available in Victoria. Samples may be sent to laboratories in QLD or WA, but concerns around the accuracy of the diagnostic tests exist. Therefore, most outbreaks are presumptively diagnosed. Botulism spores persist in the environment. Numerous weather and water factors contribute to the overgrowth of *C. botulinum* and toxin production. Anseriformes are predisposed, however all bird species may be vulnerable during an outbreak. Recent local outbreaks include Mud Island in 2018 and Melbourne Water Western treatment plant 2016 (see Table 1). Outbreaks occur periodically throughout Australia and overseas.	Yes		Yes	(McKenzie et al., 1982, Friend, 1987, Woodall, 1982, Hoque et al., 2012, Galvin et al., 1985, Whiteley, 2020, Brandis et al., 2019)
Chlamydiosis	*Chlamydia psittaci*	Widespread globally, including Australia. Most bird species likely to be susceptible, although clinical disease is less common. Wild birds possibly have lower prevalence than captive.	Yes	Yes		(Blomqvist et al., 2012, Amery-Gale et al., 2019, Friend, 1987)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Enteric pathogens	*Campylobacter jejuni* Other *Campylobacter* spp.	No clinical disease seen in free-living birds. Outbreaks of disease have been recorded in emus, Gouldian finches and poultry in captivity in Australia	Yes	Yes	Yes	(Ladds, 2009)
	Clostridial/necrotising enteritis *Clostridium perfrigens*	Outbreaks have been reported mainly in lorikeet species in Victoria and Australia. Overseas, outbreaks have occurred in crows and waterfowl.				(McOrist and Reece, 1992, Wobeser and Rainnie, 1987, Asaoka et al., 2004, Grillo and Post, 2012)
	Escherichia albertii	Widespread presence in Australia and worldwide. Unknown prevalence in waterfowl and seabirds in Australia. Cause of mortality events in Europe and North America, none reported in Australia.	Yes	Yes	Yes	(Gordon and Cowling, 2003, Oh et al., 2011, Oaks et al., 2010, Gordon, 2011)
	Escherichia coli	Can cause acute or subacute disease or be carried asymptomatically. *E. coli* has been isolated from birds in the Ramsar site – significance is currently unknown. Species carrying significant antimicrobial resistance genes have been isolated from gulls in NSW.	Yes	Yes	Yes	(Dolejska et al., 2016, Gordon and Cowling, 2003, Oh et al., 2011, Whiteley, 2020)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
	Edwarsiella tarda	Wild birds in Australia are assumed to be carriers. Overseas there have been clinical disease cases, including in species that may visit Australia.	Yes	Yes	Yes	(Ladds, 2009)
	Salmonellosis	Wild birds may be carriers without showing signs of disease with studies in white ibis and silver gulls in NSW showing a prevalence of 25-30%. *S.* Typhimurium DT160 has spread into areas of Australia with mass mortality outbreaks in house sparrows and spotted turtle doves in Tasmania and Melbourne. Other bird species may be affected.	Yes	Yes	Yes	(Dolejska et al., 2016, Friend, 1987, Hoque et al., 2012, Maute et al., 2019, McOrist and Reece, 1992, Grillo et al., 2017)
Erysipelas	*Erysipelothrix rhusiopathiae*	Significant disease in livestock production. Wild birds are a potential reservoir, but epidemiology not well described. Disease has been identified in wild birds in Australia.	Yes	Yes	Yes	(Ladds, 2009, Munday, 1972, Eamens et al., 1988)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Mycobacteriosis/ Avian tuberculosis	*Mycobacterium avium* subsp. *avium* *M. genavense*	Causes sporadic disease of progressive and chronic nature. Found worldwide, including Australia with a higher prevalence in captive birds. Persists in the environment. Cases in Brolgas and Australian Bustard have been identified in Lara, VIC and in pigeons and a Nankeen kestrel elsewhere in Victoria.	Yes	Yes		(Hodge et al., 2019, Reece et al., 1992, Whiteley, 2020)
Pasteurellosis/ Avian cholera	*Pasteurella multocida*	Mortality of a Chestnut teal occurred at Melbourne Water Western Treatment Plant in March 2013. Mortalities of a Black Swan and Eurasian coots occurred at Lake Tootiorook (Lismore, VIC) in May 2013. Cause of significant disease in the poultry industry. Mass mortality events seen in the last fifty years in North America. Mortality events have also been seen in China and Europe. Multi-host infection.	Yes	Yes		(Wang et al., 2009, Pedersen et al., 2003, Thomas et al., 2008, Ladds, 2009, Samuel et al., 2007, Friend, 1987) Whiteley, P., pers. comm.
Staphylococci & Streptococci	*Staphylococcus aureus* *S. intermedius* *Streptococcus spp.*	Sporadic and opportunistic infections, generally cause septicaemia or pododermatitis.				(Ladds, 2009)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Yersiniosis	*Yersinia pseudotuberculosis* *Y. enterocolitica*	Causes sporadic disease but may result in larger mortality events. Widespread carriage in wild birds is suspected throughout the world and in Australia	Yes	Yes	Yes	(Otsuka et al., 1994, Elmberg et al., 2017, Cork et al., 1995, Ladds, 2009)
FUNGAL						
Aspergillosis	*Aspergillus fumigatus* *Aspergillus spp.*	Sporadic cases occur in Australia. Mass mortality events have occurred in North America and New Zealand. Cases have been reported in little penguins in VIC, a magpie goose in Lara, VIC and a black swan in Lake Bolac, VIC.	Yes	Yes	Yes	(Obendorf and McColl, 1980, Friend, 1987, Whiteley, 2020)
Candidiasis	*Candida albicans*	Normal flora of birds that may cause opportunistic infections. May have a role in nestling deaths. Increased cases should trigger investigation of underlying cause of stress or disease. A Spanish outbreak in vultures and eagles was linked to ingestion of antimicrobial residues in livestock carcasses.	Yes			(Rippon et al., 2010, Pitarch et al., 2017, Ladds, 2009)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Cryptococcosis	*Cryptococcus neoformans* var. *grubii* *C. bacillisporus*	Cases of disease seen in Australia likely primary rather than opportunistic. Wild birds may carry *Cryptococcus* spp. showing antimicrobial resistance. A Spanish outbreak in vultures and eagles was linked to ingestion of antimicrobial residues in livestock carcasses.	Yes			(Malik et al., 2003, Lord et al., 2010, Pitarch et al., 2017)
Proventricular disease	*Macrorhabdus ornithogaster* (Avian gastric yeast, AGY)	No clinical disease published in wild birds but causes disease in captive and domestic birds. Infection has been confirmed in orange-bellied parrots. Suggestion of outbreaks in wild birds in QLD and WA. Likely widespread in wild birds in Australia.		Yes		(Ladds, 2009, Doneley and Phalen, 2013) Wicker, L. pers. comm.
INTERNAL PARASITES						
PROTOZOA						
Coccidiosis	*Atoxoplasma/Isospora spp.*Lankesterella *Eimeria spp.*	Many coccidial species found in a wide range of birds. Some clinical disease reported in wild birds, often associated with other diseases or periods of stress (e.g. migration).				(Obendorf and McColl, 1980, Ladds, 2009)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Cryptosporidiosis	*C. baileyi* *C. meleagridis*	Carried by a range of wild birds. Clinical disease is rare but has occurred in Australia and overseas.	Yes (unknown)	Yes		(Cano et al., 2016, Moore et al., 2002)
Giardiasis	*Giardia* spp.	Clinical disease reported in Australia in wild ibises and sulphur-crested cockatoo. Overseas it has been identified in a variety of species in Europe and North America.	Yes	Yes		(Cano et al., 2016, Elmberg et al., 2017, Ladds, 2009)
Haemosporidia	*Haemoproteus* spp. *Leucocytozoon* spp. *Plasmodium relictum*	Widespread prevalence in Australia and overseas. Most species are co-evolved and therefore don't cause clinical disease. Some cases of clinical disease have been recorded in wild birds. Various species have been detected in anatids in Lara, Victoria and little penguins in WA.		Yes		(Bennett et al., 1977, Cannell et al., 2013, Merino et al., 2012, Beadell et al., 2004, Verwey et al., 2018)
Sarcocystosis	*Sarcocystis* spp. *S. neurona* *S. falcatula*	There is widespread prevalence in a wide range of bird species in Australia but no reports of mortality or clinical disease. It has been identified in passerine, birds of prey and psittacines in Victoria. The epidemiology in Australia is poorly understood.			Yes	(Friend, 1987, Munday et al., 1979, Ladds, 2009)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Spironucleosis	*Spironucleus* spp.	May cause disease in wild and captive birds, fish and some mammals. It has been detected in king parrots in Benalla and Anglesea, VIC and rainbow lorikeets in Bairnsdale, VIC. King parrots are especially predisposed to disease.		Yes	Yes	(Philbey et al., 2002, Whiteley, 2020)
Toxoplasmosis	*Toxoplasma gondii*	*T. gondii* can cause clinical disease in birds, humans and many species of mammals around the world. Subclinical infections are present in a wide range of wild bird species in Australia. It has been identified in passerines, psittacines, columbids and birds of prey. Clinical disease has been seen in a little penguin in Tasmania, a powerful owl in Victoria In European studies the seroprevalence in waterfowl ranged from 9-31%.	Yes	Yes	Yes	(Hartley and Dubey, 1991, Mason et al., 1991, Reece et al., 1992, Mancianti et al., 2013, O'Donoghue and Adlard, 2000, Dubey, 2002)
Trichomoniasis	*Trichomonas gallinae*	Widespread prevalence globally with pigeons being the main carriers. Identified throughout Australia and Victoria. Trichomoniasis has caused widespread declines in finch populations in the UK as well as declining columbid populations in North America and other parts of Europe.		Yes		(Park, 2011, Ladds, 2009, Lawson et al., 2012, Ostrand et al., 1995, Lehikoinen et al., 2013)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
HELMINTHS						
Cestodes (Tapeworms)	Intestinal cestodiasis	Rarely causes clinical disease but have been identified as part of disease complexes in Australia. May cause disease in Black swans. Found to possibly be impacting on health in little penguins, a crested tern, magpie, black swans, Pacific black ducks, sacred kingfisher, sulphur-crested cockatoo, a blue bonnet, black-winged stilts, Australian raven, Torresian crow, superb lyrebird, seagull and pelicans in Victoria.		Yes	Yes	(Reece et al., 1992, Ladds, 2009) Beveridge, I., pers. comm.
	Sparganosis	Found throughout the world due to a lack of host preference and the distribution of cats and dogs, who act as definitive hosts. Noted in several Australian species, including in a sacred kingfisher and kookaburras in Victoria.	Yes	Yes	Yes	(Ladds, 2009, Reece et al., 1992)
Nematodes	*Acuaria* spp.	A range of birds including passerines, psittacines, raptors, waterfowl and seabirds have been identified as carrying *Acuaria* spp. in Australia. Clinical disease is seen in finches				(Ladds, 2009, Reece et al., 1992), Beveridge, I., pers. comm.

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
	Angiostrongylus cantonensis	Reports of bird infections and disease in Australia. *A. cantonensis* not known to be present in Victoria.	Yes	Yes	Yes	(Monks et al., 2005, Montali et al., 2004, Prociv and Carlisle, 2001)
	Capillaria sp.	May affect a wide range of wild birds in Australia. Clinical disease is uncommon but does occur.				(Ladds, 2009)
	Contracaecum sp.	Has been identified in a wide range of wild bird species with few reports of clinical disease. Ulcers have been observed in Australian pelicans and little penguins in Victoria with other concurrent helminth infestation. Their clinical significance alone is unknown.				(Ladds, 2009, McKenzie et al., 1982, Reece et al., 1992), Beveridge, I., pers. comm.
	Syngamus trachea	Likely widespread prevalence in Australia. Lesions or clinical disease has only been reported in magpies, brush-turkey, emus and yellow-tailed black cockatoo.		Yes		(Ladds, 2009, Reece et al., 1992)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Trematodes (Flukes)	Alimentary trematodiasis	There are few reports of clinical disease in wild birds in Australia, although there may be heavy subclinical burdens. A pelican and little penguins have been identified as having infections that may have been clinically significant. Black swans and pelicans can have very heavy burdens of very small <2mm intestinal fluke, health impacts not known. Mortalities have been seen in domestic Muscovy ducks in NSW.		Yes		(Ladds, 2009, Obendorf and McColl, 1980) Beveridge, I., pers. comm.
	Hepatic trematodiasis	Mortalities have been recorded in little penguins in Victoria.				(Ladds, 2009, Obendorf and McColl, 1980)
	Trichobilharzia spp.	Duck and swan species can carry these parasites (also known as nasal schistosomiasis) in their nasal cavity without signs of disease. Causes cercarial dermatitis (or "swimmer's itch") in humans.	Yes		Yes	(Horák et al., 2015, Ladds, 2009) Beveridge, I., pers. comm.
EXTERNAL PARASITES						

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Feather mites	*Dermanyssus* spp. *Ornithonyssus* spp. *Titanolichus seemani*	Present in many species in Australia. Subclinical infections are common, but disease may occur in large infestations in young birds. Cause significant disease in domestic poultry and may rarely cause zoonotic infestations. *T. seemani* is reported only from the orange-bellied parrots, has been found on museum specimens of wild birds and in captive collections. It is not known to cause clinical signs in OBPs, and is believed to be important for feather health.	Yes	Yes	Yes	(Dabert et al., 2006, Ladds, 2009, McKenzie et al., 1982)
Fleas	Many species	Many species present throughout Australia and Victoria. Clinical disease may occur with large numbers and vulnerable wild birds.				(Ladds, 2009, Obendorf and McColl, 1980)
Knemidocoptiasis	*Knemidocoptes* spp.	Present in a range of wild species across Australia.				(Holz et al., 2005, Jaensch et al., 2003, Vogelnest, 2003, Whiteley)
Lice		Many species of lice have been identified on wild birds in Australia and Victoria. They are mostly associated with debilitated animals with concurrent disease.				(Obendorf and McColl, 1980, McKenzie et al., 1982, Whiteley, 2020)

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Ticks	*Argas persicus* *Ixodes kohlsi* *Ixodes eudyptuli*	Ticks are not usually associated with clinical disease but may act as disease vectors or cause anaemia in heavy infestations.	Yes	Yes	Yes	(Obendorf and McColl, 1980, Ladds, 2009)

Hazards with an unknown cause to avian populations of interest

Disease	Causative Agent	Susceptible avian species with relevant comments	Potential zoonosis/ human risk?	Risk to/from domestic animals?	Risk to local (non-bird) wildlife?	References
Rainbow lorikeet paralysis/ Clenched claw syndrome	Unknown	Neurological disease of rainbow and scaly-breasted lorikeets with an unknown aetiology. Reported in Victoria, NSW and Queensland				(McOrist and Perry, 1986, Holz, 2014)
"Black and white" neurological syndrome	Unknown	Neurological disease of Australian magpies, pied currawongs and Australian ravens with an unknown aetiology. Mainly reported in coastal NSW, but has been seen in Victoria and Queensland				(Wildlife Health Australia, 2017a)
Disease X	Unknown	Represents the knowledge that newly emerging disease could be caused by a pathogen currently unknown	Yes (potentially)	Yes (potentially)	Yes (potentially)	(WHO, 2018)

Potential non-infectious hazards to avian populations of interest

Non-infectious hazards		Comment	References
TOXINS			
Heavy metals	Lead toxicosis	Mass mortality events from lead toxicosis have occurred in Victoria, around Australia and around the world. In Victorian wetlands, lead exposure has been measured from 1-30% of sampled ducks. A mass mortality event occurred in Esperance, WA in 2006 in which thousands of birds and many children were affected by lead toxicosis. Waterfowl and birds of prey are at a higher risk. Contamination of the environment may occur from lead shot (in the environment or embedded in animal tissues), presence in soil from historic human activity, run off in aquatic environments and mining dust.	(Norman et al., 1993, Wickson et al., 1992, Sweet and Phalen, 2014, Gulson et al., 2009)
	Iron	High iron levels and haemosiderosis has been commonly found in little penguins and other species. Clinical disease is uncommon; however, it may contribute to a higher than average rate of neoplasms.	(Ladds, 2009)
	Mercury	Mercury toxicity may occur in any predator species, but especially those eating fish. Great egrets and bald eagles in Florida, US have been shown to have high levels of mercury.	(Rumbold, 2005)
	Selenium	Selenium toxicity may reduce birds' resistance to infectious disease. A case of selenium toxicity was reported from California.	(Ohlendorf et al., 1988) (Whiteley and Yuill, 1990)

Non-infectious hazards		Comment	References
Insecticides, herbicides and other pesticides	1080	A toxin used in baits for fox, dog and cat control, it may cause primary or secondary toxicity in birds eating the bait or predating the carcasses. Baiting programs using 1080 should target the type of bait used and locations as closely as possible to the target species to reduce non-target deaths. 1080 breaks down rapidly in the environment and long-term contamination is thought to be minimal.	(Ladds, 2009)
	Anticoagulants	Sporadic deaths of birds of prey caused by ingestions of poisoned prey are common across Victoria and Australia. Occasional deliberate poisonings also occur. Winter/spring 2018 saw a marked increase in barn owl and Nankeen kestrel morbidity and mortality. These were partly attributed to rodenticide toxicity but likely involved a range of factors.	(Grillo et al., 2014a, Grillo et al., 2014b, Grillo and Post, 2010, Grillo, 2011, Whiteley and Hawes, 2018, Lohr, 2018, Lohr and Davis, 2018, McOrist, 1989)
	Metaldehyde	Metaldehyde is used as a molluscicide and may cause disease in birds if ingested. Occurrences are rare.	(Ladds, 2009)
	Organophosphates	Sporadic or mass mortalities may occur from accidental contact or by deliberate poisoning. Several house sparrows died of OP toxicity in Werribee, VIC in 2018.	(Story and Cox, 2001, Whiteley, 2020)
	Persistent Organic Pollutants (POPs)	This chemical group includes organochlorine pesticides, polychlorinated biphenyls (PCB) and dioxins (PCDD and PCDF). They may cause acute toxicity with sudden death or chronic disease. They are very stable in the environment and can accumulate in body fat. Pelicans in Queensland were confirmed to have died of dieldrin toxicity.	(McKenzie et al., 1982, World Health Organization)

Non-infectious hazards		Comment	References
Other toxins	Algal toxins	An unconfirmed outbreak occurred in waterfowl at Melbourne Water Western treatment plant in the summer 2005/06. The deaths were likely multifactorial with changes to infrastructure having prevented any similar outbreaks.	(Steele, 2008)
	Cyanide	High concentrations of cyanide are mostly associated with the tailings from gold mines. Mortality events have occurred in WA.	(Donato et al., 2007, Griffiths et al., 2009)
	Crude oil toxicosis	Crude oil spills can cause mass mortality events and chronic effects on egg and foetal health. Little penguin mortality events have been reported in Victoria. Deaths following oil toxicosis often occur due to secondary diseases.	(Friend, 1987, McOrist and Lenghaus, 1992)
HUMAN ACTIVITY RELATED			
Antimicrobial resistance		Bacteria and fungi resistant (or carrying resistance genes) of importance for human and animal disease have been found in wild birds	(Dolejska et al., 2016, Mukerji et al., 2019, Marcelino et al., 2019)
Medication residues		Medications and drug residues may be present in wetland areas due to agricultural run-off and from human sewage. The effects and significance of these chemicals depend largely on their class and concentration. Little evidence about their effects on wild birds exist.	
Plastics and rubbish		Contamination of water with rubbish causes trauma to aquatic species, disruption to food sources and may disrupt nestling behaviours. Micro-plastics may cause internal pathology. Trauma secondary to fishing wire has been documented in Victoria.	(Fry et al., 1987, Whiteley, 2020)

Non-infectious hazards	Comment	References
Trauma and shooting	Trauma secondary to human contact (such as vehicles, boats etc) are common sporadic events.	(Johnston et al., 2016)
ENVIRONMENTAL		
Climate change, biodiversity changes and associated changes	Climate change has far reaching effects on wetlands and wild bird life. Changes in water levels, rainfall, water temperature, water acidity, salinity and significant weather events will all impact insect numbers, biodiversity and the distribution of bird species. There are likely to be significant effects on arboviruses due to changes in insect activity.	(Carver et al., 2015, Kingsford and Norman, 2002, Jin et al., 2009, Russell, 2009, Hart et al., 1990, Loyn et al., 2014)
Electrocution	Electrocution from powerlines are sporadic events common to all areas of human inhabitation with external powerlines. Electrocution events may trigger infectious disease outbreaks such as botulism by increasing the carcass burden.	(Malcolm, 1982)
Drowning	A mass mortality event of Pink-eared ducks and Australasian Shovelers in full moult was reported at Melbourne Water Western treatment plant in summer 2005/06. Drowning due to water infrastructure as well as possible algal toxicity was assumed to be the cause.	(Steele, 2008)
Fire	Bushfires can cause short and long-term disruption to wetland ecosystems and bird populations. Birds are displaced during and immediately after fire events, which may destroy vital vegetation and other animal populations that serve as habitat and food source. Fire may also be used in areas as a land management tool, especially to control invasive species. Studies from other parts of Australia show a positive effect on bird abundancy.	(McGregor et al., 2010)
Flooding	Water level changes and flooding may impact water bird numbers and behaviours.	(Poiani, 2006, Loyn et al., 2014)

Non-infectious hazards	Comment	References
Invasive species	Invasive species of plants may change the wetland conditions and affect bird feeding and nesting behaviours. Changing flora may also impact the numbers and species of insects and other animals present. Invasive invertebrates and vertebrates may change the food available, impact disease presence and spread and impact native bird species directly through competition or predation (cats, foxes, rodents etc.). Birds themselves can act as dispersal agents of invasive aquatic species.	(Kingsford et al., 2016, Grice et al., 2013, Grice, 2006, Reynolds et al., 2015)
Starvation	Starvation events may be an expected consequence of population change, seasonal variation, disease and migration. For example, Australia sees large numbers of shearwater mortalities every year due to migration exhaustion and starvation. Starvation may, however, also be a consequence of changing disease dynamics and climate/ecological change caused by local or global change in climate and biodiversity. Mass starvation events should be investigated to rule out large-scale infectious disease outbreaks and monitor changing trends.	(Obendorf and McColl, 1980, Siah et al., 2014, Whiteley and Hawes, 2018)

APPENDIX 3. DISEASE SUMMARIES

VIRAL

Adenovirus

Avian adenoviruses cause disease in captive birds and domestic poultry. There are several reports of Australian native birds being affected in captivity and it has been recorded in wild birds, however the significance is unknown. A species of adenovirus has been isolated from captive orange-bellied parrots. Egg-drop syndrome 76 virus is of significance in domestic poultry and seropositivity in wild birds has been detected in parts of Australia.

Clinical Signs & pathology

Subclinical infections are likely common in co-evolved species.

Clinical signs

- Sudden death
- Jaundice
- Tremors
- Weakness, depression and inability to fly
- Ataxia

Pathology

- Hepatomegaly with yellow discolouration, pale foci
- Splenomegaly and pallor
- Pulmonary oedema
- Hydropericardium
- Caecal haemorrhage
- Inclusion bodies may be similar to polyoma virus lesions

Transmission and Epidemiological factors

Stress and concurrent disease may play a role.

References

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Arboviruses

Flaviviruses

The Flavivirus genus, which is part of the Family Flaviviridae, has over 50 species of flaviviruses, many of which use arthropods as vectors. They cause disease in many different species of vertebrates, including mammals and birds. From a human health perspective, perhaps the most important ones are Japanese encephalitis virus (JEV, not present in Australia), Murray Valley Encephalitis virus (MVEV), St Louis encephalitis virus (SLEV, not present in Australia), Dengue virus (DENV), Yellow fever (YFV, not present in Australia), Zika virus (ZIKV, not present in Australia), Kunjin (KUNV) and West Nile virus (WNV, not present in Australia). KUN is closely related to WNV and also referred to as WNV Kunjin subtype.

In Australia, human infection has also, though rarely, been demonstrated by Kokobera (KOKV), Alfuy (ALFV) (sub-type of MVEV), Stratford (STRV) and Edge Hill (EHV). Only the first was associated with disease in humans. There are likely to be a number of other, yet unidentified, flaviviruses in Australia, some of which circulate in native animals such as macropods (Hufschmid J., unpublished data); a flavivirus positive long-billed corella was also found in Kaniva (western Victoria) (Wildlife Health Victoria (WHV) - Surveillance data) and in a shearwater from Lady Bay (WHV data).

While many flaviviruses don't seem to affect their bird hosts, Usutu (USUV) and Bagaza (BAGV) virus have recently caused some concern as they are causing clinical signs in affected birds. Both USUV and BAGV are currently exotic to Australia and are found in a wide range of bird hosts. There are some other exotic flavivirus that may cause disease in animals and have bird reservoirs, such as Tembusu virus (south-east Asia and China), Tick-borne encephalitis virus (Europe and Asia), Omsk Haemorrhagic fever virus (Siberia), and Tyuleniy virus (Russia, North America, northern Europe)

Japanese encephalitis is considered exotic to Australia, but the range of the disease is moving southwards towards the islands in our north. There has been one human case of JEV on the mainland in Cape York (1998), and some sporadic cases on Australian islands off the north of the mainland. The virus has also been isolated from a mosquito on Cape York (in 2004).

Murray Valley encephalitis virus is endemic to Australia, especially the northern part of the country although until the 1970s, outbreaks in humans were seen in the south (including Victoria) as well.

Birds are also the primary host for **Kunjin virus** (and other WNVs), which is thought to be endemic to northern Australia. Kunjin virus has been demonstrated from a range of mammals and reptiles, and it has caused significant mortality in horses. Other viruses from the same **West Nile Virus** lineage, which are exotic to Australia, may cause severe disease in birds, especially the lineage recently emerged in North America.

Usutu virus was first found in South Africa but has more recently caused significant bird mortalities in Europe, killing both wild and pet birds. It can affect other vertebrates, including humans, where it rarely causes disease (though can in immunosuppressed individuals). It is very pathogenic for passerines, esp. *Turdus* spp. (thrushes) and birds of prey.

Bagaza virus seems to have spread from the middle East to Europe and is particularly affecting Galliformes and wood pigeons. It has not been reported from humans.

The primary vertebrate host for **Tembusu virus** are ducks and chickens; affected birds may see reduced reproductive rate and mortality of up to 15%.

Tick-borne encephalitis virus is thought to have bird species as hosts, but details are not known. It may cause mortality in some bird species, including sparrows, the common red poll and the Eurasian coot; it can also cause disease in humans and domestic livestock.

Some birds of prey are susceptible to **Omsk haemorrhagic fever virus**, including the Western marsh harrier, common kestrel, the long-eared owl, as well as rooks.

Tyuleniy virus can be found in seabirds, including the little penguin, but no disease following natural infection has been reported from animals. Three human cases who were in contact with seabirds had developed disease.

Dengue virus, **Zika virus** and **Yellow fever** do not appear to use birds as host, nor do they cause disease in birds. **St Louis encephalitis** virus does use wild birds as primary reservoir host, but it does only occur in the Americas and has never been detected in Australia. **Kokobera** is thought to have mammalian reservoirs hosts, possibly macropods.

Clinical Signs and Pathology

Restricted to those that have been recorded from Australia or are emerging diseases with potential threat to birds in Australia.

Virus	Birds	Human	Other hosts
JEV	**None**	• **No signs (99%)** • Encephalitis (25% mortality)	• **Pigs** • Horses
MVEV	**None**	• **Encephalitis** • meningitis	• **Horses** • Sheep • Monkey
KUNV	**None**	• **No signs** • Encephalitis • Flu-like symptoms • Arthralgia or myalgia • Only 13 confirmed cases	• **Horses**
WNV	• **Low** BUT: virulent N-American strain: • **Anorexia and weakness** • Weight loss • Neurological signs • Recumbency • Sudden death	• **Flu-like symptoms** • Encephalitis/meningitis • Acute flaccid paralysis or poliomyelitis	• **Horses** • **Other mammal species**
USUV	• **Lethargy** • Inability to fly • Neurological signs (encephalitis) • Death • Mortality in blackbirds up to 100%	• **Rare (only in immune suppressed people)**	• **May be found in bats, but no clinical signs**
BAGV	• **Incoordination** • Disorientation • Ataxia • Death	• **unknown**	

Pathology:

USUV: encephalitis, carditis, hepatomegaly and splenomegaly

BAGV: meningoencephalitis, carditis, haemosiderosis (liver and spleen)

Transmission and Epidemiological Factors

Flaviviruses are generally transmitted through arthropods, including mosquito, midge and tick vectors. The virus is acquired during a blood meal from a vertebrate host, and then transmitted through a bite to another host.

Reservoir species or amplifying hosts are those hosts where primary infection with the virus results in extended presence of the virus in the blood (viraemia), allowing another vector to pick the virus up again and thus continue the life cycle of the virus.

The primary host of **JEV** are birds, especially herons and egrets – with pigs and wild birds thought to be amplifying hosts. Dead end hosts include humans and equines.

Birds, especially herons and cormorants are the primary host of **MVEV**, and the rufous night heron is thought to be a major amplifying host. In a 1982 study in south-west NSW and northern Victoria, antibodies to MVEV was found at high prevalence in cormorants (and a darter) (40%) and herons (55%), and much lower prevalence in grebes, ducks and Eurasian coot. Surveillance over the years has found positive antibody samples in a very wide range of birds, especially waterbirds (see WHA fact sheet appendix).

Kunjin virus has been identified from a wide range of Australian birds, with the rufous (Nankeen) night heron being a major reservoir host. One study found a very high prevalence of **WNV** in waterbirds in Iran, especially common coots where over 50% of animals tested had antibodies to the virus.

The incubation period varies between viruses, and will be listed here for those most relevant to Australia:

- JEV: Humans 6-16 days, Pigs 1 day, Horses 8-10 days, Herons 1-2 days; extended incubation in bats, reptiles and amphibian hosts
- MVEV: Humans 7-12 days, Birds 1-2 days (based on experimental evidence)
- KUNV and WNV: Humans 2-14 days, Birds 7-10 days
- USUV: unknown
- BAGV: unknown

Diagnosis

Sampling and specimens:

- Serum
- Plasma (for MVEV)
- Aseptically collected brain or other tissues (chilled)
- Formalin-fixed tissues
- CSF
- Kidney, heart, brain and intestine (WNV)

Testing

- Virus isolation/culture
- Serology for antibodies or antigen (e.g. complement fixation, haemagglutination inhibition, serum neutralisation or ELISA)
- PCR

Control & Prevention

There is no specific treatment for flavivirus infection. Vaccination of mammalian hosts, including humans, pigs and horses is possible for JEV. Insecticidal treatment of domestic animals may have some use in those hosts for prevention.

For MVEV, sentinel chicken flocks are used to warn of increasing viral activity or spread; for JEV, pigs can be surveyed. Surveillance may be useful, including serum banking of potential hosts for later

testing. There is active surveillance for JEV in northern Queensland during the wet season. In Australia, MVEV and JEV are notifiable diseases in humans and humans and animals, respectively.

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Ross River virus

Ross River virus (RRV) causes zoonotic neurological disease and is transmitted by mosquitoes. It is only present in Oceania and is endemic to Australia.

Clinical signs & Pathology

None in birds.

Transmission and Epidemiological factors

Birds have been investigated as reservoirs hosts of RRV, however research suggests that marsupials are better amplifying hosts. Possums, horses and other mammals are possibly involved in transmission and many mosquito species can act as vectors, however the epidemiology of the virus and how it causes disease in humans is not yet clear.

Diagnosis

Specimens and samples

- Serum

Testing

- Serology
    - ELISA
    - Haemagglutination inhibition
- Viral culture

Control and Prevention

Control of mosquito numbers is important in reducing RRV infections in humans. Surveillance of mosquito numbers, virus detection in mosquitoes and active surveillance of sentinel animals is important in monitoring the disease.

References

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Avian influenza

Avian influenza (AI) is caused by influenza type A and is found in birds across the world with the primary reservoirs being *Anseriformes* and *Charadriiformes*. Pathogenic strains (highly pathogenic AI

– HPAI) associated with epidemics in poultry and wild birds have not been found in Australia. Low pathogenicity AI (LPAI) has been detected in wild Australian birds, although there have been outbreaks in poultry. The zoonotic potential of HPAI and threat of significant impact on poultry production makes it a disease of international importance.

Clinical Signs & pathology

LPAI generally does not cause clinical disease. Respiratory lesions may be present.

HPAI may cause sudden death. There may not be any significant pathology however organ haemorrhage, necrosis and inflammation may be present.

Transmission and Epidemiological factors

Influenza viruses are classified by two difference surface proteins – haemagglutinin (H) and neuraminidase (N). This gives them the subtype names of HxNx. H5 and H7 subtypes can mutate from LPAI to HPAI in poultry, of which H5N1 has caused the most severe outbreaks to date. In Australia, LPAI subtypes of H1-6, H11, H12 and H15 have been identified.

Faecal-oral and airborne transmission with viral shedding of one month or more.

Incubation period ranges from hours to seven days.

AIV persists in the environment, including in water.

The epidemiology of AI in Australia is likely different to the rest of the world with rainfall, population numbers and age-structures being the strongest predictors of prevalence.

Diagnosis

Specimens and sampling

- Cloacal, oropharyngeal and faecal swabs
- Serum
- Tissue samples of gastrointestinal tract and respiratory tract

Testing

- PCR – influenza A test then subtyped
- Virus isolation and pathogenicity testing
- Serology
    - ELISA

Control and Prevention

Surveillance as per the National Avian Influenza Wild Bird Surveillance Program. Vaccination of captive birds is possible.

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Avian paramyxovirus (APMV)

APMV can cause significant disease in avian species (both domestic and wild), however the widespread strains in native birds in Australia are avirulent APMV-1. There has been no evidence of virulent strains causing clinical disease in wild birds in Australia, however outbreaks in cormorants in North America have caused mass mortality events.

The term Newcastle Disease (ND) is only used for virulent APMV-1 infections in domestic poultry.

Pigeon paramyxovirus 1 (PPMV-1) was first discovered in Australia in domestic pigeons in 2011. A collared sparrowhawk tested positive to PPMV-1, however comorbidities may have contributed or predisposed to disease.

Clinical Signs and Pathology

No clinical disease in wild birds with avirulent APMV-1 infection.

Pigeons infected with PPMV-1 show signs of depression, anorexia, excessive drinking and polyuria, diarrhoea, neurological signs and death within three days.

Pathology

- Red legs
- Pancreatic degranulation and necrosis of acinar cells
- Pale foci of necrosis (spleen, kidney, pancreas)
- Splenomegaly
- Focally extensive lymphoplasmacytic interstitial nephritis

North American outbreaks of virulent APMV in cormorants have presented in neurological signs and sudden death.

Transmission and Epidemiological Factors

Faecal-oral

The virus survives in the environment for several weeks and survives well in water. Birds may shed the virus for weeks to months.

Diagnosis

Sampling and specimens:

- Serum
- Cloacal and oropharyngeal swabs
- Tissue samples from respiratory tract, digestive tract and brain +/- kidney, spleen, liver, heart
- Faeces
- Whole birds may be recommended.

Testing

- RT-PCR
- Viral isolation and molecular pathotyping
- Serological tests:
    - ELISA
    - HI

Virulent APMV-1 and PPMV-1 are notifiable.

Control & Prevention

An ND vaccine is available for poultry which may be used and provide some protection for at-risk birds in captivity.

Control is aimed at identifying outbreaks in pigeons and preventing spill over into native wild birds.

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Avian polyomavirus

Mainly a disease of captive nestling psittacines and passerines, there is evidence of endemic infections in wild sulphur-crested cockatoos and a possibility of more widespread infection in other species.

Clinical Signs & pathology

In wild birds there tend to be no signs of disease.

In captive birds:

- Feather abnormalities
- Budgerigar fledgling disease
  - Failure to thrive and stunting
  - Abdominal distension
  - Feather abnormalities
- Other birds
  - Sudden death of nestlings
  - Weakness
  - Renal failure
  - Signs of haemorrhage

Pathology

- Ascites
- Hydropericardium and cardiomegaly
- Enlarged liver and kidneys with pale and yellow foci and petechial haemorrhages
- Other signs of haemorrhage

Transmission and Epidemiological factors

Outbreaks usually triggered by stress and coinfection with other chronic diseases common.

Diagnosis

Specimens and samples

- Full post-mortem samples

Testing

- PCR
- Post-mortem findings

Control and Prevention

Unlikely to cause significant disease in the wild. Monitoring and surveillance of nestling and fledgling mortality events.

References

Hulbert, C. L., et al. (2015). "Survey of captive parrot populations around Port Phillip Bay, Victoria, Australia, for psittacine beak and feather disease virus, avian polyomavirus and psittacine adenovirus." Aust Vet J **93**(8): 287-292.
Ladds, P. (2009).Viral diseases in birds in Pathology of Australian native wildlife, Csiro Publishing.
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Avian poxviruses

There are many poxviruses, affecting most vertebrate species; all bird species are susceptible. The avian poxviruses form the genus Avipoxvirus, which consists of 10 defined species and many more unclassified isolates (60 unique strains have been identified). Avipoxviruses have traditionally been placed in three genetic clades (fowlpox, canarypox and psittacinepox), with several subclades. More recently additional clades, some with one species only have been suggested.

They are generally thought to be quite host-specific and endemic in many populations where they cause mild disease. However, there are examples of exotic poxviruses establishing in new populations and species, where they may be responsible for more significant disease. These include a likely role in Hawaiian forest bird declines (avian poxviruses introduced in 1800s) and epizootic impacts on native birds in Galapagos, Falkland Islands and the Canary Islands. Consequently, there are some question marks around the host specificity of some of these viruses. It is, however, not thought to be zoonotic – and no descriptions of infection in mammals were found.

Poxvirus has been reported from a large range of native Australian species, including ones occurring in Victorian wetlands. While Australia is not thought to have psittacinepox, more recently, a novel poxvirus from Crimson Rosellas has been described which may belong to a separate clade.

Most records of Australian avipoxvirus are from captive animals (including sea- and waterbirds, passerines and raptors), but records from wild birds include little eagle, magpie-larks, Australian magpie, Australian kestrel, collared sparrowhawk, red wattlebird, raven, crimson rosella, black currawongs, silvereye (all WHA Factsheet), and pacific shearwaters (Sarker et al., 2017). There are likely to be many undetected cases in wild birds.

Clinical Signs and Pathology

Most infections, especially in endemic situations are mild and recovered birds remain immune to infection with the same strain for up to a year.

The clinical presentation is usually one of:

- **large nodules (dry form),** primarily on unfeathered body regions such as the legs and feet or the face; sometimes the nodules may extend into the nasal or oral cavity. Facial nodules may obstruct vision. The nodules may ulcerate and bleed, and sometime result in secondary bacterial infection or flystrike; this may lead to more severely ill birds showing signs of depression and loss of body condition.
- However, more rarely lesions may extend into the oesophagus and/or trachea **(wet form)**; this usually occurs after aerosol infection and can cause more severe clinical signs including septicaemia and difficulty breathing. The septicaemic form results in lesions in the lung and may also cause damage to the heart.

Pathology:

- Epithelial hyperplasia, vacuolation and necrosis
- Superficial secondary infection with bacteria or fungi
- Multiple discrete, pale yellow/cream, raised necrotic lesions on mucosa
- Rhinitis and tracheitis

- Bronchopneumonia

Transmission and Epidemiological Factors

The virus may be transmitted in a number of ways, including:

- Insect vector
- Direct contact with infected individual or contaminated object (but won't penetrate intact skin)
- Inhalation

The period from infection to clinical signs may vary from 4 days to three weeks, depending on the viral species and host. Avipoxviruses may persist for long periods in the environment and in dried scabs. Susceptibility to disinfectants is variable, depending on the virus species/strain.

Diagnosis

Sampling and specimens:

- Biopsy affected lesions and submit
- Complete necropsy, collect tissues from affected lesions
- Fresh or frozen tissues and formalin-preserved

Testing

- PCR
- Electron microscopy
- Cell culture
- Immunohistochemistry
- Serology (detects antibodies, so may not be specific for current infection)

Control & Prevention

Vaccines are available against some avipoxvirus strains. Chickenpox, pigeonpox, turkeypox and canarypox virus strain vaccines are commercially available – and have been used in a range of species with variable success.

Insecticidal sprays may be used to control vectors.

References

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Coronavirus

Coronaviruses (CoV) cause a wide range of diseases in various species around the world. Some significant cross-species spillovers have occurred (e.g. SARS, MERS). Infectious bronchitis is an important coronavirus of domestic poultry but is not closely related to the CoV detected in wild birds in Australia.

Clinical Signs & pathology

None known in wild birds

Diagnosis
PCR in surveillance studies.

References
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Herpesvirus

Avian herpesviruses are widespread and numerous. Only the most important herpesviruses are covered here. The following are general characteristics of avian herpesviruses. See below for specific notes on individual species.

Clinical Signs & pathology
The majority of infections are sub-clinical. Clinical disease is usually seen in immunocompromised birds or with a herpesvirus infection affecting an unfamiliar species.

Clinical signs

- Sudden death
- Respiratory distress
- Lethargy
- Leg weakness (geese)
- Conjunctivitis (finches)

Pathology

- Inflammation and necrosis of the respiratory tract (raptors and finches)
- Focal necrosis of liver and spleen
- Haemorrhage and necrosis of gastrointestinal tract
- Inclusion bodies may be found in lesions

Transmission and Epidemiological factors
Transmission is by close contact and inhalation of infected dust, crop milk transmission and predation on infected birds. Raptors are most likely to be infected after eating infected pigeons.

Infection results in lifelong latent infections with intermittent shedding related to stress. Disease events are often triggered by stress.

Diagnosis
Specimens and samples

- Tissue samples (fixed, fresh and frozen)
- Pharyngeal swabs

Testing

- PCR

Control and Prevention
Avoiding the feeding of pigeons to raptors.

Vaccination has been suggested for captive populations, however, concerns regarding ongoing shedding and therefore infection of in-contact birds exist.

Surveillance and identification of outbreaks and new species affected will give a better understanding of the epidemiology and significance of the diseases.

Psittacine herpesvirus

Psittacine herpesvirus 1 (PsHV-1) is also known as Pacheco's disease or internal papillomatous disease. Psittacine herpesvirus 3 (PsHV-3) has been shown to cause similar disease to PsHV-1. There are no reports of psittacine herpesvirus in wild birds in Australia. However, PsHV-1 and PsHV-3 have been identified in captive birds in Australia and Australian native species held in captivity overseas. It mainly affects psittacines, however rare infections have been recorded in passerines.

Columbid herpesvirus

Feral rock pigeons have been identified as the most likely reservoir in Australia and generally show no signs of disease. Outbreaks of clinical disease with pigeon herpesvirus have been seen in black swans and Cape Barren geese in Victoria and in several raptor species in Victoria and New South Wales. Disease is also seen in racing pigeons.

Goose herpesvirus

A novel herpesvirus was identified to have caused a large outbreak in domestic geese in Queensland in 1989. It was initially thought that the causative agent was duck viral enteritis (DVE) virus (see below) but was later confirmed to be a novel herpesvirus.

Duck viral enteritis (DVE)

DVE (or duck plague) is exotic to Australia. It causes large outbreaks and mortality events in domestic poultry and wild birds overseas. Disease is seen in Anatids, however other waterbird species can carry the virus.

Australia's lack of international migratory duck and goose species may reduce the risk of incursion; however, the range and movement of host species does pose a risk which has not been quantified.

Clinical signs:

- Photophobia
- Increased thirst
- Diarrhoea
- Drooping plumage and partially closed eyes
- Ataxia
- Tremors
- Sudden death
-

Pathology:

- Internal haemorrhage and vascular damage
- Lymphoid lesions
- Degeneration of parenchymatous organs

Diagnosis:

- Necropsy findings
- Inclusion bodies in gastrointestinal tract and liver
- Viral isolation

A vaccine is available and used extensively in domestic poultry overseas.

Surveillance is especially important for an exotic disease. Any outbreaks where DVE is a differential should be fully investigated to arrive at a diagnosis. This may include viral genome sequencing to classify it if it is a novel virus (such as in the case of the goose herpesvirus outbreak in Queensland).

References

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Wildlife Health Australia and D. Phalen (2017). Psittacine Herpesvirus and Papillomatosis in Australian Birds. WHA Fact sheet**:** 9 pp.

Infectious bursal disease

Infectious bursal disease or Gumboro disease is caused by infectious bursal disease virus (IBDV). It is primarily a disease of domestic poultry; however, it has been detected in wild birds across a wide range of geographic areas and species.

Clinical Signs & pathology

No clinical signs have been reported in wild birds.

Transmission and Epidemiological factors

Transmission dynamics between domestic and wild birds have not been described.

Diagnosis

Specimens and samples

- Serum
- Bursal tissue

Testing

- Serology with microneutralisation test
- PCR

Control and Prevention

None recommended.

References

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Wilcox, G. E., et al. (1983). "Infectious bursal disease in Western Australia." Aust Vet J **60**(3): 86-87.
Wildlife Health Australia, et al. (2017). Infectious Bursal Disease. WHA Fact sheet**:** 5 pp.

Proventricular dilatation disease

Proventricular Dilatation Disease (PDD) is thought to be caused by avian bornaviruses (ABVs) and is mainly a disease of parrots, however passerines, waterfowl and gulls can also be infected. To date there have been no positives found in wild birds in Australia, however it is present in captive birds. Waterfowl populations in North America are known to be infected with an unknown prevalence.

Clinical Signs & pathology

Most birds will show no clinical signs.

Central nervous system disease

- Progressive ataxia
- Progressive weakness and paralysis
- Rarely seizures and blindness

Neurological digestive disease

- Reduced gut motility – passing whole seeds
- Diarrhoea
- Delayed crop emptying
- Weight loss and emaciation

Pathology

- Distended proventriculus and ventriculus +/- dilatation of intestines
- Enlarged myenteric nerves
- Non suppurative encephalomyelitis
- Enlargement and lymphoplasmacytic infiltration of the myenteric nerves +/- peripheral nerves
- Inflammation of cardiac nerves
- Lymphoplasmacytic infiltration of the adrenals

Transmission and Epidemiological factors

Ingestion and possibly aerosol infection. There is some evidence of vertical infection.

Incubation periods vary from weeks to years and birds may never develop disease. Birds may be lifelong shedders in oral secretions, faeces and urine as well as in feathers.

Diagnosis

Specimens and sampling

- Blood
- Droppings
- Oral swabs
- Feathers
- Crop biopsy
- Tissue samples of all tissues with both fixed and frozen brain, ventriculus and proventriculus

Testing

- PCR (repeat testing if bird alive to improve specificity)
- Serology
    - Western blot assay
    - ELISA
- Viral isolation

- Immune histochemistry (not available in Australia)

Control and Prevention

Surveillance is important to further research and classify this disease. There are currently no formal surveillance programs.

References

Doneley, R., et al. (2007). "Proventricular dilatation disease: an emerging exotic disease of parrots in Australia." Aust Vet J **85**(3): 119-123.
Doneley, R. and D. Phalen (2013). Avian Bornavirus and Proventicular Dilatation Disease. WHA Fact sheet**:** 12 pp.
Weissenbock, H., et al. (2009). "Avian bornaviruses in psittacine birds from Europe and Australia with proventricular dilatation disease." Emerg Infect Dis **15**(9): 1453-1459.

Psittacine Beak and Feather Disease (PBFD)

Psittacine Beak and Feather Disease is caused by a circovirus which is endemic to Australia; it appears to have spread globally from here through the exotic bird trade.

It is primarily a disease of psittacine birds (i.e. parrots and cockatoos), but recently, high prevalence of infection has also been found in a range of non-psittacine species (without associated clinical signs), such as tawny frogmouth, laughing kookaburra, sacred kingfisher, hardhead duck (captive), southern boobook, powerful and barn owls, white ibis, brown goshawk, Australian magpie and Australian raven. Nevertheless, clinical signs of PBFD have been described in some of those species (laughing kookaburra, columbids, corvids, raptors, finches, rainbow bee-eaters and powerful owls).

The disease is listed as a Key Threatening Process under the EPBC Act 1999 and is thought to cause a significant threat to the remaining populations of orange bellied parrot, swift parrot and Norfolk Island green parrot.

Clinical Signs and Pathology

Not all infected birds develop disease, even among species where disease prevalence is high. Disease may be peracute, acute or chronic, depending on age of infection. Peracute disease is usually seen in neonates, acute disease in fledglings. Chronic disease is seen in adult birds or fledglings infected after complete feather development but before their first moult.

Clinical signs

Peracute disease:

- Septicaemia
- Pneumonia
- Enteritis
- Rapid weight loss
- Death

Acute disease:

- Depression
- Diarrhoea
- Crop stasis
- Feather abnormalities
- Death

Chronic disease:

- Loss of powder down
- Beak overgrowth and oral ulceration
- Feather loss
- Abnormal feather development (retention of feather sheath, haemorrhage in pulp, deformed curled feathers, constriction at base of feathers)

- Immune suppression and secondary infection

Pathology:

- Hepatic necrosis and pancreatitis
- Small spleen (chronic infection)
- Feather lesions
- Widespread inclusion bodies
- Enlarged liver, atrophy or shrunken thymus or bursa (peracute infections)
- Secondary infection (other viruses, bacteria, parasites etc)

Transmission and Epidemiological Factors

It is thought that infection resulting in disease, especially in older fledglings and adult birds, requires very high doses of virus, which may account for high prevalence of infection in some wild bird populations (one study found seroprevalence of 41 – 93% in wild cockatoo populations, with few birds showing clinical signs). Acute disease leading to death is most common in birds less than 2 years of age.

Incubation may be as short as 3 weeks, but maximum incubation period could be more than 6 months (period from infection to next moult). Virus is shed in nest hollows through feather dander, crop secretions, bile and faeces. In addition, it is thought that the virus can be transmitted vertically. Infection may be thus be through:

- Ingestion
- Intra-cloacal uptake
- Within the egg

Infected birds may shed massive amounts of virus. Offspring from birds with protective antibody levels (i.e. have been infected by not succumbed to disease) may have temporary immunity to the disease protecting them from infection during the nestling and fledgling stages. Similarly, flocks which have no longer been exposed to the virus (e.g. in captive breeding program), may be more susceptible to the virus when introduced. Captive birds may also be more susceptible than wild birds.

Surveys of captive psittacines in the Port Phillip Bay area showed a prevalence of infection by PCR of 30.5% (n=118) across a range of native and exotic species. Only 5.5% of the PBFD positive birds (n=36) showed any clinical signs.

Non-psittacine birds, which appear generally to be relatively resistant to the development of disease, may act as reservoirs for more susceptible psittacine species. Some psittacine species may also act as shedding reservoirs but rarely succumb to disease themselves, such as rainbow lorikeets or Eclectus parrot. Gang gang cockatoos, on the other hand, are highly susceptible to the acute form of the disease.

Diagnosis

Sampling and specimens:

- Feathers
- Blood (in EDTA)

Testing

- PCR – detects virus
- Haemagglutination assay (HA) – measures virus

- Haemagglutination Inhibition Assay (HI) – measures antibody

Control & Prevention

PBFD readily persists in the environment and can survive temperatures of up to 80 degrees Celsius for at least 30 minutes. Contact with peroxygen for at least 10 minutes is an effective disinfectant.

There is no effective treatment for PBFD; however, work on a vaccine is ongoing.

References

Amery-Gale, J., Marenda, M. S., Owens, J., Eden, P. A., Browning, G. F., and Devlin, J. M. (2017). A high prevalence of beak and feather disease virus in non-psittacine Australian birds. *Journal of Medical Microbiology* **66**, 1005-1013. doi: doi:10.1099/jmm.0.000516.
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Quaranjaviruses

Quaranjaviruses are part of Orthomyxoviridae. Several novel viruses have been detected and described in birds, humans and ticks. Mass mortality events of ducks in the United States have been linked to Wellfleet Bay virus(WFBV) and disease events in domestic ducks in South Australia detected Cygnet River virus (CyRV). Quaranfil virus has been diagnosed in humans without a known animal host and Johnston Atoll virus has been detected in seabirds and ticks in Australia and New Zealand.

Clinical Signs & pathology

Clinical signs

- Sudden death
- Incoordination
- Respiratory distress
- Diarrhoea
- Emaciation

Concurrent parasite burdens are common.

Pathology (WFBV)

- Coagulative necrosis of pancreas, spleen and intestines, most severe in liver

Pathology description of CyRV cases are likely complicated by concurrent Salmonellosis lesions.

- Necrotising fibrinous enteritis
- Multifocal piecemeal hepatitis
- Fibrinous multifocal splenitis

Transmission and Epidemiological factors

Multiple mortality events on Cape Cod, US have occurred in common eiders with some events numbering up to 3000 birds.

Most disease events in the US occur in late summer or early autumn, likely linked to arrival of migratory birds.

One mass mortality event of CyRV has been reported in South Australia. It occurred in domestic Muscovy ducks and several factors may have contributed to the outbreak including salmonellosis and a change in feeding regime.

Prevalence and significance of Quaranjaviruses in Australia are unknown.

Diagnosis

Specimens and samples:

- Whole carcasses should be collected for necropsy.
- Serum

Testing

- Microneutralisation assays have been used to detect antibodies to WFBV
- PCR for Quaranfil virus is available and may be useful for detection of CyRV

Control and Prevention

Surveillance recommended, especially in mortality events.

References

Allison, A. B., et al. (2015). "Cyclic avian mass mortality in the northeastern United States is associated with a novel orthomyxovirus." J Virol **89**(2): 1389-1403.
Ballard, J. R., et al. (2017). "PREVALENCE AND DISTRIBUTION OF WELLFLEET BAY VIRUS EXPOSURE IN THE COMMON EIDER (SOMATERIA MOLLISSIMA)." J Wildl Dis **53**(1): 81-90.
Doherty, R., et al. (1969). "Isolation of viruses from Ornithodoros capensis Neumann from a tern colony on the Great Barrier Reef, North Queensland." Australian J Sci.
Kessell, A., et al. (2012). "Cygnet River virus, a novel orthomyxovirus from ducks, Australia." Emerg Infect Dis **18**(12): 2044-2046.
Shearn-Bochsler, V., et al. (2017). "Experimental Infection of Common Eider Ducklings with Wellfleet Bay Virus, a Newly Characterized Orthomyxovirus." Emerg Infect Dis **23**(12): 1958-1965.

Rotavirus

Avian rotaviruses cause significant disease in domestic poultry. They have been identified in a wide range of wild birds. Most infections tend to be subclinical, however there has been one reported widespread outbreak of a novel rotavirus in pigeons in Australia.

Clinical Signs & pathology

The majority of infections are likely to be subclinical.

Clinical signs:

- Regurgitating
- Diarrhoea
- Sudden death

Pathology

- Hepatomegaly with congestion or mottled appearance
- Hepatocellular dissociation, necrosis and macrophage infiltration
- Splenomegaly with friable and mottled appearance
- Splenic histiocytosis

Transmission and Epidemiological factors

Coinfection with other enteric pathogens is common in domestic birds.

Wild birds are likely to have a low prevalence although large surveys have not been undertaken in Australia.

There is no evidence that the pigeon rotavirus outbreak affected native pigeon species, although feral pigeons did show signs of disease and mortality.

Diagnosis

Specimens and samples

- Full necropsy
- Liver and splenic tissue

Testing

- PCR

Control and Prevention

Pigeon deaths should be monitored to further describe the epidemiology of rotaviruses in wild birds.

References

McCowan, C., et al. (2018). "A novel group A rotavirus associated with acute illness and hepatic necrosis in pigeons (Columba livia), in Australia." PLoS One **13**(9): e0203853.
Ursu, K., et al. (2011). "Monitoring of group A rotaviruses in wild-living birds in Hungary." Avian Dis **55**(1): 123-127.

BACTERIAL

Borreliosis

Borrelia bacteria is a tick-borne pathogen and causes disease in humans and other mammals overseas. The most important of these (Lyme borreliosis) is not present in Australia.

Borrelia spp. have been found in birds overseas and may have a role as transport host of ticks, however the significance as a natural reservoir and to the epidemiology of the disease is unknown.

References

Chalada, M. J., et al. (2016). "Is there a Lyme-like disease in Australia? Summary of the findings to date." One Health **2**: 42-54.
Wildlife Health Australia (2016). Borrelia in Australian Wildlife. WHA Fact sheet**:** 8 pp.

Botulism

Botulism is caused by a toxin produced by *Clostridium botulinum*. *C. botulinum* spores are resistant in the environment and common in soil and aquatic environments. Invertebrates such as maggots can act as accumulators from carcasses. Avian botulism is present throughout the world and regularly causes bird mortality events.

Clinical Signs & pathology

Clinical signs

- Progressive weakness, paralysis, inability to fly
- Flaccid paralysis
- Third eyelid protrusion
- Neck weakness
- Death by respiratory muscle paralysis, drowning, predation or starvation

No specific pathology. Maggots may be found in the gastrointestinal tract.

Transmission and Epidemiological factors

While all birds may be affected by it, waterfowl are the most predisposed. In Australia botulism is seen mainly in Anseriformes.

Botulism is mainly seen in the summer months in Australia

Individuals may die of either botulism or other disease and this can trigger a botulism outbreak as maggots from carcasses accumulate more toxin and are eaten. Concurrent disease and death events may trigger an outbreak by increasing the number of carcasses present. Changes in water quality, level and contamination can precipitate an outbreak by promoting toxin production and clostridial growth as well as changing feeding habits.

Botulism can have a big effect on water bird populations overseas with mass mortality events of hundreds of thousands of birds in North America. Unlike in North America, outbreaks in Australia haven't occurred in breeding flocks, which changes the methods of control used.

Type C, C/D and E have caused bird mortality events around the world, but there is no evidence of Type E in Australia.

Diagnosis

Definitive diagnosis is difficult and often a presumptive diagnosis is made based on the species, clinical signs, ruling out other diseases on necropsy (where possible) as well as local factors such as outbreaks and current conditions. Non-infectious disease causes such as blue-green algae toxicity as well as other contaminants are common differential diagnoses.

Specimens and samples:

- Blood samples
- Gut contents
- Full post-mortem

Detection of toxin is possible from blood and gut samples however false negatives in serum are common and a positive from ingesta doesn't prove causation. Detection of *C. botulinum* is not enough evidence.

- Capture ELISA (cELISA) binding toxins may have high false negative rates in serum.
- PCR for Type C or D

Toxin detection tests are not currently available in Victoria.

Botulism (including suspected cases) is notifiable.

Control and Prevention

Avoidance of stagnant water bodies, excess organic matter and warmer water temperatures may reduce the risk of a botulism outbreak but may not be possible in natural water bodies and coastlines.

Carcass disposal is always important to minimise the number of maggots and reduce the risk of triggering a botulism outbreak. It is very important during an outbreak in order to reduce ongoing transmission.

Once an outbreak has been detected, moving birds off the affected area may help to reduce the number of cases (as long as it is not a breeding ground).

References

Brandis, K., et al. "Avian-botulism risk in waterbird breeding colonies and implications for environmental water management." Marine and Freshwater Research.
Galvin, J. W., et al. (1985). "An outbreak of botulism in wild waterbirds in southern Australia." J Wildl Dis **21**(4): 347-350.
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Rocke, T. E. and T. K. Bollinger (2007). Avian botulism. Infectious diseases of wild birds**:** 377-416.
Wildlife Health Australia (2019). Botulism in Australian Wild Birds. WHA Fact sheet**:** 7 pp.
Wildlife Health Australia (2019). Diagnosis of Avian Botulism in Australia. WHA Fact sheet**:** 4 pp.
Woodall, P. F. (1982). "Botulism outbreak in waterbirds at Seven-Mile Lagoon in south-east Queensland." Wildlife Research **9**(3): 533-539.

Chlamydiosis

Chlamydiosis, caused by *Chlamydia psittaci* is a zoonotic bacterial disease that causes significant disease in domestic poultry, humans (called psittacosis) and some wild birds around the world. The severity of disease caused in birds depends on the strain of bacteria and the susceptibility of bird species. All species of wild birds in Australia are assumed to be susceptible, although disease has been most frequently described in psittacines.

Clinical Signs & pathology

Most wild birds do not show signs of disease.

Clinical signs

- Conjunctivitis
- Rhinitis
- Depression
- Diarrhoea

Pathology

- Pulmonary congestion
- Pleural and pericardial fibrinous exudate
- Splenomegaly and necrosis
- Cardiomegaly with fibrin plaques and yellow exudate
- Hepatomegaly and necrosis with adherent fibrin

Transmission and Epidemiological factors

Wild birds are thought to have lower prevalence than captive birds in Australia, although pigeons in Europe have been shown to have high prevalence. Species which congregate in large numbers to roost or on water habitat are possibly at higher risk.

Transmission pathways in wild birds are unknown but likely to be via inhalation or ingestions, possibly with insect vectors playing a role. *C. psittaci* causes persistent infection and shedding in subclinical infections. Outbreaks of clinical disease are generally triggered by stress.

Diagnosis

Care must be taken when sampling given the zoonotic risk.

Specimens and samples

- Swabs from conjunctiva, choana and cloaca
- Fixed and fresh liver, splenic and lung tissue

There is no definitive test and diagnosis ideally requires a combination of serology and bacterial detection.

- ELISA – unknown characteristics in most species
- PCR

Control and Prevention

Avoidance of artificial close contact of birds (e.g. feeding stations) may help to reduce transmission.

Care should be taken to minimise human exposure of both staff and the general public if a large-scale outbreak has been detected.

References

Amery-Gale, J., et al. (2019). "Surveillance for Chlamydia spp. with multilocus sequence typing analysis in wild and captive birds in Victoria, Australia." J Wildl Dis.

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Blomqvist, M., et al. (2012). "Chlamydia psittaci in Swedish wetland birds: a risk to zoonotic infection?" Avian Dis **56**(4): 737-740.
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Enteric Pathogens

Clostridial enteritis or necrotising enteritis

Clostridial species may cause disease death after ingestion of toxins or production of toxins in the gastrointestinal tract. Avian clostridial enteritis causes disease outbreaks in domestic poultry and wild birds.

Clinical Signs & pathology

Clinical signs

- Sudden death
- Depression
- Regurgitation of clear fluid
- Distended abdomen
- Diarrhoea
- Dehydration
- Weakness, unable to fly, neck outstretched

Pathology

- Swelling of intestines with red/brown fluid and gas
- Pale yellow diphtheritic membranes, small and discrete or larger and confluent
- Often intestinal perforations have occurred leading to peritonitis

Transmission and Epidemiological factors

Cases in wild birds may be sporadic or cluster in outbreaks. Outbreaks are often associated with feeding stations and other areas resulting in poor food hygiene. Carbohydrate overload from human food sources may predispose to disease. Outbreaks in Australia have mostly been seen in lorikeet species and king parrots; however, waterfowl outbreaks are seen overseas.

Juvenile birds are at higher risk and it is most commonly seen in winter.

Diagnosis

Concurrent bacterial infections are common and may make definitive diagnosis difficult.

Specimens and samples

- Necropsy
- Intestinal tissues

Testing

- Toxin identification by immunohistochemistry or immunofluorescence

Control and Prevention

Avoidance of situations which promote poor hygiene (such as bird feeders).

References

Asaoka, Y., et al. (2004). "Fatal necrotic enteritis associated with Clostridium perfringens in wild crows (Corvus macrorhynchos)." Avian Pathology **33**(1): 19-24.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
McOrist, S. and R. L. Reece (1992). "Clostridial enteritis in free-living lorikeets (Trichoglossus spp.)." Avian Pathol **21**(3): 503-507.
Wobeser, G. and D. Rainnie (1987). "Epizootic necrotic enteritis in wild geese." J Wildl Dis **23**(3): 376-385.

Campylobacteriosis

Campylobacter spp. cause gastrointestinal disease in many mammalian species, including humans. Clinical disease in wild birds is rare, but occasional outbreaks in captive birds have occurred in Australia.

Clinical Signs & pathology

Most infections are subclinical.

Clinical signs

- Diarrhoea, sometimes haemorrhagic
- Green urates
- Nestling deaths

Pathology

- Necrotic enteritis
- Hepatomegaly with yellow necrotic foci and irregular focal haemorrhages below the capsule
- Necrotising hepatitis

Transmission and Epidemiological factors

Campylobacter in wild birds is primarily a concern due to the zoonotic nature of the disease. Outbreaks of severe disease have been confined to captive native birds.

Diagnosis

- Culture
- PCR

Control and Prevention

None required.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.

Edwardsiella

Edwardsiella is a zoonotic enteric pathogen that may cause disease in domestic poultry, reptiles, amphibians and fish. Wild birds in Australia have not been reported to show clinical signs, however there are reports of clinical disease in a pelican overseas and in rockhopper penguins in Chile.

Clinical Signs & pathology

Pathology

- Reddening and red fluid of jejunal mucosa
- Pulmonary haemorrhage

Diagnosis

Specimens and samples

- Intestinal tissue and contents
- Full necropsy

Testing

- Bacterial culture

- PCR

Control and Prevention
None recommended

References
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.

Escherichia albertii

E. albertii is an emerging pathogen of humans and birds that has been found all over the world. Reports of *E. albertii* infection have increased since its discovery, likely due to increased sampling and differentiation from other species.

Clinical Signs & pathology
Infection may be subclinical or acutely cause death.

Acute onset sepsis and death is likely in birds showing clinical disease.

Pathology

- Often no gross lesions and few microscopic lesions.
- Necrotising proventriculitis
- Acute heterophilic enteritis

Transmission and Epidemiological factors
E. albertii has been found in most bird orders as well as mammals (including humans) worldwide. In Australia, there are reports in passerines, psittacines and poultry with high prevalence in some species (e.g. 40% in Australian magpies in Canberra). No surveys of seabirds and waterfowl have been undertaken in Australia.

Transmission is faecal-oral.

Mortality events in birds have only been recorded in European and North American finches.

Diagnosis
Specimens and samples

- Faecal and cloacal swabs
- Intestinal contents
- Liver and splenic tissue

Testing

- Culture
- PCR

Control and Prevention
Avoidance of situations which promote poor hygiene (such as bird feeders).

Surveillance to improve understanding of presence and epidemiology. There have been no reported mortalities in Australia, however it should be considered where there are mortality events.

References
Berrell, E. and D. Phalen (2013). Escherichia albertii in Birds in Australia. WHA Fact sheet**:** 8 pp.
Gordon, D. M. (2011). "Reservoirs of infection: the epidemiological characteristics of an emerging pathogen, Escherichia albertii." Department of Agriculture, Fisheries and Forestry. Division of Ecology, Evolution and Genetics.

Gordon, D. M. and A. Cowling (2003). "The distribution and genetic structure of Escherichia coli in Australian vertebrates: host and geographic effects." Microbiology **149**(12): 3575-3586.
Oaks, J. L., et al. (2010). "Escherichia albertii in wild and domestic birds." Emerg Infect Dis **16**(4): 638-646.
Oh, J. Y., et al. (2011). "Epidemiological investigation of eaeA-positive Escherichia coli and Escherichia albertii strains isolated from healthy wild birds." J Microbiol **49**(5): 747-752.

Escherichia coli

E. coli can cause disease in humans, domestic animals and wild birds but is also found as normal gastrointestinal flora in many mammals. It generally causes sporadic infections in wild birds. The pathogenicity of *E. coli* varies between strains.

Clinical Signs & pathology

Acute colisepticaemia

- Visceral hyperaemia, swelling and haemorrhage, especially liver, subcutis and joints
- Reticuloendothelial hyperplasia
- Focal necrosis of liver, spleen, lymphoid tissue

Subacute fibrinopurulent serositis

- Fibrinopurulent polyserositis
- Airsacculitis

Chronic granulomatous pneumonitis, hepatitis and enteritis

- Usually birds that have survived acute disease
- Fibrotic lungs

Transmission and Epidemiological factors

E. coli is often opportunistic and may occur with concurrent diseases. Even pathogenic strains may be carried without causing clinical disease.

Diagnosis

Specimens and samples

- Faeces
- Intestinal tissue and content

Testing

- Bacterial culture
- PCR
- Isolation of *E. coli* is not enough to assume pathogenicity. Virulence markers may need to be identified for complete assessment.

Control and Prevention

Surveillance for *E. coli* strains of significance in humans and other animals as well as antimicrobial resistance is recommended.

References

Dolejska, M., et al. (2016). "High prevalence of Salmonella and IMP-4-producing Enterobacteriaceae in the silver gull on Five Islands, Australia." J Antimicrob Chemother **71**(1): 63-70.
Gordon, D. M. and A. Cowling (2003). "The distribution and genetic structure of Escherichia coli in Australian vertebrates: host and geographic effects." Microbiology **149**(12): 3575-3586.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Oh, J. Y., et al. (2011). "Epidemiological investigation of eaeA-positive Escherichia coli and Escherichia albertii strains isolated from healthy wild birds." J Microbiol **49**(5): 747-752.

Salmonellosis

Many strains of Salmonella may cause disease in different species. Mass mortality events have occurred due to *S.* Typhimurium DT160 in North America, Europe, New Zealand and Australia. Tasmania saw mass mortalities in sparrows in 2013 along with human cases and subsequent cases in domestic animals. In 2016 there was an outbreak in Melbourne that affected house sparrows as well as spotted turtle doves. Sporadic cases are also seen around the world. *S.* Typhimurium is also a zoonotic pathogen that can cause disease in humans, domestic animals and wildlife.

Clinical Signs & pathology

Clinical signs

- Sudden death
- Foetid watery diarrhoea
- Anorexia
- Polydipsia

Pathology

- Pale, swollen liver
- Red and large spleen
- Multifocal necrosis with lymphocytic and plasmacytic infiltration
- Coagulative necrosis of the brain stem
- Caseous granulomas of the crop
- Localised peritonitis

Transmission and Epidemiological factors

Transmission is by the faecal-oral route with infected animals shedding via their faeces. Some birds may continue to shed without showing clinical signs.

Disease can be exacerbated or triggered by stress and concurrent disease such as candidiasis and oil toxicosis.

Diagnosis

Specimens and samples

- Faeces
- Necropsy
- Tissue samples from spleen, liver, intestines, crop – fixed and fresh
- Intestinal contents

Testing

- Bacterial culture
- PCR
- Phage typing or MLVA

Control and Prevention

Outbreaks may centre around areas of poor food hygiene, such as feeding stations. Therefore, these should be avoided or kept clean.

Good personal hygiene for humans in contact with birds or investigating an outbreak.

Surveillance of disease events especially related to house sparrow mortality events is indicated.

References

Dolejska, M., et al. (2016). "High prevalence of Salmonella and IMP-4-producing Enterobacteriaceae in the silver gull on Five Islands, Australia." J Antimicrob Chemother **71**(1): 63-70.
Grillo, T., et al. (2017). "Salmonella isolated in two mass moralities of house sparrows." Animal Health Surveillance Quarterly Report **21**(3).

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Maute, K., et al. (2019). "Clean bill of health? Towards an understanding of health risks posed by urban ibis." Journal of Urban Ecology **5**(1): juz006.
McOrist, S. and C. Lenghaus (1992). "Mortalities of little penguins (Eudyptula minor) following exposure to crude oil." Vet Rec **130**(8): 161-162.
Wildlife Health Australia (2018). Salmonella Typhimurium DT160 in House Sparrows in Australia. WHA Fact sheet**:** 9 pp.

Erysipelas

Erysipelothrix rhusiopathiae is a zoonotic bacterial disease of concern in domestic pigs and poultry. Wild birds may play a role as reservoirs; however, humans are rarely infected from wild birds.

Clinical Signs & pathology

Clinical signs

- Weakness and depression
- Diarrhoea
- Incoordination and flaccid paralysis
- Paresis
- Sagging necks
- Dyspnoea
- Injected mucous membranes
- Acute death

Pathology

- Septicaemia
- Multifocal haemorrhages throughout abdomen, serosa
- Hepatomegaly and splenomegaly
- Petechiation of kidneys, liver, heart
- Gram-positive bacilli in blood vessels

Transmission and Epidemiological factors

E. rhusiopathiae has been found in a range of bird species, including a little penguin, cockatoo and welcome swallow.

Most clinical disease cases described in Australia were in emu and malleefowl.

E. rhusiopathiae is found in livestock effluent and survives for a long time in the environment.

Diagnosis

Specimens and samples

- Tissue from liver and spleen – fixed and fresh
- Impression smears from liver and spleen

Testing

- Identification from impression smear
- Culture
- PCR

Control and Prevention

Suspected larger outbreaks should result in carcass collection to reduce contamination.

References

Eamens, G. J., et al. (1988). "Serotypes of Erysipelothrix rhusiopathiae in Australian pigs, small ruminants, poultry, and captive wild birds and animals." Aust Vet J **65**(8): 249-252.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.

Munday, B. L. (1972). "A serological study of some infectious diseases of Tasmanian wildlife." J Wildl Dis **8**(2): 169-175.

Mycobacteriosis/ Avian tuberculosis

Mycobacteriosis in birds can be caused by *Mycobacterium avium* subsp. *avium* (MAA) and *M. genavense* (MG), with some other species also being capable of causing disease. It causes sporadic disease that is progressive and eventually fatal. A wide range of bird species, including domestic poultry may be affected.

Clinical Signs & pathology

Clinical signs depend on what tissues the infection is present in.

- Very slow progression
- Chronic wasting disease, often with pectoral muscle atrophy
- Delayed moults and poor feathering
- Granuloma in various locations
- Depending on location, dyspnoea and abdominal distension can result

Pathology – vary depending on location of granulomas

- Peritoneal effusion
- Cobblestone mucosa of intestine
- Hepatomegaly or splenomegaly with raised pale foci/nodules
- Granulomas in lungs, air sacs
- Amyloidosis of the liver

Transmission and Epidemiological factors

Both species of *Mycobacterium* are found in birds throughout the world. In Australia most reports in birds have been in domestic or captive birds. There have, however, been several cases in free-living birds in Victoria (and around Australia).

MAA has only been found in birds, with no other environmental or animal host identified. In contrast, MG may infect mammals and grow in the environment. *Mycobacterium spp.* can survive for long periods in the environment with MG also contaminating water.

Transmission is likely via ingestion or inhalation via soil, water or infected prey. Captive birds are at higher risk and show higher prevalence, possibly due to increased population density and poorer hygiene.

Diagnosis

Specimens and samples

- Serum
- Full necropsy

Testing

- Characteristic necropsy and histology findings
- Acid fast stain of impression
- Serology
- Bacterial culture
- Intradermal skin testing (may be of little value in wild birds)
- PCR

- Compliment fixation assay
- ELISA
- Western blot assay

Control and Prevention

Surveillance of wild birds to improve the understanding of prevalence and epidemiology of the diseases.

Contact between wildlife and domestic livestock and poultry should be avoided. Any wastewater entering the environment should have adequate testing and treatment.

References

Hodge, P. J., et al. (2019). "Avian mycobacteriosis in captive brolgas (Antigone rubicunda)." Aust Vet J **97**(3): 81-86.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Reece, R. L., et al. (1992). "Some unusual diseases in the birds of Victoria, Australia." Vet Rec **130**(9): 178-185.
Wildlife Health Australia (2013). Mycobacteriosis in Australian Birds. WHA Fact sheet**:** 11 pp.

Pasteurellosis

Pasteurellosis, also known as avian cholera, is caused by *Pasteurella multocida.* Different strains of *P. multocida* cause disease in many vertebral species, including humans. Although classed as a zoonotic disease, avian pasteurellosis rarely causes human disease other than wound infections.

Clinical Signs & pathology

Clinical signs

- Sudden death
- Good body condition
- Diarrhoea
- Mucoid discharge from nostrils and beak
- Neurological disturbances including convulsions

Pathology

- Multifocal hepatitic necrosis
- Granulomatous hepatitis
- Acute septicaemia
- Enteritis
- Haemorrhage of heart and gizzard
- Nasal discharge
- Thick yellow fluid in distal intestine

Transmission and Epidemiological factors

Waterfowl, other waterbirds and scavenging bird species are most often affected in disease events. In the United States there have been increasing mass mortality events in the last fifty years caused by pasteurellosis, some killing tens of thousands of waterbirds. China and European countries have also reported mass mortality events.

It is transmitted via close contact, inhalation or ingestion. The bacteria are shed in all bodily secretions, especially in faeces. Birds that recover are considered to have life-long infections and continue to shed. Environmental contamination of soil and water caused by carcasses is likely the most important source of infection.

P. multocida may also occur as an opportunistic pathogen with a more chronic disease course.

Pasteurellosis causes disease in domestic poultry and chicken, turkey and duck farms in Victoria (and around Australia) have been affected. The impact of wild bird disease on captive birds is poorly

described; however, transmission dynamics are likely impacted by the presence of bacteria in both free-range poultry and wild bird populations.

Diagnosis

Specimens and samples

- Whole carcasses
- Tissue samples, especially liver, intestine and heart – fresh and fixed
- Wings of decomposed carcasses

Testing

- Bacterial culture

Control and Prevention

Surveillance to identify outbreaks early in the disease course and to further describe the epidemiology in wild birds in Australia is recommended.

During a confirmed or suspected outbreak, carcasses should be removed to avoid increasing environmental contamination. Vaccination has been used in the US in wild birds, however, is not generally practical.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Pedersen, K., et al. (2003). "Pasteurella multocida from outbreaks of avian cholera in wild and captive birds in Denmark." J Wildl Dis **39**(4): 808-816.
Samuel, MD, Botzler, RG, Wobeser, GA (2007) Avian Cholera. In 'Infectious diseases of wild birds.' (Eds NJ
Thomas, DB Hunter, CT Atkinson.) pp. 239-269. (Blackwell Publishing: Ames, Iowa).
Wang, C., et al. (2009). "An outbreak of avian cholera in wild waterfowl in Ordos wetland, Inner Mongolia, China." J Wildl Dis **45**(4): 1194-1197.
Wildlife Health Australia and P. L. Whiteley (2015). Pasturellosis in Australian waterbirds. WHA Fact sheet**:** 6 pp.
Whiteley, P., personal communication

Staphylococci and Streptococci

Staphylococcus spp. and *Streptococcus* spp. are normal gastrointestinal flora of some bird species. They can cause disease in similar ways, often triggered by stress or opportunistically.

Clinical Signs & pathology

Wounds on feet may develop into pododermatitis or infectious arthritis.

Sudden death from septicaemia may result from stress or secondary to severe pododermatitis.

Lesions depend on nature of infection and any concurrent illness.

Transmission and Epidemiological factors

Staphylococcus spp. and *Streptococcus spp.* cause sporadic disease that is often opportunistic and related to other concurrent disease.

Diagnosis

Specimens and samples

- Tissue samples from affected organs

Testing

- Bacterial culture

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.

Yersiniosis

Yersiniosis is caused by *Yersinia pseudotuberculosis* and, more rarely, *Y. enterocolitica.* The following focuses on *Y. pseudotuberculosis.* Yersiniosis is found in wild and captive birds throughout the world and can cause human disease.

Clinical Signs & pathology

Yersiniosis may present as acute sudden death or subacute/chronic.

Acute disease:

- Sudden death
- Transient diarrhoea

Pathology

- Acute septicaemia
- White-yellow foci in liver, spleen and lung (small, <1mm)
    - Necrotic foci containing coccobacilli

Subacute/chronic disease:

- Dyspnoea
- Weakness
- Lameness
- Diarrhoea (variable)

Pathology

- Focal lesions in organs larger, discrete or confluent
    - May become hard and yellow
    - Resembles mycobacteriosis
- Variable microscopic pyogranulomas in oesophagus, intestine, lung, heart, bone marrow

Transmission and Epidemiological factors

It is assumed that wild birds are a reservoir for *Y. pseudotuberculosis*, however surveys of wild birds from various countries have shown only a low prevalence. This may be due to difficulties in isolating the organism, however the definitive epidemiology and role of wild birds is not currently known.

Disease is normally sporadic but may cause larger mortality events, especially after heavy rainfall and in winter. Concurrent disease such as enteric parasitosis is common.

Diagnosis

Specimens and samples

- Faecal sample
- Full necropsy
- Tissue samples from liver, spleen +/- other affected organs – fixed and fresh

Testing

- Bacterial culture
- PCR

Control and Prevention

Surveillance, especially during mortality events is recommended.

References

Cork, S., et al. (1995). "The role of wild birds and the environment in the epidemiology of Yersiniae in New Zealand." N Z Vet J **43**(5): 169-174.
Elmberg, J., et al. (2017). "Potential disease transmission from wild geese and swans to livestock, poultry and humans: a review of the scientific literature from a One Health perspective." Infect Ecol Epidemiol **7**(1): 1300450.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Otsuka, Y., et al. (1994). "Isolation of Yersinia pseudotuberculosis from city-living crows captured in a zoo." J Vet Med Sci **56**(4): 785-786.

FUNGAL

Aspergillosis

Aspergillosis is a respiratory disease of captive and wild birds as well as other animal species and humans. The majority of disease in birds is caused by *Aspergillus fumigatus.* It is present on all continents except Antarctica.

Clinical Signs & pathology

In domestic poultry an acute form of aspergillosis is seen in chicks and ducklings after being placed in contaminated brooders. In wild birds the chronic form of disease is seen.

Clinical signs

- Respiratory signs
  - Sneezing
  - Coughing
  - Nasal discharge
  - Respiratory rales and dyspnoea
- Lethargy
- Emaciation and stunting
- Yellow faeces
- Ocular lesions may be seen
- Neurological signs may occur in late stages

Pathology

- White, yellow/green or black plaques and nodules in respiratory tract
  - Large nodules may be caseous and necrotic
- Abdominal, hepatic and renal lesions may occur (similar appearance)
- Vasculitis

Transmission and Epidemiological factors

Aspergillosis is more common in captive birds, but cases do occur in wild birds. Mass mortality events have been reported in North America and New Zealand; however, it is usually a sporadic disease. Concurrent disease and stress predispose to aspergillosis.

The fungal spores are present in the environment and cause disease when inhaled. There is no direct transmission between animals. Wild birds feeding on agricultural wastage such as mouldy silage or straw are at higher risk.

Diagnosis

Samples and specimens

- Full necropsy
- Affected tissues – fixed and fresh

Testing

- Morphological identification
- Fungal culture
- PCR

Control and Prevention
Surveillance to identify outbreaks and monitor underlying causes of stress and disease is important.

References
Friend, M. and (Ed.) (1987). Field Guide to Wildlife Diseases - General Field Procedures and Diseases of Migratory Birds. Washington, DC, National Wildlife Health Center, Fish and Wildlife Service - United States Department of the Interior.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Obendorf, D. L. and K. McColl (1980). "Mortality in little penguins (Eudyptula minor) along the coast of Victoria, Australia." J Wildl Dis **16**(2): 251-259.

Candidiasis

Candidiasis is a yeast infection caused by *Candida albicans.* It is considered a gastrointestinal commensal organism in birds and is found in the environment worldwide. Strains vary in their pathogenicity and some species of bird may be more predisposed to clinical disease.

Clinical Signs & pathology
Clinical signs

- Depression
- Anorexia and weight loss
- Diarrhoea with whole seeds in droppings (species dependant)
- Crop stasis
- Regurgitation

Pathology

- White streaks or yellow/white necrotic plaques in upper gastrointestinal tract
    - May involve intestines
- Thick and rough crop epithelium
- Superficial invasion of mucosa of fungal pseudohyphae
- Granulomas in muscle, liver, heart, kidneys

Transmission and Epidemiological factors
Infections are mainly opportunistic, and disease is usually triggered by immune disturbance, concurrent disease or changes in normal flora. As such, disease is more common in captive birds.

Although an opportunistic pathogen, some strains carry virulence factors that make them more pathogenic. It is classed as a zoonotic disease, although most disease in humans is also from commensal species.

Clinical disease has been reported in wild birds and is often associated with increased carbohydrate feeding, poor hygiene, concurrent disease such as oil toxicosis or stress events. Reports of clinical disease are increasing. There is a report of an outbreak of fungal disease (including candidiasis) in vulture and eagle species nestlings in Spain associated with feeding on livestock carcasses that contained antibiotic residues.

Diagnosis
Samples and specimens

- Faecal sample
- Oral swabs
- Necropsy and histopathology of lesions

Testing

- Faecal smears may show budding yeast or germ tube forms
- Fungal culture

Control and Prevention

Avoidance of artificial feeding situations that may result in poor food hygiene. Detection of large amounts of wild birds with signs of candidiasis should be investigated for an underlying disease source causing stress.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Pitarch, A., et al. (2017). "Oral mycoses in avian scavengers exposed to antibiotics from livestock farming." Sci Total Environ **605-606**: 139-146.
Rippon, R. J., et al. (2010). "Candida albicans infection in free-living populations of hihi (stitchbird; Notiomystis cincta)." N Z Vet J **58**(6): 299-306.

Cryptococcosis

Cryptococcosis is a fungal disease caused by *Cryptococcus neoformans* var. *grubii* or *C. bacillisporus.* There are limited reports in wild birds but have been detected as causing clinical disease as well as asymptomatically.

Clinical Signs & pathology

Disease may be primary or opportunistic.

Clinical signs

- Proliferative lesions of the beak and nares
- Stridor
- Dyspnoea
- Dysphagia
- Ocular discharge
- Weakness
- Muscle atrophy
- Diarrhoea
- Paresis/paralysis
- Blindness

Pathology

- Gelatinous upper respiratory tract lesions
- Copious cryptococcus fungal bodies
- Pathology seen overseas:
    - Invasive and disseminated disease of lung, air sacs, brain

Transmission and Epidemiological factors

Infections reported globally are primarily opportunistic, however, in Australia it is thought that there is more primary disease.

Particular species of eucalyptus are associated with disease in Australia.

Concerningly, *Cryptococcus spp.* isolated from wild birds in Malaysia found a high percentage of antimicrobial resistance which may be of significance to humans.

Diagnosis

Specimens and samples

- Necropsy

Pathology

- Histopathology of lesions
- Morphological identification
- Fungal culture

Control and Prevention

None recommended.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Lord, A. T., et al. (2010). "Multidrug resistant yeasts in synanthropic wild birds." Ann Clin Microbiol Antimicrob **9**: 11.
Malik, R., et al. (2003). "Avian cryptococcosis." Medical Mycology **41**(2): 115-124.

Proventricular disease – *Macrorhabdus*

Proventricular disease is caused by *Macrohabdus ornithogaster* (previously known as Megabacteria). It is a significant disease of captive birds, also known as avian gastric yeast and may infect domestic poultry. It is present in wild birds in Australia, but details of the epidemiology and significance are unknown.

Clinical Signs & pathology

Clinical disease has not been identified in wild birds although there has been a suggestion of seasonal outbreaks in Queensland and Western Australia. These have not been further described.

Transmission and Epidemiological factors

Macrorhabdus is present in captive and domestic birds across much of the world (no reports from Africa) but has only been identified in wild birds in Europe and Australia. It has been found in a variety of species but is mainly seen in psittacines and passerines in captivity.

Transmission is most likely faecal-oral and asymptomatic birds shed the organism in their faeces.

Diagnosis

Identification of *Macrorhabdus* on faecal smears, however care should be taken that it is not confused with bacterial species.

Control and Prevention

Surveillance to enable better description of the epidemiology.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Sutherland Saines, A. and D. Phalen (2013). Macrorhabdus (Megabacteria) in Wild Birds in Australia. WHA Fact sheet**:** 10 pp.

INTERNAL PARASITES

PROTOZOA

Coccidiosis

A large amount of coccidian species across *Eimeria* and *Isospora/Atoxoplasma* genera have been identified in wild birds in Australia and internationally. The clinical significance of most of these infections is unknown.

Clinical Signs & pathology

Clinical **intestinal coccidiosis** is mainly seen in captive birds, but some cases in wild birds have been recorded.

- Ill thrift
- Anorexia
- Regurgitation
- Death
- Diarrhoea, melena or haemorrhagic

Pathology

- Haemorrhagic enteritis
- Varying necrosis of intestines
- Splenomegaly
- Coccidial organisms present in mucosa

Hepatic coccidiosis has been reported in a magpie lark.

- Hepatomegaly with small white foci
- Oocysts present in bile ducts

Renal coccidiosis may be an incidental finding but is known to cause disease in short-tailed shearwater nestlings. It may also cause disease opportunistically.

- Weight loss
- Muscle atrophy
- White granular excrement around vent

Pathology

- Pale large kidneys
- Yellow discolouration of fat
- Large intestinal impaction
- Coccidial organisms present in ureters

Atoxoplasmosis is a disease seen in introduced birds in Australia caused by *Atoxoplasma (Isospora) spp.* There are no reports of clinical disease in native birds.

Clinical signs

- Abdominal enlargement
- Increased hunger
- Inappetence
- Loss of balance

Pathology

- Granulomas in spleen
- Hepatomegaly and splenomegaly

- Pale streaking of ventricular myocardium with monocytic-macrophage infiltrates
- Lymphoplasmacytic enteritis (sparrows)

Transmission and Epidemiological factors

Passerines are mostly affected by atoxoplasmosis overseas with fledglings having the most severe disease and mortalities.

Coccidial disease in wild birds is rare but is often associated with concurrent disease when it occurs. The effect on populations is unknown.

Transmission is via the faecal-oral route.

Diagnosis

Samples and specimens

- Faecal samples or cloacal swabs
- Full necropsy

Testing

- Morphological identification

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Obendorf, D. L. and K. McColl (1980). "Mortality in little penguins (Eudyptula minor) along the coast of Victoria, Australia." J Wildl Dis **16**(2): 251-259.
Terio, K. and M. Adkesson (2019). Systemic Isosporosis in Passerine Birds**:** 454-458.

Cryptosporidiosis

Cryptosporidium spp. cause disease in a wide range of bird and mammal species, including humans. Clinical disease is mainly seen in captive and domestic birds, but wild bird disease has been reported around the world. *C. baileyi* and *C. meleagridis* are the species most likely to cause disease in birds.

Clinical Signs & pathology

Respiratory disease

Clinical signs

- Sudden death
- Convulsive sneezing
- Rales
- Mucoid discharge
- Coughing
- Conjunctivitis

Pathology

- Petechiae in trachea
- Creamy deposits in upper respiratory tract
- Airsacculitis
- Mottled liver
- Congested, grey/red lungs
- Splenomegaly
- Loss of cilia
- Bursal atrophy

- Presence of organisms in respiratory tract and conjunctiva

Gastrointestinal disease

Clinical signs

- Weight loss
- Diarrhoea
- Inappetence
- Decreased pigmentation

Pathology

- Muscle atrophy
- Villous atrophy and fusion
- Intestinal distension with gas and fluid
- Presence of organisms in brush border

Transmission and Epidemiological factors

The zoonotic potential of avian *Cryptosporidium spp.* has not been well described, although *C. parvum* is responsible for most of the human disease burden.

Australian passerine, psittacine and waterfowl species have been reported to have cryptosporidiosis. Concurrent disease is commonly associated with clinical disease and young birds are at higher risk.

Transmission is via the faecal-oral route.

Diagnosis

Specimens and samples

- Full necropsy
- Faecal sample (enteric disease)

Testing

- Morphological diagnosis
- PCR

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Moore, J. E., et al. (2002). "Occurrence of Campylobacter spp. and Cryptosporidium spp. in seagulls (Larus spp.)." Vector Borne Zoonotic Dis **2**(2): 111-114.
Robertson, L. J., et al. (2019). "Are molecular tools clarifying or confusing our understanding of the public health threat from zoonotic enteric protozoa in wildlife?" Int J Parasitol Parasites Wildl **9**: 323-341.

Giardiasis

Giardia duodenalis can cause disease in captive and wild birds as well as mammals, however there are several different genotypes which have species preferences. Reports of clinical disease in wild birds is rare.

Clinical Signs & pathology

Clinical signs

- Stunting
- Weight loss
- Diarrhoea
- Weakness
- Anorexia
- Death

- Depression

Pathology

- Non-specific changes
- Mild focal heterophilic infiltration of small intestinal villi

Transmission and Epidemiological factors

Young birds and those with concurrent disease are more susceptible to giardiasis.

Transmission is via the faecal-oral route and water contamination is common. Humans are most often affected after ingesting contaminated water or contact with waterways.

Diagnosis

Specimens and samples

- Faecal sample
- Intestinal contents

Identified microscopically based on morphology.

References

Cano, L., et al. (2016). "Identification and genotyping of Giardia spp. and Cryptosporidium spp. isolates in aquatic birds in the Salburua wetlands, Alava, Northern Spain." Vet Parasitol **221**: 144-148.
Elmberg, J., et al. (2017). "Potential disease transmission from wild geese and swans to livestock, poultry and humans: a review of the scientific literature from a One Health perspective." Infect Ecol Epidemiol **7**(1): 1300450.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.

Haemosporidia

Haemosporidia are single-celled blood parasites of the genus *Haemoproteus, Plasmodium* or *Leucocytozoon.* The significance and impact of infection on wild birds is unknown, however introduction of a new species into a naïve population has severe consequences. Changes in climate and therefore vector ranges are affecting the distribution of haemosporidia species around the world.

Clinical Signs & pathology

Generally, no signs of disease will occur.

Where pathogenic, clinical signs may vary greatly depending on the body systems affected:

- Neurological signs
- Respiratory distress
- Blindness, intraocular bleeding
- Anaemia +/- stained urates
- Sudden death

Pathology:

- Hepatomegaly
- Haemosporidia in red blood cells
- Schizonts on impression smears of organs
- Haemolysis
- Variation of haemorrhagic, inflammatory and vascular abnormalities

Transmission and Epidemiological factors

All haemosporidia have a life cycle with two hosts: birds and a biting insect species (biting midges, louse flies, mosquitoes and blackflies). *Plasmodium* has a far greater host range than the other two genera. Transmission to birds is via an insect bite.

Co-evolved haemosporidia are not thought to be pathogenic and do not seem to affect the survivability of the infected individual. Birds may be infected with multiple species for long periods of time. Introduction of haemosporidia into new populations may result in clinical disease. Prevalence in Australian studies has varied between 1.7% and 51% in different geographical areas, host and parasite species. A survey of anatids in Lara, Victoria in 1975 found a 7% prevalence.

Climate change and its associated changes in vector populations, temperatures and rainfall patterns may impact on the distribution of haemosporidia in Australia.

Diagnosis

Specimens and samples

- Blood
- Organ tissues (fresh, fixed and frozen)

Testing

- Morphological identification
- Genome sequencing
- PCR

Control and Prevention

Surveillance for clinical disease and changes in prevalence or host species is recommended.

References

Beadell, J. S., et al. (2004). "Prevalence and differential host-specificity of two avian blood parasite genera in the Australo-Papuan region." Mol Ecol **13**(12): 3829-3844.
Bennett, G., et al. (1993). "Avian haematozoa: mortality and pathogenicity." Journal of Natural History **27**(5): 993-1001.
Bennett, G. F., et al. (1977). "Blood parasites of some waterfowl from Victoria, Australia." J Wildl Dis **13**(2): 202-204.
Cannell, B. L., et al. (2013). "The pathology and pathogenicity of a novel Haemoproteus spp. infection in wild Little Penguins (Eudyptula minor)." Vet Parasitol **197**(1-2): 74-84.
Fernandez-Davila, M. A. R. and D. Phalen (2013). Haemosporidia and Australian Wild Birds Dec 2013 (1.2). WHA Fact sheet**:** 11 pp.

Sarcocystosis

There are thought to be a wide range of *Sarcocystis* species that infect birds throughout the world and in Australia in many species of wild bird. The lifecycle involves at least one intermediate host that may be infected via contaminated food or water. The primary host is infected by ingesting infected muscle tissue. The role of birds in the lifecycle is poorly understood, and the severity and presentation of disease depends on whether the bird is acting as a primary or intermediate host and how adapted it is to that species of *Sarcocystis.*

Clinical Signs & pathology

There have been no reports of clinical disease in wild native birds in Australia despite a high prevalence of pathology.

Clinical signs (captive birds and wild birds overseas)

- Sudden death
- Dyspnoea
- Weakness
- Blood in mouth
- Yellow urates
- Neurological signs

- Lethargy
  - Inability to perch
  - Posterior paresis

Pathology

- Elongate white sarcocysts (if primary host)
- Dark red lungs with black foci
- Hepatomegaly
- Splenomegaly with white foci
- Sarcocysts in skeletal muscle +/- myocardium

Transmission and Epidemiological factors

S. falcatula has been the most studied species overseas but Australian species seem to be distinct from it. Although surveys have shown a high prevalence in Australia wild birds, there was no evidence of macroscopic organisms. This is likely due to birds not being the definitive host in Australia.

Transmission to birds is by ingestion of an infected prey species. The cockroach has been implicated as a transport host, but this has not been fully described.

Diagnosis

Specimens and samples

- Necropsy with tissue samples from lesions

Testing

- Morphological identification
- Immunohistochemistry

Control and Prevention

Further surveillance and research to allow better understanding of the epidemiology, life cycle and clinical relevance of the organism.

References

Friend, M. E. (1987). Field Guide to Wildlife Diseases - General Field Procedures and Diseases of Migratory Birds. Washington, DC, National Wildlife Health Center, Fish and Wildlife Service - United States Department of the Interior.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.

Spironucleosis

Spironucleus spp. (formerly known as *Hexamita*) cause disease in wild, captive and domestic birds, fish and mammals. It has been detected in birds in south-eastern Australia and in captive Australian species in the United Kingdom.

Clinical Signs & pathology

Subclinical infection can occur. Of clinically affected animals the mortality rate is high.

Clinical signs

- Emaciation
- Depression
- Diarrhoea, faecal matting
- Death within 1-14 days

Pathology

- Dilated loops of intestine with fluid
- Lymphocytic and plasmacytic enteritis

- Protozoa may be seen in intestinal crypts but often are not

Transmission and Epidemiological factors

Transmission is faecal-orally and situations resulting in poor food hygiene such as feeding stations are associated with disease. Subclinically infected birds may continue to shed the organism.

Young birds are at a higher risk of disease and Australian king parrots are the host species most reported; however, outbreaks have occurred in other psittacine species and *S. columbae* is known to infect pigeons.

The epidemiology and prevalence in wild birds is unknown. Cases are mainly seen in winter.

Diagnosis

Specimens and samples

- Full necropsy
- Faecal sample

Testing

- Morphological identification on faecal wet prep

Control and Prevention

Avoidance of situations which promote poor hygiene (such as bird feeders).

References

Holz, P., et al. (2014). Spironucleosis in Australian Wild Birds. WHA Fact sheet**:** 5 pp.
Philbey, A. W., et al. (2002). "Spironucleosis in Australian king parrots (Alisterus scapularis)." Aust Vet J **80**(3): 154-160.

Toxoplasmosis

Toxoplasmosis caused by *Toxoplasma gondii* is a common mammalian disease thought to be spread in Australia by feral cats. It is less common in birds but does infect captive and wild birds. Reports of clinical disease in wild birds in Australia are sporadic and have occurred in a wide range of species.

Clinical Signs & pathology

Most infections are subclinical.

Clinical signs

- Depression
- Anorexia
- Emaciation
- Diarrhoea
- Pale mucous membranes
- Neurological disturbances
  - Disorientation
  - Convulsions
- Blindness

Pathology

- Cloudy pleura and air sacs
- Yellow thoracic fluid
- Necrotic foci may be present on other organs
- Congested and oedematous lungs with white military necrotic foci
- Crescentic tachyzoites in lesions
- Cysts containing bradyzoites may be present

Transmission and Epidemiological factors

Toxoplasma may be transmitted to birds by ingestion of oocysts from faeces or by ingestion of infected meat. The majority of infections are in captive birds and most are subclinical. Cysts on neurological tissues may be found incidentally during necropsy without there being appreciable clinical signs. In Australia there have been reports of clinical toxoplasmosis in a little penguin in Tasmania, a range of columbids, passerines and psittacines from around the country and a powerful owl in Victoria.

Serological surveys in Europe have found a prevalence of 9% - 31% in waterfowl, although their study designs differed, and some had small sample sizes.

Diagnosis

Specimens and samples

- Serum
- Necropsy
- Tissues from affected organs, especially respiratory tract, liver, spleen and brain – fresh and frozen

Testing

- Histology and morphological identification
- Serology
- PCR

Control and Prevention

None recommended.

References

Dubey, J. (2002). "A review of toxoplasmosis in wild birds." Vet Parasitol **106**(2): 121-153.
Hartley, W. J. and J. P. Dubey (1991). "Fatal toxoplasmosis in some native Australian birds." J Vet Diagn Invest **3**(2): 167-169.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Mancianti, F., et al. (2013). "Toxoplasma gondii in waterfowl: the first detection of this parasite in Anas crecca and Anas clypeata from Italy." J Parasitol **99**(3): 561-563.
Mason, R. W., et al. (1991). "Lethal toxoplasmosis in a little penguin (Eudyptula minor) from Tasmania." J Parasitol **77**(2): 328.
O'Donoghue, P. J. and R. D. Adlard (2000). "Catalogue of protozoan parasites recorded in Australia." Memoirs of the Queensland Museum **45**(1): 1-163.
Reece, R. L., et al. (1992). "Some unusual diseases in the birds of Victoria, Australia." Vet Rec **130**(9): 178-185.

Trichomoniasis

Trichomoniasis is caused by *Trichomonas gallinae* and is found around the world and in Australia. It has caused substantial declines in some bird populations in Europe and North America.

Clinical Signs & pathology

Clinical signs

- Anorexia
- Weight loss
- Yellow lesions of oral cavity and crop
- Depression
- Vomiting
- Dysphagia
- Respiratory distress may occur if lesions block trachea

Pathology

- Yellow caseous lesions in oral cavity, crop, oesophagus

- Necrotic tissue with heterophilic and mononuclear cell infiltrate
- Lesions may also be present in lungs and liver

Transmission and Epidemiological factors

Originally thought to be a disease of pigeons, it now affects a wide range of wild and captive bird species but is still spread mainly by Columbiformes. There are several strains which vary in virulence. Subclinical carriers may occur.

Transmission may be from food or water contaminated with oral fluids, via crop milk or through predation of infected birds. The organism is stable in water and food, surviving for up to several days.

Diagnosis

Specimens and samples

- Oral swab
- Necropsy with tissue samples from lesions – fixed and fresh

Testing

- Morphological identification of organisms under microscopy
- Culture
- PCR

Control and Prevention

Avoidance of feeding stations and other artificial situations that promote poor food hygiene and congregation of birds.

References

Holz, P. and R. Woods (2014). Trichomoniasis in Australian Wild Birds. WHA Fact sheet**:** 5 pp.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Lawson, B., et al. (2012). "The emergence and spread of finch trichomonosis in the British Isles." Philosophical Transactions of the Royal Society B: Biological Sciences **367**(1604): 2852-2863.
Park, F. J. (2011). "Avian trichomoniasis: a study of lesions and relative prevalence in a variety of captive and free-living bird species as seen in an Australian avian practice." Aust Vet J **89**(3): 82-88.

HELMINTHS

"The role of helminths in diseases of native birds is poorly understood and warrants significant attention."(Spratt and Beveridge, 2019)

Cestodes

Cestodes are parasitic organisms that include tapeworms. They have complex lifecycles that generally include one or multiple intermediate hosts which include invertebrates. The majority of cestode infections in wild birds are subclinical and incidental findings. Below are summaries of known disease syndromes and occurrences due to cestodiasis.

Intestinal cestodiasis

Most birds reported to have clinical intestinal cestodiasis also had other concurrent disease, complicating the assessment of clinical significance. Multiple species of cestodes have been reported as part of disease complexes in Australia.

Clinical Signs & pathology

Clinical signs are often nonspecific and dependant on concurrent disease.

- Diarrhoea
- Hypoproteinaemia
- Emaciation
- Depression

Pathology is variable depending on diseases and parasites present.

- Presence of tapeworms
  - Often autolysed
  - Large number of worms may be present
- Haemorrhagic gastritis
- Ulceration and nodules in cloaca

In domestic poultry, intestinal nodules are commonly seen with *Raillietina* sp. infection, but this hasn't been seen in native birds.

Transmission and Epidemiological factors

The significance of cestodiasis without concurrent disease is unclear.

Table 29. Cestode species identified as causing disease in Australian native birds in Australia

Cestode species	**Bird species**
Amoetotaenia sp.	Magpie
Choanotaenia spp.	Blue-faced parrot-finches, Australian raven, Torresian crow, superb lyrebirds, barn owls
Cloacotaenia sp.	Black swans
Davainidae cestode	Magpie, sulphur-crested cockatoo, blue bonnet, black-winged stilts
Dicranotaenia sp.	Pacific black ducks
Dilepididae cestode	Sacred kingfisher
Diorchis thomarsorum	Pacific black ducks
Drepanidotaenia sp.	Black swans
Hemiparonia sp.	Psittacines
Hymenolepid cestodes	Black swans, sacred kingfisher, seagull, pelicans
Idiogenes spp.	Australian bustards
Parnoia sp.	Psittacines
Raillietina spp.	Emus, cassowaries, crested tern, psittacines
Tetrabothrius sp.	Little penguins, Antarctic petrels, short-tailed shearwaters

Diagnosis

Specimens and samples

- Faecal sample
- Necropsy with tissue samples from affected lesions (nodules), ingesta and worms

Testing

- Faecal examination
- Histology

Control and Prevention

As the epidemiology and clinical significance of intestinal cestodiasis is unclear, there should be monitoring of parasites found and potential disease caused.

Sparganosis

Sparganosis is caused by the larval stage of a cestode parasite, usually *Spirometra erinacei* in Australia but other *Spirometra* species may also cause disease. It can infect almost any animal including humans, other mammals, amphibians, reptiles, invertebrates and birds with cats and dogs being the primary hosts. It is widespread around the world and present in domestic and wildlife species in Australia. The prevalence and clinical significance are unknown.

Clinical Signs & pathology

Clinical signs and pathology depend on the migration path of the parasite.

Pathology

- Larvae visible in muscle, viscera or subcutis
- Focal haemorrhage and necrosis
- Granulomatous inflammation

Transmission and Epidemiological factors

Spirometra sp. require two intermediate hosts and one definitive host. Humans acquire the infection by eating an infected intermediate host (such as an insect or bird) and are a dead-end host. Infection with the larval stage in birds is likely a consequence of ingesting an infected invertebrate, amphibian or reptile. The larvae migrate through abdominal viscera, muscle and subcutis.

Diagnosis

Specimens and samples

- Necropsy

The larvae are visible with the naked eye.

Control and Prevention

Control of feral cat and dog numbers as well as the control of owned pets (e.g. limiting access to outdoors for cats) is recommended.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Obendorf, D. L. and K. McColl (1980). "Mortality in little penguins (Eudyptula minor) along the coast of Victoria, Australia." J Wildl Dis **16**(2): 251-259.
Reece, R. L., et al. (1992). "Some unusual diseases in the birds of Victoria, Australia." Vet Rec **130**(9): 178-185.

Nematodes

Nematodes or roundworms are a large group of parasites. Their lifecycles may or may not involve an intermediate host and generally infections in wild birds do not cause clinical disease.

Species noted below are most likely to cause disease in wild birds, but a full list of nematodes identified in birds in Australia has not been provided.

Alimentary tract nematodes

Nematodes may be found in the gastrointestinal tracts of wild birds and are usually incidental findings. Large worm burden may occur, especially in juvenile birds.

Clinical Signs & pathology

Species	Birds	Clinical signs	Pathology
Acuaria spp.	Crows, pelicans, peregrine falcon, finches, mute and black swans, wood duck, chestnut teal, sacred ibis, pied cormorant, Cape Barren goose	• Lethargy • Anorexia • Emaciation • Diarrhoea	Lesions in proventriculus or gizzard. • Ulceration of mucosa • Concurrent *Yersinia* infection common • Granulomatous or fibrous nodules
Capillaria spp. May affect either upper alimentary or intestinal	Range of passerines, psittacines and raptors. Clinical disease uncommon.	• Emaciation • Dehydration • Haemorrhage • Anaemia • Diarrhoea	Upper alimentary: • Tunnels in epithelium or lamina propria • Haemorrhage • Diphtheritic membranes Intestinal: • Dilated intestines • Petechiated mucosa
Contracaecum sp.	Clinical disease noted in pelicans and penguins	• Emaciation • Weakness	• Gastric ulceration and haemorrhage • Chronic ulcers with nodular lesions • Oesophageal inflammation • Marked mucous in proventriculus and gizzard • Worms visible attached to ulcers

Diagnosis

Diagnosis of gastrointestinal nematodiasis relies on necropsy and identification of worms or eggs in lesions and faecal examination. Worms should be visible in affected gastrointestinal lumens but may autolyse or be difficult to see due to size.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
McKenzie, R. A., et al. (1982). "Dieldrin poisoning and botulism in Australian pelicans (Pelecanus conspicillatus)." Aust Vet J **58**(4): 148-152.
McOrist, S. and C. Lenghaus (1992). "Mortalities of little penguins (Eudyptula minor) following exposure to crude oil." Vet Rec **130**(8): 161-162.
Obendorf, D. L. and K. McColl (1980). "Mortality in little penguins (Eudyptula minor) along the coast of Victoria, Australia." J Wildl Dis **16**(2): 251-259.
Reece, R. L., et al. (1992). "Some unusual diseases in the birds of Victoria, Australia." Vet Rec **130**(9): 178-185.

Central nervous system nematodes

Angiostrongylus

Angiostrongylus cantonensis is endemic to the east coast of Australia with Sydney being the most southern report. The natural reservoir of *A. cantonensis* (or rat lungworm) is introduced rats, however a native species (*A. mackerrasae)* is found in native rats with a wider geographical range (including Tasmania). While *A. cantonensis* has a wide range of primary and intermediate hosts, including humans, *A. mackerrasae* has not been reported as zoonotic.

Clinical Signs & pathology

Neural angiostrongyliasis causes

- Depression
- Muscle wasting
- Ataxia, ascending paresis and limb paralysis
- Lumbar hyperalgesia

Pathology:

- Eosinophilia
- Eosinophilic meningoencephalitis (brain and spinal cord)
- Larval nematodes
- Wallerian degeneration of white matter

Transmission and Epidemiological factors

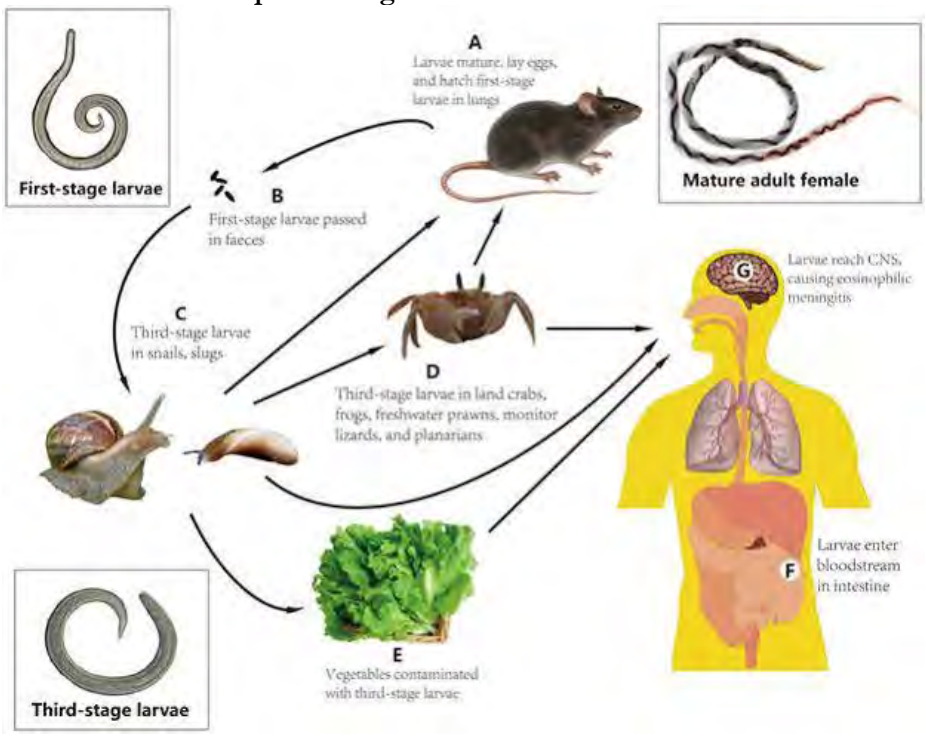

Figure 10. Angiostrongyliasis transmission cycle (credit: http://www.antimicrobe.org/b028.asp).

Tawny frogmouths in Sydney have had a high prevalence for unknown reasons.

Diagnosis

Specimens and samples

- Fresh brain and CNS (frozen or fixed)

Testing

- Histology of CNS
- No current serology or PCR tests available for animals
- Recovery of worms
- Faecal testing not useful as accidental hosts don't pass first-stage larvae and *Angiostrongylus* can't be differentiated from other lungworm species.

Control and Prevention

Surveillance is important to track any changes in the geographical distribution of the parasite.

In captive environments a snail, slug and rodent control program is important.

References

Ma, G., et al. (2013). "Tawny frogmouths and brushtail possums as sentinels for Angiostrongylus cantonensis, the rat lungworm." Vet Parasitol **192**(1-3): 158-165.

Monks, D. J., et al. (2005). "Angiostrongylus cantonensis as a cause of cerebrospinal disease in a yellow-tailed black cockatoo (Calyptorhynchus funereus) and two tawny frogmouths (Podargus strigoides)." J Avian Med Surg **19**(4): 289-293.

Montali, R., et al. (2004). Cerebrospinal Angiostrongylus cantonensis in tawny frogmouths in Sydney, Australia. Proceedings of the WDA (Australasian section) Annual meeting, Kinchega National Park, NSW, Australia, Kinchega National Park.

Prociv, P. and M. S. Carlisle (2001). "The spread of Angiostrongylus cantonensis in Australia." Southeast Asian journal of tropical medicine and public health **32**: 126-128.

Reece, R., et al. (2013). "Neuroangiostrongyliasis due to A ngiostrongylus cantonensis in gang-gang cockatoos (C allocephalon fimbriatum)." Aust Vet J **91**(11): 477-481.

Spratt, D. M. (2015). "Species of Angiostrongylus (Nematoda: Metastrongyloidea) in wildlife: A review." International Journal for Parasitology: Parasites and Wildlife **4**(2): 178-189.

Spratt, D. M. and I. Beveridge (2019). "Wildlife parasitology in Australia: past, present and future." Australian Journal of Zoology **66**(4): 286-305.

Spratt, D., et al. (2019). Angiostrongyliasis and Australian Wildlife. WHA Fact sheet**:** 8.

Respiratory tract nematodes

Syngamus trachea

Syngamus trachea, also known as gape worm, causes respiratory tract disease in infected birds. It may cause clinical disease but has also been found incidentally.

Clinical signs & Pathology

- Dyspnoea
- Sneezing
- Wheezing and moist rales
- Coughing
- Head shaking
- Difficulty feeding
- Sudden death from tracheal occlusion

Pathology

- Worms in cranial trachea
- Congested tracheal mucosa

Transmission and Epidemiological factors

Invertebrates such as earthworms act as paratenic hosts and larvae remain encapsulated until ingested by a bird. Significant disease occurs in domestic poultry.

Juvenile birds are more susceptible to disease.

Diagnosis

Specimens and samples

- Necropsy

Testing

- Morphological identification of worms

Control and Prevention
None recommended.

References
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Reece, R. L., et al. (1992). "Some unusual diseases in the birds of Victoria, Australia." Vet Rec 130(9): 178-185.

Trematodes

Trematodes or flukes are parasites that can infect a range of body systems and various species are found all over the world. Subclinical infections are common but clinical disease has been reported in a range of wild birds in Australia. It is often difficult to attribute significant disease as trematodiasis often occurs with concurrent parasite infections and other disease.

Alimentary trematodiasis

There have been few definitive reports of alimentary trematodiasis in wild birds in Australia. Overseas it has been associated with mortalities in wild birds and there have been occurrences of clinical disease in domestic ducks in New South Wales.

Clinical signs & Pathology
- Weight loss
- Depression
- Diarrhoea

Pathology

- Severe enteritis
- 1mm flukes attached to mucosa

Hepatic and biliary trematodiasis

Trematodiasis from infection with *Mawsonotrema eudyptulae* and *Renicola* sp. has been reported to cause mortalities of little penguins. *Renicola* sp. are normally found as subclinical renal trematode infections but have been reported as causing aberrant disease.

Clinical signs & Pathology
- Emaciation
- Abdominal distention

Pathology

Mawsonotrema eudyptulae

- Ascites
- Hepatomegaly
- Oedematous thickening of air sacs
- Occasional hepatic rupture
- Flukes visible
- Loss of normal architecture and tracts throughout liver with minimal normal hepatic tissue

Renicola sp.

- Brown or white nodules in liver
- Cystic dilations of bile ducts containing flukes and debris
- Chronic nodules are necrotic cores surrounded by inflammatory cells

Diagnosis

Specimens and samples

- Necropsy with tissue samples from affected organs

Testing

- Morphological identification on fresh preps or histology

Control and Prevention

None recommended

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Obendorf, D. L. and K. McColl (1980). "Mortality in little penguins (Eudyptula minor) along the coast of Victoria, Australia." J Wildl Dis **16**(2): 251-259.

EXTERNAL PARASITES

Feather mites

Two species of mites (*Dermanyssus* spp. and *Ornithonyssus* spp.) cause disease through large infestations and loss of blood. Infection is common in wild birds and domestic poultry. It may also infect humans. Other mites also known as feather mites only feed on feather debris and are not pathogenic.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.

Knemidocoptiasis

Knemidokoptes (or *Cnemidokoptes*) spp. are also known as scalyleg mites and cause disease in captive, domestic and wild birds. It has been reported in passerines and psittacines throughout Australia.

Clinical signs & Pathology

- Hyperkeratosis and crusting of beak, ceres, eyelids
  - Feet often involved
  - Primary feathers may be involved
- Inability to perch
- Dyspnoea

Pathology

- Hyperkeratosis
- Mites visible in lesions

Diagnosis

Specimens and samples

- Skin scrape
- Tissue samples from lesions on necropsy

Testing

- Mites are visible on microscopy

Control and Prevention

Avoidance of feeding stations and other artificial situations that promote congregation of birds and increase the risk of disease transmission.

References

Holz, P. H., Beveridge, I. & Ross, T. 2005. Knemidocoptes intermedius in wild superb lyrebirds (Menura novaehallandiae). Aust Vet J, 83, 374-5.
Jaensch, S. M., et al. (2003). "Knemidocoptes intermedius in a wild currawong (Strepera graculina)." Aust Vet J **81**(7): 411.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Vogelnest, L. (2003). "Knemidocoptes intermedius in a wild currawong (Strepera graculina)." Aust Vet J **81**(10): 597.

Lice and Flies

Many species of louse and fly may infest wild birds. Most infections will occur in individuals debilitated by other disease. Infestations may cause irritation or worsen already ill health.

Ticks and Fleas

Many species of tick and flea occur in wild birds. Infestations may be subclinical, cause mild irritation or signs of disease related to loss of blood, leading to anaemia, weakness and sometimes death. External parasites (especially ticks) may act as vectors for a range of viruses, bacteria and parasites.

UNKNOWN CAUSES

Rainbow lorikeet paralysis

Rainbow lorikeet paralysis or clenched claw syndrome has been described in eastern Australia. The cause is unknown despite investigation.

Clinical Signs & pathology

Clinical signs worsen over one to two weeks

- Head tilt
- Uni- or bilateral flexed hocks with clenched feet, resulting in inability to perch, strange gait and resting on hocks
- Intention tremor
- Collapse and death (rarer)

Pathology

- No significant gross lesions
- Non-suppurative inflammation of cerebellum (white matter), caudal brainstem, spinal cord
- Wallerian degeneration of the spinal cord and peripheral nerves

Transmission and Epidemiological factors

Rainbow and scaly-breasted lorikeets have been affected, although it is mainly seen in rainbow lorikeets. It has only been reported in New South Wales, Queensland and Victoria; there are no international reports.

Cases are generally sporadic and can occur year-round but may be clustered in spring and summer. Juveniles may be more commonly affected, but the disease is seen in adults as well. Birds are often found near feeding stations.

The disease may cause mortality; however, some birds adapt to resting on hocks but are at higher risk of predation, trauma and starvation. There are few reports of birds recovering in captivity and being rereleased into the wild.

The disease has been thought to be caused by lead toxicity, thiamine deficiency and viral infections. Although affected lorikeets have been shown to have high lead levels, unaffected lorikeets have similar results.

Diagnosis

Specimens and samples

- Full necropsy
- Tissue samples from central and peripheral nervous system – fixed and frozen

A wide range of tests may be applicable as the underlying cause is unknown.

Control and Prevention

Avoidance of feeding stations and other artificial situations that promote poor food hygiene and congregation of birds, although a link to this syndrome has not been found.

References

Holz, P. (2014). Rainbow Lorikeet Paralysis. WHA Fact sheet**:** 5 pp.
McOrist, S. and R. A. Perry (1986). "Encephalomyelitis In Free-Living Rainbow Lorikeets (Trichoglossus Haema Todus)." Avian Pathol **15**: 783-789.

"Black and white" neurological syndrome

A neurological syndrome seen in black and white birds mainly in New South Wales with an unknown cause. Mass mortality events have been recorded.

Clinical Signs & pathology

Clinical signs

- Alert and responsive
- Dyspnoea
- Paresis
- Lack of righting reflex and inability to fly
- May have diarrhoea
- Normal PLR, withdrawal reflexes and cloacal tone
- Death usually within 24 hours.

Pathology

- Dehydration
- Haemorrhage of epicardial and gastrointestinal tract
- Multisystemic perivascular inflammation
- Non-suppurative encephalitis

- Possibly hydropericardium

Transmission and Epidemiological factors

Two mass mortality events have been described in New South Wales in 2003 and 2005-06 with sporadic cases reported at other times. Disease mainly occurs in Australian magpies, pied currawongs and Australian ravens; however, some other species have shown similar signs during mortality events.

The syndrome is thought to be viral or parasitic, but no causative agent has been isolated. The accepted case definition is "Birds of the species Australian magpie (*Gymnorhina tibicen*), pied currawong (*Strepera versicolour*) or Australian raven (*Corvus coronoides*) from the coastal areas of NSW (and possibly other regions) with neurological signs (inability to fly, paresis, retaining mental alertness) progressing rapidly to death and with histopathological findings of non-suppurative encephalitis."(Wildlife Health Australia, 2017a)

Diagnosis

Specimens and samples

- Full necropsy
- Tissue samples – fixed and frozen

A wide range of testing may be applicable to rule out differentials and find source of disease.

- Viral culture
- PCR

References

Wildlife Health Australia (2017). Neurological Syndrome in Black and White Birds. WHA Fact sheet: 6 pp.

NON-INFECTIOUS DISEASES

Toxins

Heavy metals

Lead

Lead can cause acute and chronic toxicity of wild birds and has been responsible for mass mortality events in Victoria, other Australian states and other countries.

Clinical Signs & pathology

Exposure to high levels of lead causes rapid and acute death. Chronic exposure to low levels can result in a variety of more subtle clinical signs.

- Sudden death
- Neurological signs
    - Altered mentation
    - Loss of balance
    - Seizures
- Digestive neurological signs
    - Anorexia
    - Vomiting
- Watery droppings with green/yellow urates
- Oedema of head and neck (waterfowl)

- Leg weakness

Pathology

Non-specific and dependent on secondary problems

- White and swollen kidneys
- Pale streaked myocardium
- Lead objects in stomach
- Schwann cell degeneration

Transmission and Epidemiological factors

Environmental contamination is caused by a range of sources of lead. Mine sites, refineries and the areas surrounding their infrastructure (including transport) may have high levels of contamination. Other industries and urban settings may see lead paint, petrol and dust entering water and soil. Hunters using lead shot contribute to lead toxicity in two ways: bullets being left in the environment and animals being shot with lead bullets being left (either dead or injured). Lead ammunition is illegal in many areas, but there may be a large accumulation of old shot, especially in waterways that can be exposed with changing water levels. Lead accumulates in animals and is exacerbated higher up the food chain. Many areas have high lead concentrations due to long term contamination, for example from road run-off from lead fuel additives.

All species of bird are susceptible to lead toxicity; however, waterfowl are at a higher risk due to their feeding behaviour. Predators such as birds of prey and seabirds are at risk if they feed on animals with high lead concentrations or containing lead shot.

Diagnosis

Specimens and samples

- Blood sample
- Full necropsy
- Tissue samples from liver, kidney and bone

Testing

- Blood lead levels

Control and Prevention

Environmental health precautions regarding lead contamination.

Mass mortality events should be investigated, other differential diagnoses should be ruled out and environmental sampling should take place if there is likely a new source of lead.

References

Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
Sweet, N. and D. Phalen (2014). Lead Poisoning in Australian Birds. WHA Fact sheet**:** 15 pp.
Wickson, R., et al. (1992). "Concentrations of lead in bone and other tissues of Victorian waterfowl." Wildlife Research **19**(2): 221-231.

Insecticides, herbicides and other pesticides

Multiple classes of chemicals are used in the control of vertebrate, invertebrate, plant and fungal species deemed pests. Wild birds may be affected through direct poisoning during a pest control program, secondary toxicity from eating poisoned animals or plants or environmental contamination through the use, misuse and disposal of the chemicals.

Control and Prevention

For the prevention of all pesticide toxicities in wild birds, all baiting and pest control programs should be carefully designed to avoid secondary poisoning. Chemicals that are stable in the environment should be avoided.

Anticoagulants

Anticoagulants are generally used for rodent control. First generation anticoagulants require multiple feeds to cause death and are not likely to cause disease when the carcass is eaten by another animal. Second generation toxins have a much more toxic and can cause secondary toxicity.

Clinical Signs & pathology

Clinical signs

- Haemorrhage from mouth or other orifices
- Wounds

Pathology

- Pallor of tissues
- Free blood in body cavities

Transmission and Epidemiological factors

Toxicity usually results from wild birds eating poisoned prey animals but can occur from primary ingestion as well. As such birds of prey are at higher risk as they predate many species that are actively targeted by pest control (rodents, rabbits).

Mortalities tend to be seasonal in line with increased rodent numbers and therefore increased control measures and baiting.

Diagnosis

Specimens and samples

- Blood (from living bird)
- Necropsy

Testing

- Prolonged clotting times
- High performance liquid chromatography (HPLC)

References

Grillo, T. (2011). "Poisoning events in wildlife." Animal Health Surveillance Quarterly Report **16**: 8-9.
Grillo, T., et al. (2014). "Australian Wildlife Health Network Report." Animal Health Surveillance Quarterly Report **18**: 5-7.
Grillo, T. and L. Post (2010). "Australian Wildlife Health Network Report." Animal Health Surveillance Quarterly Report **15**: 5-8.

Organophosphates (and carbamates)

Organophosphates (OPs) can cause acute toxicity in a vertebrates and invertebrates but degrade rapidly in the environment. Carbamates are a similar class but are less commonly used.

Clinical Signs and pathology

- Excessive salivation
- Ataxia
- Regurgitation
- Diarrhoea

- Dyspnoea
- Tremors, convulsions
- Death within hours or recovery within two days

Pathology

No specific lesions

- May show signs of
    - Pulmonary oedema
    - Haemorrhagic gastro/enteritis

Transmission and Epidemiological factors

Toxicity occurs through contact, inhalation or ingestion of the toxin or poisoned plants and animals.

Feral bird species may be targeted as part of a pest control program. Native species may be targeted by illegal deliberate poisoning. Mortality events generally involve hundreds of birds.

Diagnosis

Specimens and samples

- Blood
- Tissue samples from brain, liver, kidneys – fixed and frozen (stored in aluminium foil)
- Ingesta – frozen (stored in aluminium foil)

Testing

- AChE activity (not feasible due to cost)
- OP concentrations

Persistent Organic Pollutants (POPs)

Persistent Organic Pollutants (POPs) include a large number of chemical groups such as dieldrin, DDT, DDE, dioxins and many others. Toxicity is seen in all vertebrates and may have chronic health effects at low levels.

Clinical Signs & pathology

Clinical signs

- Eggshell thinning and reduced chick viability (chronic toxicity)
- Sudden death
- Behavioural changes
- Weakness with inability to fly
- Head tilt
- Dilated pupils
- Leg extension
- Hyper-excitability and convulsions when stimulated

Pathology

- Muscle pallor
- Small liver
- Emaciation
- Concurrent infection with nematodes and cestodes common

Transmission and Epidemiological factors

POPs are slow to degrade in the environment and cause long-term contamination, however levels in Australia a slowly reducing. Many POPs have been banned in Australia for several decades.

The chemicals accumulate in fat and organs and animals higher on the food chain bioaccumulate and therefore have higher levels. Signs of toxicity are often seen after a period of stress when the bird has metabolised fat stores, which liberate the stored toxins.

Birds are exposed via inhalation, contact or ingestion.

Diagnosis

Specimens and samples

- Full necropsy
- Tissue samples of liver, brain, fat, ingesta – fixed and frozen (stored in aluminium foil)

Testing

- Mass spectrometry
- HPLC

References

Grillo, T., et al. (2014). "Wild bird poisoning events - fenthion detected." Animal Health Surveillance Quarterly Report **19**(1): 7-9.
Ladds, P. (2009). Pathology of Australian native wildlife, Csiro Publishing.
McKenzie, R. A., et al. (1982). "Dieldrin poisoning and botulism in Australian pelicans (Pelecanus conspicillatus)." Aust Vet J **58**(4): 148-152.
Story, P. and M. Cox (2001). "Review of the effects of organophosphorus and carbamate insecticides on vertebrates. Are there implications for locust management in Australia?" Wildlife Research **28**(2): 179-193.
Wildlife Health Australia (2017). Pesticide Toxicity in Australian Native Birds. WHA Fact sheet

APPENDIX 4. WORKSHOP PARTICIPANTS

Name	Area of expertise	Organisation / Relevant Expertise	Small workshop groups
Alistair Legione	Diagnostics	The University of Melbourne, Metagenomics and Microbiomes	Land-based birds Pasteurellosis
Andrew Peters	Disease management	Wildlife Health and Pathology, Wildlife Health Australia Management committee	Land-based birds
Anke Wiethoelter	Epidemiology	The University of Melbourne, Veterinary Epidemiology, One Health, Risk analyses	Shore and water-based birds Botulism
Anna Meredith	Disease management	The University of Melbourne, Head of Melbourne Veterinary School, Wildlife Health and Conservation Medicine	Pollutants
Clare Death	Diagnostics	Wildlife toxicology research	Predatory birds Pollutants
Elyse Herrald-Woods	Disease management and policy	Director Environmental Biosecurity Office, Department of Agriculture, Water and the Environment	Predatory birds Pollutants
Jasmin Hufschmid	Disease management	The University of Melbourne, Wildlife Health and Disease	Shore and water-based birds Pollutants
Jemma Bergfeld	Diagnostics	CSIRO Australian Centre for Disease Preparedness (ACDP), Veterinary Pathologist	Land-based birds Pasteurella
Kate Brandis	Ecology	UNSW Research fellow, Wetland ecology, Avian botulism and water management, waterbird breeding, waterbirds and stable isotopes	Shore and water-based birds Botulism
Keren Cox-Witton	Disease management	Wildlife Health Australia, Senior Project Officer	Shore and water-based birds Botulism
Leanne Wicker	Disease management	Senior Veterinarian, Australian Wildlife Health Centre, Healesville Sanctuary - Zoos Victoria	Predatory birds Pollutants
Lee Berger	Diagnostics	The University of Melbourne, Wildlife Health and Conservation Medicine	Shore and water-based birds Botulism
Naomi Davis	Wetland management	Parks Victoria, Fauna Science and Management Effectiveness Branch	Predatory birds Pollutants
Pam Whiteley	Disease management	The University of Melbourne, Wildlife Health Victoria: Surveillance Coordinator	Shore and water-based birds Pasteurellosis

Name	Area of expertise	Organisation / Relevant Expertise	Small workshop groups
Paul Eden	Disease management	Associate Veterinarian, Werribee Open Range Zoo, Chair of the Veterinary Technical Reference Group of the Orange-bellied Parrot recovery program	Land-based birds
Rachel Pritchard	Wetland management	Natural Environment Program Officer, Chair of the Orange-bellied Parrot Recovery Program, DELWP representative on the Glenelg Estuary Ramsar Coordinating Committee	Land-based birds Pasteurellosis
Rebecca Gang	Epidemiology	The University of Melbourne, Research assistant	Land-based birds Pasteurellosis
Richard Jakob-Hoff	Wildlife disease risk analysis	Conservation Planning Facilitator and Disease Risk Analyst, Co-Convenor CPSG Australasia Regional Resource Centre	Facilitator
Robert Bone	Wetland management	Corangamite Catchment Management Authority	Shore and water-based birds Botulism
Simon Firestone	Epidemiology	The University of Melbourne, Veterinary Epidemiology, Risk analyses	Predatory birds
Stacey Lynch	Disease management	AIV wild bird program manager at DJPR AgriBio	Land-based birds
Steven Liu	Wetland management	Royal Botanic Gardens, Team Leader Landscape Systems	Land-based birds Botulism
Suelin Haynes	Wetland management	Melbourne Water, Environmental Officer / Planner, Western Treatment Plant	Predatory birds Pollutants
Will Steele	Ecology	Melbourne Water, Senior Biodiversity Scientist	Shore and water-based birds Botulism
Yonatan Segal	Disease management	Principal Veterinary Officer, Poultry and Emerging Diseases at Agriculture Victoria	Land-based birds Pasteurellosis

APPENDIX 5. WORKSHOP SCHEDULE

Day 1: Wednesday 11 March, 2020

Time	Topic	Who
8.30	Registration	
9.00	Welcome and housekeeping Background, goal, scope, focus and questions for this Workshop	PW/AW
9.20	Introduction to the IUCN-SSC and Conservation Planning Specialist Group (CPSG) with an overview of workshop program.	RJH
9.40	Self-introductions and working agreement	All
10.10	Coffee break	
10.30	Plenary Session #1 Overview of the IUCN/OIE Disease Risk Analysis (DRA) process, context of the workshop and DRA template for information capture.	RJH
10.50	**STEP 1: PROBLEM DESCRIPTION** Discussion of pre-circulated briefing notes and agreement on populations of interest for this DRA Introduction to concept of 'Acceptable Risk' Small group set up	RJH
11.15	**STEP 2: HAZARD IDENTIFICATION** Develop hazard prioritization criteria for populations of interest	RJH
11.30	Prioritization exercise: apply agreed prioritization criteria to each hazard for each population of interest. Identify and record key knowledge gaps at each step.	Small groups
12.15	LUNCH	
1.00	Presentation and review of small group outputs	Plenary
1.30	**STEP 3: RISK ASSESSMENT (high priority hazards)** Overview of steps and review membership of small groups: ● Hazard pathways for high priority disease hazards. ● Host, agent, environmental interactions ● Risk assessments	Small groups
3.00	Coffee break	
3.20	Risk Assessments (continued)	Small groups
4.20	Risk Assessments review	Plenary
5.00	Discussion of acceptable risk for populations of interest*	RJH
5.30	Review of day and agenda for day 2	RJH
5.40	**END OF DAY 1**	

*3-4 people to refine acceptable risk statement for review by plenary on day 2

PM Social evening - Dinner at 'The Views Bar and Grill', 350 K Rd, Werribee South VIC 3030

Day 2: Thursday 12 March, 2020

Time	Topic	Who
8.30	Introduction to the day and review of acceptable risk statement	RJH
8.45	**STEP 4: RISK MANAGEMENT**	
8.45	Review of risk assessments vs agreed acceptable risk statement	Plenary
9.00	ID Critical Control Points (CCPs) on hazard risk pathways Brainstorm risk management options for each CCP on risk pathway diagram Risk management option evaluation: effectiveness and feasibility (operationally and technically)	Small groups
10.30	Coffee break	
11.00	Small group reports: Risk management recommendations, knowledge gaps, action priorities and review common ground	Plenary
11.40	**STEP 5: RISK COMMUNICATION** Review of stakeholder list and draft communications strategy	Plenary
12.10	**STEP 6: IMPLEMENTATION AND REVIEW** Draft action plan to implement recommendations arising from risk management review	Small groups
1.00	LUNCH	
1.45	**STEP 6: IMPLEMENTATION AND REVIEW cont.** Draft action plan to implement recommendations arising from risk management review	Small groups
2.40	Small group reports: Implementation and review	Plenary
3.00	Coffee break	
3.20	Complete draft action plans including review and integration against timeline/next steps	Plenary
4.30	Workshop evaluation	All
4.45	**Farewell**	PW/AW

RJH – Richard Jakob-Hoff; PW – Pam Whiteley; AW – Anke Wiethoelter