

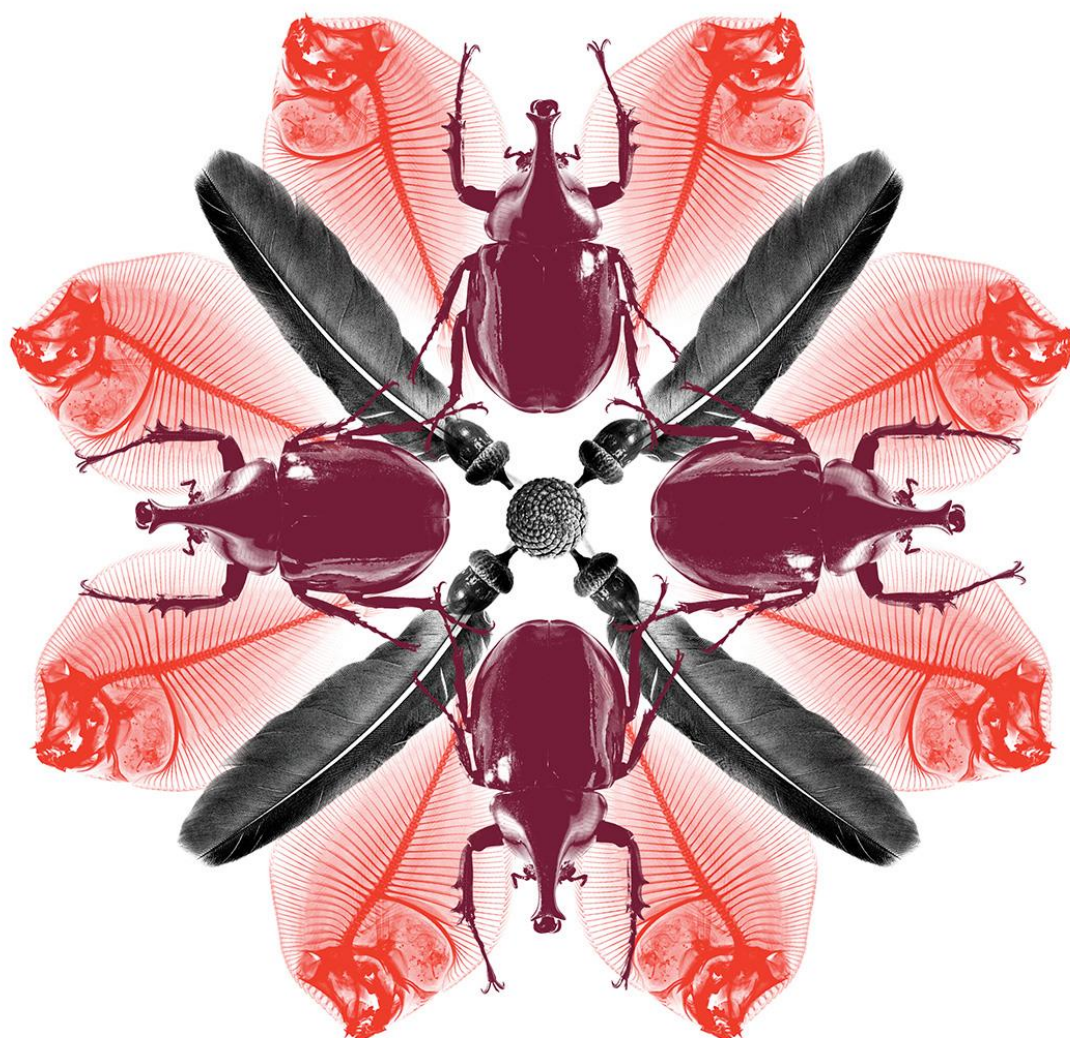


Australian Government
Department of Agriculture,
Water and the Environment

Import risk review for psittacine birds from all countries

Draft report

July 2020



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Stakeholder submissions on draft reports

This draft report allows interested parties to comment on relevant technical biosecurity issues. A final report will consider any comments received.

Submissions should be sent to the Department of Agriculture, Water and the Environment and must meet the conditions specified in the relevant [Biosecurity advice notice](#).

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Summary

The Australian Government Department of Agriculture, Water and the Environment has prepared this draft risk review to consider the biosecurity risks associated with the importation of live household pet and aviary psittacine birds into Australia.

Australia has previously permitted the importation of live psittacine birds. However, the policy was suspended in 1995 due to incomplete knowledge of certain diseases of psittacine birds and a lack of suitable methods for testing imported birds for the presence of these diseases.

This draft risk review takes into account new and relevant scientific information and advice from scientific experts. It proposes that the importation of live household pet and aviary psittacine birds to Australia be permitted, subject to a range of biosecurity risk management measures.

This draft risk review identifies hazards (disease agents) that require risk management measures to reduce biosecurity risk to a very low level in order to achieve Australia's appropriate level of protection (ALOP). The hazards requiring risk management measures are avian orthoavulavirus 1, internal and external parasites (excluding protozoa), parrot bornavirus, psittacine herpesvirus 1 and psittacine pox virus. In addition, risk management measures for avian influenza viruses are required in accordance with World Organisation for Animal Health (OIE) Terrestrial Animal Health Code recommendations for the importation of live birds other than poultry.

This draft risk review proposes a combination of risk management measures that will reduce the biosecurity risk associated with the importation of live household pet and aviary psittacine birds into Australia to achieve Australia's ALOP. Proposed measures include:

- sourcing from approved countries
- pre-export and post-entry quarantine
- veterinary inspection
- testing for diseases of biosecurity concern.

The release of this draft risk review will be followed by a 60-day stakeholder consultation period. Following consideration of stakeholder comments, the department will release a final risk review for the importation of household pet and aviary psittacine birds into Australia.

1 Introduction

1.1 Australia's biosecurity policy framework

Australia's biosecurity policies aim to protect Australia against risks that may arise from exotic pests and diseases entering, establishing and spreading in Australia, thereby threatening Australia's unique flora and fauna, agricultural industries that are relatively free from serious pests and diseases, and human health.

Risk analysis is an important part of Australia's biosecurity policies. It enables the Australian Government to formally consider the level of biosecurity risk that may be associated with proposals to import goods into Australia. If the biosecurity risks do not achieve Australia's ALOP, risk management measures are proposed to reduce the risks to an acceptable level. If the risks cannot be reduced to an acceptable level, the goods will not be imported into Australia until suitable measures are identified.

Successive Australian Governments have maintained a conservative, but not a zero risk, approach to managing biosecurity risks. This approach is reflected in Australia's ALOP, which reflects community expectations through government policy and is currently described as providing a high level of protection aimed at reducing risk to a very low level, but not to zero.

Australia's risk analyses are undertaken by the Department of Agriculture, Water and the Environment using technical and scientific experts from relevant fields, and involve consultation with stakeholders at various stages during the process.

Risk analyses conducted by the department are consistent with Australia's international biosecurity obligations including those under the World Trade Organization (WTO) Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) and the World Organisation for Animal Health (OIE). Risk analyses aim to establish a balance between our international obligations and the various risks that goods may pose.

Risk analyses may take the form of a regulated biosecurity import risk analysis (BIRA) or a non-regulated risk analysis (such as scientific review of existing policy and import conditions, or scientific advice).

More information about Australia's biosecurity framework is provided in the [Biosecurity import risk analysis guidelines 2016](#).

The department recognises that new scientific information and technologies, or other combinations of measures, may provide an equivalent level of biosecurity protection for the disease agents identified in this draft review as requiring risk management. The department will consider technical submissions that objectively demonstrate alternative biosecurity measures.

1.2 This risk review

1.2.1 Background

Historically, conditions for the importation of psittacine birds from approved countries were developed and finalised in 1989. Imports commenced in 1990 and between 1990 and 1995 approximately 4,800 birds were imported. A routine review of the importation program commenced in 1992 and, in 1995, the conditions were suspended due to concerns over the

unacceptable risk of introduction of certain exotic diseases of psittacine birds. Presently, the only psittacine birds that can be imported into Australia are pet birds from New Zealand whose owners are moving to Australia to reside permanently.

The department initiated this review in response to numerous and ongoing requests from pet psittacine bird owners, hobbyists and zoos to develop a safe importation pathway. Stakeholders were notified of the formal commencement of this review via [Biosecurity Advice 2016-14](#) on 2 May 2016.

1.2.2 Scope

This draft risk review considers the biosecurity risks posed by hazards associated with the importation of live household pet psittacine birds and aviary psittacine birds into Australia from all countries. Psittacine birds include all bird species within the Order Psittaciformes. Examples include lorries, cockatoos, cockatiels, rosellas, lovebirds and parrots.

For this review, household pet psittacine birds are defined as:

Genuine household pet psittacine birds which are usually not housed outside, would not be in contact with other birds not intended for export to Australia, and are not kept for commercial or recreational breeding purposes or exhibition.

Aviary psittacine birds are defined as:

Captive bred psittacine birds that may be kept indoors, outdoors and/or in a common aviary with other birds. This category includes birds held for hobby purposes or exhibition, in zoos, wildlife parks and conservation programs, as well as birds resident in breeding centres and private collections. It also includes birds purchased from overseas and intended to be kept as household pets after import into Australia.

In this draft risk review, consideration was given to current scientific information, international standards developed by the OIE, biosecurity measures adopted by other countries, expert opinion from the scientific community, as well as the practical and welfare requirements for the international movement of live birds in a safe manner.

1.2.3 Existing policy

The *Biosecurity Act 2015* and its subordinate legislation provide the legal basis under which biosecurity requirements for the importation into Australia of live animals and products derived from animals are regulated. The department implements and administers these requirements.

Import policy exists for household pet birds from New Zealand. Pet birds must have been in the ownership and possession of the owner for a minimum period of one year immediately preceding pre-export quarantine to be eligible for export to Australia and must be accompanied by an import permit and a New Zealand Government veterinary certificate confirming that the animal has undergone pre-export inspection and quarantine in accordance with Australian requirements. The birds must also undergo post-entry inspection and quarantine in Australia. The [import requirements](#) for this commodity can be found at the department's website.

Domestic arrangements

In addition to biosecurity import requirements for live psittacine birds the importation of live animals is regulated under the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act), which is also administered by the Department of Agriculture, Water and the Environment. Under the EPBC Act, live psittacine birds may only be imported if they appear on the [List of Specimens taken to be Suitable for Live Import](#) (Live Import List).

If a specimen is not on the Live Import List then the specimen cannot be imported. Anyone, whether a member of the public, a public or private institution or a commercial enterprise, can apply to the Minister for the Environment to amend the Live Import List to include a new specimen. More information can be found on the [Live Import List](#) webpage.

Australia is also a Party to the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES). CITES is an international agreement between governments that aims to ensure that the international trade in wildlife does not threaten wild populations of animals. Australia registers a [List of CITES Species](#) under the EPBC Act. Both commercial and non-commercial trade of CITES listed animals is regulated. This includes the transfer of live animals between zoos. Animals are listed under CITES in one of the 3 Appendices, depending on the threat of international trade to the survival of the species. Under the EPBC Act, Australia has adopted a range of domestic measures that impose additional requirements and in some cases further restrict trade in CITES listed species.

Although the Australian Government is responsible for regulating the movement of animals and animal products into and out of Australia, Australian state and territory governments are responsible for animal health and environmental controls within their jurisdictions. Legislation relating to resource management or animal health may be used by state and territory government agencies to control interstate movement of animals and their products. Once animals and animal products have been cleared by Australian Government biosecurity officers, they may be subject to interstate movement conditions. Some jurisdictions require importers to apply for entry in writing, and some species are not permitted entry to some jurisdictions (despite inclusion on the Live Import List). It is the importer's responsibility to identify, and ensure compliance with all requirements. More information can be found on Australian state and territory government websites.

1.2.4 Consultation

On 2 May 2016, [Biosecurity Advice 2016-14](#) was released announcing the commencement of this review. Following the release of this draft risk review, interested stakeholders are invited to comment and draw attention to any scientific, technical, or other gaps in the data, misinterpretations or errors, within the 60-day consultation period. Any supporting scientific evidence should be provided with stakeholder comments. Stakeholder feedback will be taken into consideration in the final review.

In order to support a robust evidence base, the department has sought expert opinion from the scientific community to contribute to this draft risk review.

1.2.5 Next steps

The department will consider submissions received on this draft review and may consult further with stakeholders. The department will then prepare a final report, taking into account stakeholder comments.

The final risk review will be published on the department's website along with a notice advising stakeholders of the release. The department will also notify registered stakeholders and the WTO committee on SPS measures of the release of the final report. Publication of the final report represents the end of the process. The conditions in the final report will form the basis of any import permits issued.

2 Method

The OIE Terrestrial Animal Health Code (OIE Code) describes ‘General obligations related to certification’ in Chapter 5.1, Article 5.1.2 (OIE 2019h):

The import requirements included in the international veterinary certificate should assure that commodities introduced into the importing country comply with the standards of the OIE. Importing countries should align their requirements with the recommendations in the relevant standards of the OIE. If there are no such recommendations or if the country chooses a level of protection requiring measures more stringent than the standards of the OIE, these should be based on an import risk analysis conducted in accordance with Chapter 2.1.

In addition:

The international veterinary certificate should not include measures against pathogens or diseases which are not OIE listed, unless the importing country has demonstrated through import risk analysis, carried out in accordance with Section 2, that the pathogen or disease poses a significant risk to the importing country (OIE 2019h).

The components of risk analysis as described in [Chapter 2.1](#) of the OIE Code are:

- hazard identification
- risk assessment (entry assessment, exposure assessment, consequence assessment and risk estimation)
- risk management
- risk communication.

Hazard identification, risk assessment and risk management are sequential steps within a risk analysis. Risk communication is an ongoing process and includes both formal and informal consultation with stakeholders. The release of this draft review for stakeholder comment forms part of the risk communication process.

2.1 Risk review

Risk is defined by the OIE Code as ‘the likelihood of the occurrence and the likely magnitude of the biological and economic consequences of an adverse event or effect to animal or human health’, and is dynamic in nature, changing with time. Consequently, risk should be kept under regular review.

Although not defined or described in the OIE Code, risk review is recognised by risk analysts as an essential component of the risk analysis process (Barry 2007; FSA 2006; Purdy 2010).

Australia applies a process of risk review to the biosecurity risks associated with the importation of an animal commodity (animal product or live animal) for which current biosecurity measures exist or biosecurity measures have already been developed. The latter option may be used where policy has been suspended.

Risk review differs from the *monitoring and review component* of risk management, as described in the OIE Code, in that each component of the risk analysis process (hazard identification, risk assessment and risk management) is reviewed under the risk review process. If a change in the biosecurity risk associated with a live animal or animal product is identified based on updated scientific information, risk management measures can be revised accordingly.

This risk review has drawn on the following sources of information (this list is not exhaustive):

- the OIE Code
- previous requirements for importation of live birds into Australia from Canada, Denmark, Iceland, Ireland, New Zealand, Norway, Sweden and the United Kingdom (developed 1989, suspended 1995)
- current requirements for importation of household pet birds into Australia from New Zealand
- *Importation of psittacine birds into Australia - technical issues paper* (Department of Agriculture 1999)
- a review of relevant scientific literature
- expert opinion.

For this review, material from previous risk analyses and importation requirements was found to be outdated and a substantial body of new information relating to biosecurity risks associated with psittacine birds was available. Therefore, rather than reviewing and updating previous risk assessments and risk management policy, each component of this review was developed from the beginning, drawing on both previous risk analyses and new scientific information.

2.2 Hazard identification

Hazard identification is described in the OIE Code ([Article 2.1.2](#)) as a categorisation step that is undertaken to identify potential hazards that may be associated with the importation of a commodity.

In accordance with the OIE Code, a disease agent was considered to be a potential hazard relevant to the importation of psittacine birds if it was assessed as:

- appropriate to the species being imported
- potentially being present in exporting countries.

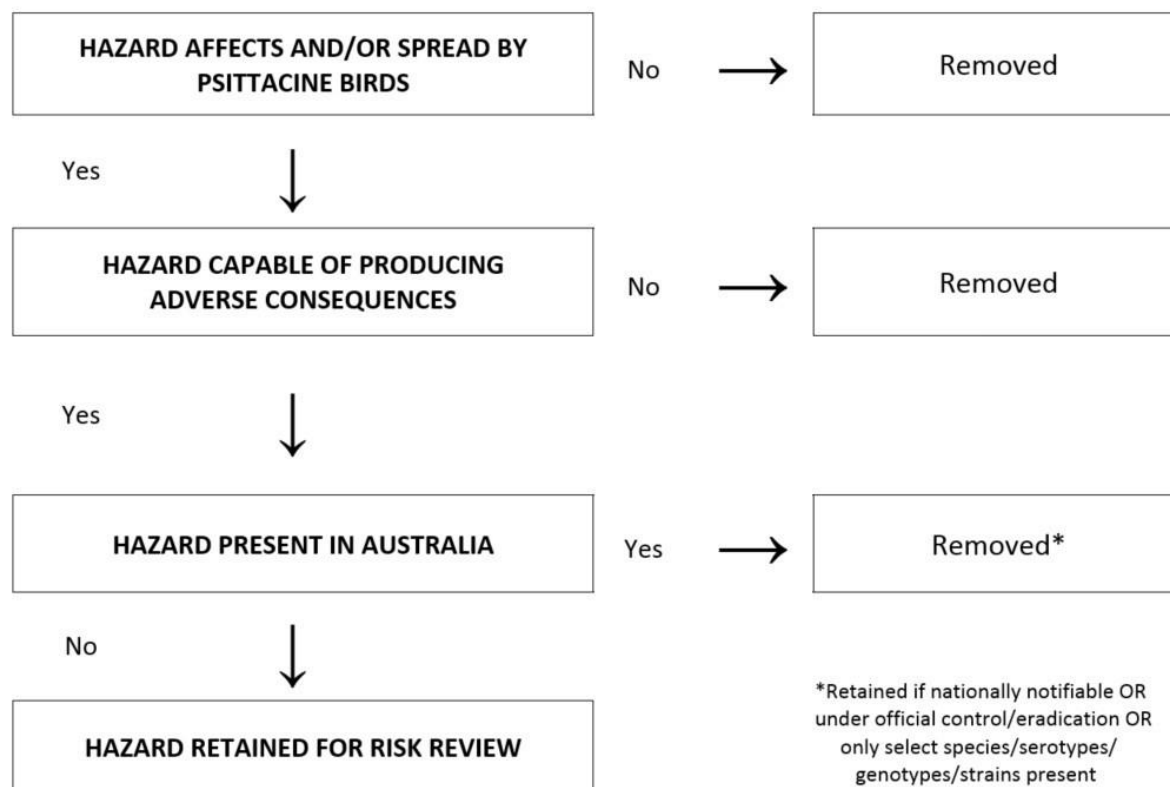
A list of potential hazards was developed following a review of scientific literature and expert consultation, and included hazards managed in previous and existing import policy, and those identified in the *Importation of psittacine birds into Australia - technical issues paper* (Department of Agriculture 1999).

A hazard was retained for further review (hazard refinement) if:

- it was identified as being capable of affecting or being spread by psittacine birds
- it was identified as emerging and/or capable of producing adverse consequences
- it is not present in Australia (or only select species/strains/etc. are present or it is present as a notifiable disease or subject to official control or eradication).

Where evidence for the inclusion or exclusion of a particular hazard was equivocal, a judgement was made based on the strength of the available evidence to implicate live psittacine birds in disease transmission.

Figure 1 Schematic diagram of hazard identification and refinement

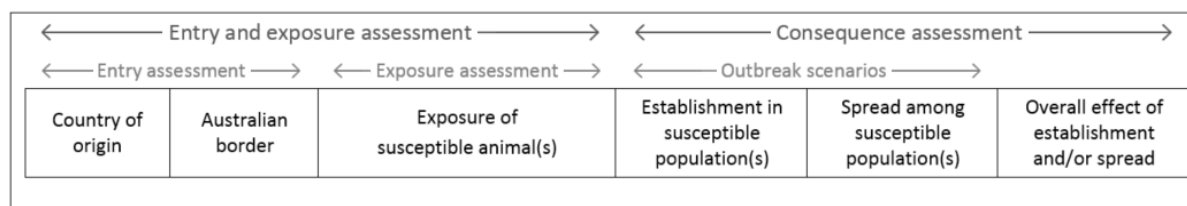


2.3 Risk assessment

Details of the risk assessment process relevant to live animals are provided in [Chapter 2.1](#) of the OIE Code.

In accordance with the OIE Code, the entry assessment describes the probability of the entry of each of the potential hazards under each specified set of conditions and how these might change as a result of various actions, events or measures. The exposure assessment describes the biological pathways necessary for exposure to the hazards from a given risk source and estimates the probability of the exposures occurring. The consequence assessment describes the potential consequences of a given exposure and estimates the probability of them occurring. The risk assessment for an identified hazard concludes with risk estimation—the integration of results from the entry assessment, exposure assessment and consequence assessment to produce overall measures of risks associated with the hazards identified—and yields the unrestricted risk estimate.

Steps in determining the unrestricted risk estimate are illustrated diagrammatically in Figure 2.

Figure 2 Components of the unrestricted risk estimate

A review of risk factors relevant to the entry, exposure and consequence assessment of hazards identified for further assessment was conducted to identify any significant changes in hazard attributes and/or geographic distribution that would be relevant to biosecurity considerations for Australia.

A review of peer-reviewed scientific literature was conducted and contact with relevant experts sought, where necessary, for each hazard retained for further assessment. Based on this information, a decision was then made whether or not to continue with the risk assessment as outlined below.

If definitive information on risk factors was not found through literature review or contact with relevant experts, any uncertainties were identified and documented in the relevant risk review (Chapter 4). Any assumptions and/or judgements made in drawing conclusions for each hazard retained for further review were also documented.

The risk assessment concluded with an unrestricted risk for each hazard. If the unrestricted risk did not achieve Australia's ALOP, then risk management measures were recommended to reduce the risk to achieve the ALOP.

2.3.1 The principle of a generic risk assessment

This risk review is 'generic', in that the risks associated with the importation of psittacine birds from any exporting country have been considered. The generic review does not consider the disease status or data of individual countries, but is based on estimates of the most likely situation in a hypothetical infected country. Country specific data may be considered at a later date should appropriate data from prospective exporting countries be supplied.

In order to carry out entry assessments that are relevant to all exporting countries, the assumption was made that if a hazard was present in a country, it would be present at the highest sustainable flock-level and within-flock level prevalence. This assumption was based on the premise that prevalence would be dictated by epidemiological characteristics of the disease, and is, by nature, dynamic and thus may differ from country to country, and through time within a country. It was further assumed that no on-going monitoring or surveillance in respect to the disease in question was generally undertaken by the exporting country for household pet and aviary birds. This assumption allowed generic assessment to be carried out, and allowed some diseases to be eliminated from further consideration on the basis that they would not present a risk in excess of Australia's ALOP, even if present at the highest sustainable prevalence in the exporting country.

Because of the generic nature of this risk review, the evaluation of the likelihood of entry was based on estimates of the most likely situation in an infected country. Where exporting countries can provide specific data on their particular disease status, the department will reconsider the

release assessment based on that data so that country specific circumstances are considered in determining whether particular biosecurity measures are required.

2.3.2 Evaluating and reporting likelihood

For those hazards retained for further risk assessment, the assessment was conducted using a qualitative approach.

2.3.3 Entry and exposure assessment

Entry assessment

The entry assessment estimates the likelihood that a given hazard would be present in a psittacine bird imported into Australia. A number of factors were taken into account in determining the likelihood of a hazard entering Australia in psittacine birds:

- exposure of a psittacine bird to a hazard
- prevalence of the hazard in psittacine bird populations in an exporting country/zone/compartiment (considered to be the highest sustainable between-flock and within-flock prevalence applicable)
- surveillance and control programs of exporting countries (considered to be the minimum standard applicable)
- epidemiology of hazard (including ease of recognising clinical signs, transmission, latency and predilection sites)
- the effect of transport (stress).

The following assumptions were applied for each entry assessment:

- Contact between reservoirs of infection (e.g. wild birds, infected poultry, vectors) is considered common for aviary birds (mainly housed outdoors, potentially with backyard poultry) and less common for household pet birds (mainly housed indoors).
- Few, if any, veterinary authorities undertake routine surveillance of aviary birds or wild psittacine birds therefore the true prevalence of infection is generally unknown.
- For hazards transmitted by oral/respiratory/mucosal routes, birds may become infected from other infected birds included in the same consignment, due to the close contact between birds during shipment.
- Elements of transport (e.g. stress, crowding, poor air ventilation, accumulation of excrement and stacking of enclosures) may increase the likelihood of recrudescence of latent disease, shedding and transmission.

For each hazard, a qualitative likelihood was assigned to describe the likelihood of the hazard entering Australia via the importation of an infected psittacine bird.

Exposure assessment

The exposure assessment estimates the likelihood that a susceptible bird in Australia will be exposed to a hazard introduced via an imported psittacine bird. It takes into account the groups of birds most likely to be affected as well as the possible pathways by which exposure of these groups could occur.

The term ‘exposure group’ categorises a group of animals that may be susceptible to one or more of the potential hazards considered in risk assessments. The most likely exposure groups to imported psittacine birds, directly or indirectly, were considered to be:

- wild birds
- captive birds
- low biosecurity poultry—backyard poultry and free-range commercial poultry (and ratites)
- medium biosecurity poultry—non-genetic stock commercial poultry, housed indoors
- non-avian species, where appropriate.

The sequence of steps for imported infected psittacine birds to potentially expose susceptible animals to the hazard (the exposure pathways) were analysed. Imported psittacine birds would move into the owner’s home or aviary. Imported psittacine birds may have direct and/or indirect exposure to all exposure groups, with some exposure pathways being more likely to occur than others.

The following exposure group dependent variables were considered for each exposure assessment:

- the likelihood that birds/animals in exposure groups will be susceptible to infection by a hazard at the time they are exposed to it (should they be exposed to it), considering the species, age and immune status within exposure groups
- the behavioural characteristics or management of the exposure groups
- the level of biosecurity within exposure groups
- the exposure groups response to infection (shedding of hazard and duration, expected morbidity rate, evidence of clinical disease).

The following pathogen dependant variables were considered for each exposure assessment:

- natural epidemiology of the hazard
- the infectivity and pathogenicity of the hazard
- method of transmission (e.g. aerosol, droplet, contact, vector) and secondary spread
- transmissibility, and for indirect transmission, the hardiness of the pathogen (resistance) and the likelihood it will remain viable after exposure in the environment over the period (persistence) before it is exposed to the susceptible animals
- the presence of suitable vectors, where applicable
- seasonal climatic conditions
- the likelihood that an infective dose is received.

The following generic factors were considered for each exposure assessment:

- For pathogen transmission to occur imported birds must have direct or indirect contact with other susceptible species. While single pet birds are commonly kept indoors, larger collections are often housed outdoors in mesh/wire enclosures that allow direct and indirect contact with wild birds, vectors and backyard poultry, if present.
- Waste generated by imported birds is most likely to be disposed of in household rubbish or in the environment.

- The number of people who would keep both psittacine birds and poultry on the same premises in Australia is unknown.
- For vector transmitted hazards, if an imported bird is not housed in an enclosure that is adequate to exclude relevant vectors, it will allow for indirect contact with other birds and backyard poultry, if present. Pet birds, being housed indoors, are likely to have less vector contact than aviary birds.

For each hazard, the final outcome of the exposure assessment was an overall estimate of the likelihood that susceptible birds/animals in exposure groups would be exposed to a hazard via an infected imported psittacine bird (i.e. the likelihood of exposure).

Estimation of likelihood of entry and exposure

The likelihood of entry and exposure was estimated by combining the likelihood of entry and the corresponding likelihood of exposure using the matrix as described in Figure 3.

Figure 3 Matrix for combining qualitative likelihoods

Likelihood of entry	<i>High</i>	Negligible	Extremely low	Very low	Low	Moderate	High
	<i>Moderate</i>	Negligible	Extremely low	Very low	Low	Low	Moderate
	<i>Low</i>	Negligible	Extremely low	Very low	Very low	Low	Low
	<i>Very low</i>	Negligible	Extremely low	Extremely low	Very low	Very low	Very low
	<i>Extremely low</i>	Negligible	Negligible	Extremely low	Extremely low	Extremely low	Extremely low
	<i>Negligible</i>	Negligible	Negligible	Negligible	Negligible	Negligible	Negligible
		<i>Negligible</i>	<i>Extremely low</i>	<i>Very Low</i>	<i>Low</i>	<i>Moderate</i>	<i>High</i>
Likelihood of exposure							

The result of this process was an estimate of the overall likelihood of entry and exposure for a particular hazard.

2.3.4 Consequence assessment

Identification of an outbreak scenario

Once exposure of a susceptible avian or non-avian population has occurred, a number of possible outbreak scenarios could follow, representing a continuum ranging from no spread to widespread establishment. Most hazards of psittacine birds assessed in this draft review are not subject to formal response arrangements in Australia, such as contractual arrangements under the Emergency Animal Disease Response Agreement (EADRA). In these cases, no agreement exists between Australia's governments and industry groups to collectively reduce the risk of disease incursions and manage a response if an outbreak occurs. This situation was considered in outbreak scenarios.

Outbreak scenarios following exposure to a hazard from an imported psittacine bird are grouped into 4 categories:

- No outbreak: the hazard does not establish or is not recognised in the exposed population.
- Local (limited) outbreak: the hazard establishes in a directly exposed population only (captive birds, wild birds, poultry and/or non-avian species) and is eradicated or is self-limiting without further spread.

- Regional outbreak: the hazard establishes in a directly exposed population and spreads to other populations in the region.
- Widespread outbreak: the hazard establishes in a directly exposed population, spreads to other populations and becomes endemic in Australia.

All categories of outbreak scenarios were evaluated for plausibility, based on the epidemiology of each hazard. In this draft review, the most likely outbreak scenario for each hazard, resulting from the exposure of susceptible animals, was considered in a single pathway resulting in infection and establishment (described in the relevant hazard risk review).

Estimation of likelihood of establishment and/or spread

The likelihood of the most probable outbreak scenario occurring was then estimated to obtain a likelihood of establishment and/or spread.

The following factors were considered for each likelihood of establishment and/or spread assessment:

- Australian native psittacine birds are likely to be susceptible to infection, however, the degree of susceptibility may vary between species. Where specific information about disease progression is not available, the progression of infection in Australian native psittacine birds is assumed to be similar to that in exotic psittacine birds, and where information is available, Australian native psittacine birds held overseas.
- It is difficult, if not impossible, to control the spread of most diseases once they have become established in a wild population. Management of disease in wild bird populations is difficult and successful control or eradication efforts by government or non-government organisations is considered unlikely.
- Imported birds will be under observation by owners and the presentation of overt clinical disease is likely to be detected and investigated. Early detection and management would assist in mitigating further disease spread.
- Shedding of disease agent may occur during the incubation period of a disease (prior to development of clinical signs), and for subclinical/latent disease may occur intermittently despite birds appearing healthy.
- Subclinical disease or latency may delay the recognition of disease spread into wild populations.

When estimating the likelihood of establishment and/or spread associated with the outbreak scenario, qualitative descriptors were used.

Determination of the overall effect of establishment and/or spread associated with the outbreak scenario

For the most likely outbreak scenario, the overall effect of establishment and/or spread were determined. This was addressed in terms of direct and indirect effects as detailed below. An effect was not assessed more than once and direct effects were considered separately from indirect effects.

Several factors considered when assessing effects are outlined below under the respective direct or indirect effect and are not repeated in individual risk assessments.

*Direct effects**Life or health (including production effects) of susceptible animals, including public health consequences:*

- Loss of life, health and production may be experienced due to morbidity and mortality of infected susceptible species.

The living environment, including life and health of wildlife, and any effects on the non-living environment:

- Impacts on the living environment may be experienced due to morbidity and mortality of wild birds.
- For vector-borne diseases, vector control using insecticides, if implemented, may result in residue issues in the environment.

*Indirect effects**New or modified eradication, control, monitoring or surveillance and compensation strategies or programs:*

- Where they occur in poultry, diseases covered by EADRA will be managed by the jurisdiction affected according to procedures outlined in the relevant Australian Veterinary Emergency Plan (AUSVETPLAN) manual. Costs will be shared between government and industry as per the diseases categorisation in the EADRA.
- For diseases not covered by EADRA, even if the disease spreads generally in captive and wild bird populations, it is unlikely to result in the implementation of government-administered eradication and control programs, or compensation. Individual owners are likely to bear associated costs of any strategies or programs they implement.

Domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries:

- Affected businesses and individuals may lose large numbers of stock, impacting income and viability. This would have flow-on effects to supplying businesses (changes to input demand) and those in sales and service (changes to output availability).
- If breeding flocks are affected, valuable genetic material may be lost.
- Morbidity and mortality due to disease may result in fewer stock being available for sale, increasing prices for customers, or seeing business move elsewhere to meet demand.

International trade, including loss of markets, meeting new technical requirements to enter or maintain markets and changes in international consumer demand:

- Impact on export trade of psittacine birds and other ornamental birds from Australia would not be significant as existing trade is very limited.
- International markets for commercial poultry and poultry products (including egg and egg products) from Australia are relatively limited, but still valuable. However, given the focus on the domestic market the impact of international trade losses should minimally affect the Australian industry.

The environment, including biodiversity, endangered species and the integrity of ecosystems:

- If the disease spreads to wild psittacine birds or other avian species there is the potential for a reduction in biodiversity. In the event that populations of endangered birds are affected there is the potential for loss of genetic diversity within a species, or potentially the species itself.

- Disposal of stock and contaminated products and cleaning/decontamination activities will impact the environment on a scale dependant on numbers of dead stock, methods of disposal and chemicals used.

Communities, including reduced tourism, reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures:

- The loss of pet and aviary birds is unlikely to contribute significantly to reductions in economic viability, however, the loss of commercial poultry may do so.
- The loss of wild bird populations could impact tourism, particularly for ecotourism including bird-watching. This does not make up a major part of the Australian tourism industry, but it is an emerging sector.
- The loss or destruction of pet birds, aviary birds, poultry and/or wild birds is likely to have social impacts on affected communities.
- Approximately 11.8% of households in Australia keep pet birds (AMA 2016) and the loss of an individual bird would be emotionally distressing due to the societal value placed by the Australian community on their pet birds.

The overall effect of establishment and/or spread associated with the outbreak scenarios took into account the level of these effects:

- local—restricted to a single locality or town
- regional—a recognised geographic area such as far north Queensland
- state or territory
- national

and the magnitude of these effects:

- indiscernible—not usually distinguishable from normal day-to-day variation
- minor significance—recognisable, but minor and reversible
- significant—serious and substantive, but reversible and unlikely to have permanent economic effects
- highly significant—extremely serious and irreversible and likely to have permanent economic effects.

Based on the level and magnitude of effects, the overall effect of establishment and/or spread was estimated using the rules described in Table 1.

Table 1 Rules for determining the overall effect of establishment and/or spread

Overall effect	Description
Extreme	The effect is likely to be highly significant at the national level. Implies that economic stability, societal values or social well-being would be seriously affected.
High	The effect is likely to be significant at the national level and highly significant within affected zones. Implies that the effect would be of national concern. However, serious effects on economic stability, societal values or social well-being would be limited to a given zone.
Moderate	The effect is likely to be recognised on a national level and significant within affected zones. The effect is likely to be highly significant to directly affected parties.
Low	The effect is likely to be recognised within affected zones and significant to directly affected parties. It is not likely that the effect will be recognised at the national level.

Overall effect	Description
Very low	The effect is likely to be minor to directly affected parties. The effect is unlikely to be recognised at any other level.
Negligible	The effect is unlikely to be recognised at any level within Australia.

Derivation of likely consequences

The likely consequences were determined by combining the likelihood of establishment and/or spread (associated with the outbreak scenario) with the overall effect of establishment and/or spread using the matrix shown in Figure 4.

Figure 4 Likely consequences: a combination of the likelihood and overall effect of establishment and/or spread

Likelihood of establishment and/or spread	<i>High</i>	Negligible	Very low	Low	Moderate	High	Extreme
	<i>Moderate</i>	Negligible	Very low	Low	Moderate	High	Extreme
	<i>Low</i>	Negligible	Negligible	Very low	Low	Moderate	High
	<i>Very low</i>	Negligible	Negligible	Negligible	Very low	Low	Moderate
	<i>Extremely low</i>	Negligible	Negligible	Negligible	Negligible	Very low	Low
	<i>Negligible</i>	Negligible	Negligible	Negligible	Negligible	Negligible	Very low
		<i>Negligible</i>	<i>Very Low</i>	<i>Low</i>	<i>Moderate</i>	<i>High</i>	<i>Extreme</i>
Overall effect of establishment and spread							

2.3.5 Risk estimation

Risk estimation is the integration of the likelihood of entry and exposure, and the likely consequences of a hazard being introduced by the importation of psittacine birds.

The risk is estimated by:

- determining the likelihood of entry and exposure
- determining the likelihood of establishment and/or spread among susceptible populations and the overall effect of establishment and/or spread to estimate the likely consequences
- combining the likelihood of entry and exposure with the estimate of likely consequences.

Prior to finalisation of all risk assessments, the assessments were considered by internal and external experts in risk assessment and avian disease, to ensure the assessments accurately estimated risks, were in accord with current scientific thinking and were consistent.

Combining the likelihood of entry and exposure and likely consequences was undertaken using the rules shown in the risk estimation matrix in Figure 5. This resulted in the unrestricted risk, which was considered the final output of the risk assessment.

Figure 5 Risk estimation matrix

Likelihood of entry and exposure	High	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
	Moderate	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
	Low	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk	High risk
	Very low	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk
	Extremely low	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk
	Negligible	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk
		Negligible	Very Low	Low	Moderate	High	Extreme
		Likely consequences					

2.4 Risk management

2.4.1 Evaluation of unrestricted risk and option evaluation

Risk evaluation is described in the OIE Code as the process of comparing the risk estimated in the risk assessment with the reduction in risk expected from the proposed risk management measures. The option evaluation process identifies, evaluates the efficacy and feasibility of, and selects measures to reduce the risk associated with an importation.

Following each risk assessment, if the unrestricted risk was 'negligible' or 'very low', then it achieved Australia's ALOP and risk management was not required. If the unrestricted risk was 'low', 'moderate', 'high' or 'extreme', risk management measures were required.

Once the unrestricted risk for a particular hazard was assessed and evaluated as exceeding Australia's ALOP, measures to manage and reduce that risk were considered.

The imposition of a particular risk management measure or a combination of measures results in the derivation of the restricted risk. The aim of risk management measures is to meet Australia's ALOP by reducing the restricted risk to 'very low' or 'negligible'.

Risk management options considered in this draft report aim to reduce the likelihood that importation of psittacine birds would lead to the entry, exposure, establishment and/or spread of hazards of biosecurity concern in Australia. These may be imposed pre-border and aim to reduce the likelihood of hazards entering Australia in psittacine birds, or post-entry aiming to reduce the exposure of the hazard in susceptible local populations.

Australia bases its import risk management measures on the standards, guidelines and recommendations set by the OIE. However, when such standards do not achieve Australia's ALOP or relevant standards do not exist, Australia exercises its right under the SPS Agreement to apply appropriate measures, justified on scientific grounds and supported by risk analysis.

The specific measures recommended for hazards where the unrestricted risk did not achieve Australia's ALOP are described in detail in Chapter 5 of this draft review.

Household pet versus aviary birds

The risk assessment and expert consultation processes identified important differences between the estimated risks posed by household pet psittacine birds compared to aviary psittacine birds.

The risk assessment process evaluated both categories of imports and resulted in an overall unrestricted risk. However, it was considered that household pet psittacine birds (as defined by this review) posed a lower likelihood of entry and exposure for diseases of biosecurity concern for the following reasons¹:

- Household pet birds are generally possessed in limited numbers by an owner and are under their personal care.
- Household pet birds are likely to be housed indoors for at least part of the year preceding import, decreasing the likelihood of disease exposure (in the country of origin). Household pet birds imported into Australia are likely to be housed indoors decreasing the likelihood of disease transmission (in Australia).
- Household pet birds are unlikely to be housed in contact with many other birds decreasing the likelihood of disease exposure (in country of origin).

To reflect the difference in risk of entry and exposure between household pet and aviary psittacine bird imports, risk management measures were developed for each category (see Chapter 4.11).

2.5 Risk communication

The OIE Code defines risk communication as:

The interactive transmission and exchange of information and opinions throughout the risk analysis process concerning risk, risk-related factors and risk perceptions among risk assessors, risk managers, risk communicators, the general public and other interested parties. (OIE 2019h)

In conducting import risk analyses and risk reviews, the department consults with internal and external stakeholders in the development of Australia's animal biosecurity measures. Furthermore, a formal process of consultation with external stakeholders is a standard procedure for all import risk analyses and risk reviews to enable stakeholder assessment and feedback on draft conclusions and recommendations about Australia's animal biosecurity measures.

2.6 References

AMA 2016, 'Pet ownership in Australia 2016', Animal Medicines Australia, Canberra, available at <http://animalmedicinesaustralia.org.au/pet-report/> accessed 1 June 2018.

Barry, M 2007, *Effective approaches to risk assessment in social work: an international literature review*, Education Information and Analytical Services, Scottish Executive, Edinburgh.

Department of Agriculture 1999, *Importation of psittacine birds into Australia - technical issues paper*, Department of Agriculture, Canberra.

¹ This information is based on the definition of household pet psittacine birds in this review and is what the department intends to be true for household pet psittacine bird imports. However, the department notes that these are general assumptions, (e.g. in some cases imported household pet psittacine birds might not be kept as indoor pets for the remainder of their lives) and has considered this variation in the development of risk management measures.

FSA 2006, *The FSA's risk-assessment framework*, The Financial Services Authority, London, available at http://www.fsa.gov.uk/pubs/policy/bnr_fim-framework.pdf (pdf 296 kb).

OIE 2019h, *Terrestrial animal health code 2019*, World Organisation for Animal Health, Paris, available at <http://www.oie.int/standard-setting/terrestrial-code/access-online/>.

Purdy, G 2010, 'ISO 31000:2009: setting a new standard for risk management', *Risk Analysis*, vol. 30, no. 6, pp. 881-6, available at <http://dx.doi.org/10.1111/j.1539-6924.2010.01442.x>, accessed 5 March 2012.

3 Hazard identification

The list of hazards of potential biosecurity concern was compiled from:

- hazards managed in previous import policy
- hazards listed by the OIE affecting birds and relevant to psittacine birds
- hazards identified in the Importation of psittacine birds into Australia - Technical Issues Paper (Department of Agriculture 1999)
- other hazards identified in the literature as occurring in psittacine birds.

The method of hazard identification and refinement is described in Section 2.2. The hazard identification decision tree is shown in Figure 1. The preliminary list of hazards is shown in Table 2. This table summarises the results of the hazard refinement process, including the reason for removal or retention of each identified hazard.

The department gave careful consideration to hazards for inclusion in the list. Ubiquitous or common commensals which may be present in Australia in addition to those that are opportunistic, not reported to be pathogenic, or of uncertain relevance in the commodity due to limited or insufficient information were not considered. Explanatory comments on selected hazards identified in the list have been added in Appendix A: Explanatory comments for identified hazards.

The hazards retained after identification and refinement (Table 2) are listed at the end of this chapter.

Table 2 Hazard identification and refinement

Hazard	Hazard affects and/or spread by psittacine birds	Hazard capable of producing adverse consequences	Hazard present in Australia	Hazard nationally notifiable/under official control/eradication	Hazard retained for risk review
<i>Aegyptianella</i> spp	Not of significance a	No	No	No	No: not likely to produce adverse effects
Astroviruses	Not of significance b	No	Unknown	No	No
Avian adenoviruses affecting psittacine birds	Yes	No	Yes: select viruses reported (see Appendix A)	No	No
Avian influenza viruses	Yes	Yes	No: domestic birds. Yes: only select subtypes present in wild birds	Yes	Yes: some subtypes not present in Australia
Avian papillomaviruses	Not of significance c	No	Unknown	No	No
Avian orthoavulavirus 1	Yes	Yes	Yes: only select strains present	Yes – infection with virulent strains only	Yes
Avian paraavulavirus 3	Yes	Yes	No	No	Yes
Avian metaavulavirus 5	Yes	Yes	Limited reports	No	Yes
Avian polyomavirus	Yes	Yes	Yes	No	No: present in Australia (see Appendix A)
Avian pox viruses (other than Psittacine pox virus)	Yes	Yes (some that are already present such as fowl pox); others unknown.	Some present; others unknown	No	No
Avian retroviruses	Unknown	Unknown	Yes	No	No
<i>Avibacterium paragallinarum</i>	Yes	No d	Yes: only select strains present	No	No

Hazard	Hazard affects and/or spread by psittacine birds	Hazard capable of producing adverse consequences	Hazard present in Australia	Hazard nationally notifiable/under official control/eradication	Hazard retained for risk review
Avihepadnavirus	Unknown	Unknown	Unknown	No	No (see Appendix A)
<i>Bordetella avium</i>	Yes	Yes	Yes	No	No
Budgerigar herpesviruses	Yes	No (see Appendix A)	Unknown	No	No
<i>Chlamydophila psittaci</i>	Yes	Yes	Yes	No	No
Coronaviruses of psittacine birds	Not of significance e	Unknown	Unknown	No	No: no evidence psittacines play a significant role in disease epidemiology
Cryptosporidium	Yes	Yes	Yes	No	No
<i>Escherichia albertii</i>	Yes	Yes	Yes	No	No
Haemosporidia	Yes	Yes	Yes: genera present in birds in Australia (see Appendix A)	No	No: possible worldwide occurrence
Internal and external parasites (excluding protozoa)	Yes	Yes	Yes: various species present	No	Yes: some species not present in Australia
<i>Macrorhabdus ornithogaster</i>	Yes	Yes	Yes	No	No
Microsporidia	Yes	Yes	Yes	No	No
<i>Mycobacterium avium</i> and <i>M. genavense</i>	Yes	Yes	Yes	Yes: for <i>M. avium</i>	No (see Appendix A)

Hazard	Hazard affects and/or spread by psittacine birds	Hazard capable of producing adverse consequences	Hazard present in Australia	Hazard nationally notifiable/under official control/eradication	Hazard retained for risk review
<i>Mycoplasma gallisepticum</i>	Yes	Yes	Yes	No	No
<i>Mycoplasma synoviae</i>	Yes	Yes	Yes	No	No
<i>Pasteurella</i> spp.	Not of significance (see Appendix A)	Not of significance	Yes	No	No
Parrot bornavirus	Yes	Yes	Yes: only select genotypes present	No	Yes: some genotypes not present in Australia
Psittacine circovirus (beak and feather disease)	Yes	Yes	Yes	No	No
Psittacid alphaherpesvirus 1 and psittacid herpesvirus 2 (PsHV-1/PsHV-2)	Yes	Yes	Yes: for PsHV-1 - only select genotypes present Unknown for PsHV-2	No	Yes: PsHV-1 - some genotypes not present in Australia Yes: PsHV-2
Psittacine pox virus	Yes	Yes	No	No	Yes
Reovirus	Yes	Yes	Not reported in psittacine birds	No	Yes
Respiratory herpesviruses including Amazon tracheitis virus and psittacine herpesvirus 3 (PsHV-3)	Yes	Yes	Yes: for PsHV-3 Unknown for others (see Appendix A)	No	No
<i>Salmonella</i> spp. f	Yes	Yes	Yes: select species present	Yes (for <i>S. enteritidis</i> in poultry, <i>S. gallinarum</i> and <i>S. pullorum</i>)	Yes
<i>Sarcocystis falculata</i>	Yes	Yes	No: definitive host not present in Australia	No	No (see Appendix A)
Spironucleus	Yes	Yes	Yes	No	No
West Nile virus	Yes	Yes	No	Yes (for clinical disease)	Yes

Hazard	Hazard affects and/or spread by psittacine birds	Hazard capable of producing adverse consequences	Hazard present in Australia	Hazard nationally notifiable/under official control/eradication	Hazard retained for risk review
<i>Yersinia pseudotuberculosis</i>	Yes	Yes	Yes	No	No

a There has been one description of this infection in a single blue-fronted Amazon (*Amazona aestiva*) imported into the United Kingdom and it has not been found outside of Europe, Asia and Africa. It would be extremely unlikely that the life cycle of this organism could be maintained in aviculture in Europe and North America (Peirce & Bevan 1977).

b There are no definitive reports of disease being caused by astroviruses in psittacine birds.

c One case of avian papillomavirus has been reported in wild caught African grey parrots (*Psittacus erithacus*) (Tachezy et al. 2002).

d Isolates with similar biochemical profiles to *A. paragallinarum* are known to occur in psittacine birds (Christensen, Blackall & Bisgaard 2009), however, these are genetically distinct from *A. paragallinarum* isolates that are found to cause disease in chickens. The bacterium is generally considered to be a poultry pathogen.

e There are 2 reports in the literature of coronaviruses being isolated from psittacine birds, however, their ability to cause disease in psittacine birds is unknown (Gough et al. 2005; Hirai, Hitchner & Calnek 1979).

f *Salmonella* spp. includes *S. arizonae*, *S. enteritidis*, *S. gallinarum*, *S. pullorum* and *S. typhimurium* (antibiotic resistant strains). Some serotypes of *S. arizonae* are present in Australia.

The following diseases/disease agents were retained for risk review on the basis of the information provided in Table 2:

- avian influenza viruses
- avian orthoavulavirus 1
- avian paraavulavirus 3
- avian metaavulavirus 5
- internal and external parasites (excluding protozoa)
- parrot bornavirus
- psittacine alphaherpesvirus 1 and psittacine herpesvirus 2
- psittacine pox virus
- reovirus
- *Salmonella* spp. (infection with)
- West Nile virus.

3.1 References

Christensen, H, Blackall, PJ & Bisgaard, M 2009, 'Phylogenetic relationships of unclassified, satellitic Pasteurellaceae obtained from different species of birds as demonstrated by 16S rRNA gene sequence comparison', *Research in Microbiology*, vol. 160, no. 5, pp. 315-21, available at <https://doi.org/10.1016/j.resmic.2009.05.006>, accessed 9 September 2019.

Department of Agriculture 1999, *Importation of psittacine birds into Australia - technical issues paper*, Department of Agriculture, Canberra. Gough, D, Ceeraz, V, Cox, B, Palya, V & Mato, T 2005, 'Isolation and identification of goose parvovirus in the UK', *Veterinary Record*, vol. 156, no. 13, p. 424, available at <http://dx.doi.org/10.1136/vr.156.13.424>, accessed 1 May 2018.

Hirai, K, Hitchner, SB & Calnek, BW 1979, 'Characterization of a new coronavirus-like agent isolated from parrots', *Avian Diseases*, vol. 23, no. 2, pp. 515-25, available at <http://www.jstor.org/stable/1589582>, accessed 9 September 2019.

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Tachezy, R, Rector, A, Havelkova, M, Wollants, E, Fiten, P, Opdenakker, G, Jenson, AB, Sundberg, JP & Van Ranst, M 2002, 'Avian papillomaviruses: the parrot *Psittacus erithacus* papillomavirus (PePV) genome has a unique organization of the early protein region and is phylogenetically related to the chaffinch papillomavirus', *BMC Microbiology*, vol. 2, no. 1, pp. 19, available at <https://doi.org/10.1186/1471-2180-2-19>, accessed 28 October 2019.

4 Risk reviews

4.1 Avian influenza viruses

4.1.1 Background

Influenza viruses of veterinary importance to birds are type A influenza viruses from the family Orthomyxoviridae (as reviewed in Swayne et al. 2013). Wild aquatic birds are thought to be the natural reservoir of avian influenza viruses (AIVs) and migratory waterfowl play a role in introducing and spreading virus within and across continents (as reviewed in Swayne et al. 2013). AIVs can infect a wide range of domestic and wild bird species as well as mammals, including humans (as reviewed in Swayne et al. 2013). Mammalian infection is less common than avian infection (WHO 2007). The clinical severity of infection with AIVs depends on factors such as host species and virus strain (as reviewed in Tollis & Di Trani 2002).

Influenza viruses are subtyped according to the expression of 2 surface proteins: haemagglutinin (H) and neuraminidase (N) (OIE 2019a). The surface of AIVs may contain one of 16 types of H protein and one of 9 types of N protein in any combination. Clinically, AIVs are assigned to one of 2 categories based on virulence in chickens and/or specific genetic features. These categories are low pathogenic avian influenza (LPAI) viruses and highly pathogenic avian influenza (HPAI) viruses (OIE 2019a).

The most devastating clinical, social, economic and trade effects occur with outbreaks of HPAI in poultry, particularly commercial poultry flocks (OIE 2019a). In commercial poultry, mortality rates may approach 100% with any remaining birds euthanised through stamping out procedures (as reviewed in CFSPH 2015). AIVs have a worldwide distribution and HPAI viruses are considered to arise from genetic changes to LPAI viruses if these strains are allowed to circulate in poultry without adequate control or eradication (Dhingra et al. 2018). To date, all reported outbreaks of HPAI in poultry have been of the H5 or H7 subtypes (OIE 2019a).

Infection with AIV is an OIE listed disease and detection of HPAI of any subtype and LPAI of H5 or H7 subtypes in poultry are notifiable to the OIE. Detection of any HPAI virus in birds other than poultry, is also notifiable to the OIE. In Australia, detection of any AIV is nationally notifiable.

The department undertook a detailed assessment of the biosecurity risks and pathophysiology of AIV infection in poultry in the [Generic import risk analysis for chicken meat](#) (*Chicken meat IRA*). This chapter focuses on AIVs associated with psittacine birds. It takes account of the findings of the *Chicken meat IRA* and also includes any relevant scientific information on disease epidemiology, pathogenesis, diagnosis and treatment that has been published since the *Chicken meat IRA* was released.

4.1.2 Technical information

Epidemiology

AIVs affect a range of domestic and captive birds in many areas of the world (as reviewed in Alexander 2000b). Compared to other avian orders, the frequency of AIV detection in psittacine birds is low and they are not considered to have a major role in the ecology and epidemiology of these viruses (Alexander 2000b; Hawkins et al. 2006; Perkins & Swayne 2003). Notwithstanding this, AIVs have been detected in a range of psittacine species from several countries. As

international trade in psittacine birds is common, a large proportion of AIV detections in psittacine birds have been made through the routine testing that occurs when birds are held in quarantine after having been imported (Pasick et al. 2003). Parrots with AIV infection display a range of clinical presentations, from subclinical infection to peracute disease (Hawkins et al. 2006). Furthermore, viruses related to zoonotic AIVs have been detected in parrots indicating that these birds may potentially act as sources for human infection (Mase et al. 2001).

Hosts/susceptible species

Several types of psittacine birds have been shown to be susceptible to AIV infection (Hawkins et al. 2006; Jiao et al. 2012a; Jiao et al. 2012b; Mase et al. 2001; Panigrahy & Senne 2003), including parakeets, budgerigars, cockatiels, lovebirds and red-lored Amazon parrots.

The Influenza Research Database (fludb.org) indicates that the vast majority of infections in psittacine birds are reported in budgerigars with the remaining made in blossom-headed parakeets, cockatiels, and rose-ringed parakeets.

A study conducted by Pillai and colleagues (Pillai et al. 2008) assessed the susceptibility of poultry species to an LPAI virus subtype H5N2 isolated from a naturally infected red-lored Amazon parrot. For each species (chickens, ducks and turkeys), 11 birds were infected intratracheally and mixed with 4 in-contact conspecifics. While no bird in this experiment showed clinical signs of disease, all experimentally inoculated and in-contact birds seroconverted and virus was commonly detected from tracheal swabs. These results show that poultry are susceptible to infection with parrot-derived AIVs and horizontal transmission from bird to bird is efficient (Pillai et al. 2008). The authors comment that the undetected circulation of LPAI viruses in poultry can give rise to mutations that produce HPAI viruses. Similarly, in a study conducted by Mase and colleagues (2001) LPAI H9N2 virus from naturally infected Indian ring-necked parakeets did not result in clinical disease in experimentally infected chickens, however, virus was recovered from tracheal and cloacal swabs suggesting horizontal transmission is possible.

Human infection with AIVs does occur with a number of subtypes showing zoonotic potential. Subtypes H5N1 and H7N9 are responsible for the majority of human infections. Illness in humans ranges from subclinical to severe and fatal disease. Human infection is most commonly associated with direct contact with infected poultry (as reviewed in CDC 2017). While there is no evidence of historical parrot to human transmission of AIVs, LPAI H9N2 viruses isolated from Indian ring-necked parakeets showed >97% genetic similarity to H9N2 subtypes isolated from humans (Mase et al. 2001). Furthermore, AIV subtype H5N1 was isolated from a caged parrot in southern China (Jiao et al. 2012a). These findings suggest that AIVs with zoonotic potential may occur in parrots.

Modes of transmission

Transmission of AIVs to parrots may occur via direct contact with infected captive or wild birds or through secondary spread facilitated by humans, e.g. transfer of faeces from infected birds to the environment of susceptible birds (as reviewed in Alexander 2000b). Transmission from parrots to other animals mainly occurs through direct contact with infectious respiratory or faecal material but also via indirect contact including via fomites (as reviewed in CFSPH 2008).

The source of AIVs that infect parrots is poorly understood. An LPAI H5N2 isolate from a red-colored Amazon parrot in the United States (US) showed genetic, antigenic and biological similarities to Guatemalan lineage AIVs detected in chickens (Pillai et al. 2008). An H5N1 isolate from a parrot in China showed >99% genetic similarity to an H5N1 virus circulating in chickens in China the previous year (Jiao et al. 2012a).

Incubation period

There is limited information available on the incubation period of AIVs in naturally infected parrots. In budgerigars experimentally infected with a chicken isolate of HPAI H5N1, clinical signs appeared 5–9 days post-infection (Perkins & Swayne 2003). In general, the incubation period depends on the dose of the virus, the route of exposure and the species exposed, as well as the ability of the virus to initiate clinical signs. For the purposes of the Terrestrial Animal Health Code, the World Organisation for Animal Health (OIE) defines the maximum incubation period for regulatory purposes as 21 days (OIE 2019a).

Persistence of agent

Environmental conditions strongly influence the duration of virus survival outside the host. High humidity and moisture levels combined with low temperatures prolong virus survival time in aerosols and faeces. AIVs can survive in faeces for at least 30–35 days at 4°C and have been isolated from dust in poultry houses up to 2 weeks post-depopulation (Webster et al. 1978). AIV is an enveloped virus and is highly susceptible to commonly used disinfectants (AHA 2011).

Distribution and prevalence

A range of AIV subtypes have been reported in the scientific literature in a range of psittacine birds around the world (Table 3).

Table 3 Detections of AIVs in psittacine birds by subtype, year of detection, bird type, country of diagnosis, and probable location of exposure

Subtype	Year	Bird type	Country of diagnosis	Location of probable exposure	Reference
H5N1	2005	Parrot	China	China	(Jiao et al. 2012a)
H5N2	2004	Red-lored Amazon parrot	United States (US)	Mexico or Central America	(Hawkins et al. 2006)
H5N2	2004	Parrot	China	China	(Jiao et al. 2012b)
H7N1	Unknown	Parrot	Ireland	Unknown	(Subbarao et al. 1998)
H9N2	1997 & 1998	Indian ring necked parakeets	Japan (post-entry quarantine)	Pakistan	(Mase et al. 2001)
H4N8	1982–1991	Parrot	US (post-entry quarantine)	Unknown	(Panigrahy & Senne 2003)
H4N6	1993	Parrot	US (post-entry quarantine)	Singapore	(Panigrahy & Senne 2003)
H4N3	1982–1991	Parrot	US (post-entry quarantine)	Unknown	(Panigrahy & Senne 2003)

Subtype	Year	Bird type	Country of diagnosis	Location of probable exposure	Reference
H3N8	1974–1981	Various	US (post-entry quarantine)	Unknown	(Senne et al. 1983)
H3N8	1982–1991	Parrot	US (post-entry quarantine)	Unknown	(Panigrahy & Senne 2003)

The Influenza Research Database indicates that most detections in psittacine birds were made in Bangladesh followed by selected Latin American countries and China. Detections in Australia have also been made in budgerigars. Information on the pathogenicity or subtypes of the viruses detected are not provided.

There is limited information available on the prevalence of AIV infection in psittacine birds, particularly in wild psittacine birds. No serosurveys are available in the literature and the majority of diagnoses have been made opportunistically through screening of captive imported birds in quarantine. It is generally considered that the prevalence of AIV infection in psittacine birds is low (Alexander 2000b; Senne et al. 1983).

Australian status

Australia has been free from HPAI and LPAI in poultry since 2013 (AHA 2018). The 2013 outbreak of HPAI H7N2 in laying chickens in Young, New South Wales, resulted in the slaughter of over 400,000 chickens across 2 premises and cost the government and the industry \$5 million (DAF QLD 2016; NSW Department of Primary Industries 2013). Outbreaks of AIVs in poultry in Australia have been attributed to known or probable contact with waterfowl or material contaminated with waterfowl faeces (AHA 2011).

Australia conducts a National Avian Influenza Wild Bird Surveillance Program (NAIWB) that utilises targeted and opportunistic sampling of healthy, diseased and dead wild birds. Between July 2005 and June 2018, over 105,000 wild birds have been tested for AIVs (WHA 2019). To date, no highly pathogenic AIVs have been identified in Australian wild birds. However, a wide range of LPAI viruses of various subtypes, including LPAI H5 and H7 subtypes, have been detected in wild birds (WHA 2019).

There has been no reported case of bird to human transmission of avian influenza in Australia (Department of Health 2015).

Pathogenesis

Pathogenesis of AIV infection has been studied extensively in poultry species. Generally, in poultry species LPAI viruses produce respiratory disease and a drop in egg production whereas HPAI viruses cause severe systemic disease with high mortalities (as reviewed in Swayne et al. 2013). The nasal cavity is the site of initial viral replication (Swayne 1997). In LPAI infection, viral infection generally remains in the respiratory and intestinal tract (Swayne 1997). In HPAI, the virus subsequently replicates in endothelial cells and then spreads systemically via the vascular or lymphatic systems and infects the visceral organs where the virus undergoes further replication (Swayne 1997). Clinical signs and death result from multi-organ failure.

It is widely known that clinical signs of AIV infection are extremely variable and depend on many factors including host species and age, virus strain and virulence, acquired immunity, presence of secondary exacerbating organisms, and environmental factors (as reviewed in Swayne et al. 2013). However, across all avian species, the ability to produce severe disease and death is associated with high replication titres of virus in the host, especially in brain and cardiac tissues (Swayne & Pantin-Jackwood 2006).

Diagnosis

Clinical signs

There are limited reports of clinical infection of psittacine birds with AIVs. Where reports are available, clinical signs range from lethargy and respiratory disease (Hawkins et al. 2006; Jiao et al. 2012a) to significant mortality (Perkins & Swayne 2003). Budgerigars experimentally inoculated with a Hong Kong-origin HPAI H5N1 virus showed moderate depression, moderate to severe neurologic signs including incoordination, opisthotonus and torticollis, and progressed to death or euthanasia between 5 and 9 days post-infection (Perkins & Swayne 2003). A 3 month-old red-lored Amazon parrot infected with an LPAI H5N2 virus, showed crop stasis, regurgitation, melaena, diarrhoea and lethargy but recovered with intensive treatment (Hawkins et al. 2006).

Pathology

Similarly, there is little published information on pathology caused by AIVs in psittacine birds. The carcasses of H5N1-inoculated budgerigars showed gross evidence of dehydration and diarrhoea, and histological lesions largely confined to the brain (Perkins & Swayne 2003). Pathological changes were not described in a report of H9N2 infection in imported parakeets (Mase et al. 2001), although 2 birds died and virus was isolated from the respiratory tract.

Testing

According to the OIE Terrestrial Manual [Chapter 3.3.4](#) for avian influenza (OIE 2019a), the diagnostic methods recommended to demonstrate individual animal freedom from infection prior to movement are virus isolation and real-time polymerase chain reaction (qPCR). Virus isolation is the 'gold standard' in clinical settings, but does not give results as rapidly as qPCR. For agent identification, samples from live birds should include both oropharyngeal and cloacal swabs.

In natural and experimental infections of psittacine birds, AIVs have been detected and isolated from pharyngeal, tracheal and cloacal swabs (Hawkins et al. 2006; Mase et al. 2001).

Little information is available on the duration of shedding of AIVs in psittacine birds. A pharyngeal-cloacal combination swab collected 2 days after admission of a lethargic parrot for veterinary care was positive for avian influenza using qPCR. Follow-up pharyngeal-cloacal combination swabs collected on day 8 post admission and 6 weeks post admission were negative for avian influenza using qPCR (Hawkins et al. 2006).

Three types of tests are available to detect anti-influenza antibodies in serum samples: the agar gel immunodiffusion test (AGID), the enzyme-linked immunosorbent assay (ELISA), and the haemagglutination inhibition test (HI). To demonstrate individual animal freedom from infection prior to movement, the OIE states that the HI test is suitable and the AGID and ELISA tests may be used in some situations but certain factors may limit their application. Commercial

ELISA kits should be validated for the specific species of interest and for the specific purpose(s) for which they are to be used (OIE 2019a). In poultry, antibodies can be detected 7–10 days after infection (Swayne & Halvorson 2003). In peracute and acute disease, birds may die before the development of an antibody response.

Treatment

There are no specific treatment options for AIV infection in birds. Aggressive supportive therapy has been shown to improve the condition of a parrot infected with LPAI H5N2 (Hawkins et al. 2006).

Control

Vaccines are available for certain subtypes of avian influenza, which may protect birds from clinical signs of disease if they are subsequently infected. Although available, routine vaccination is either discouraged or banned in many countries due to interference with the rapid detection of HPAI and other problems associated with vaccine use. Emergency vaccination has been employed in some countries once an outbreak has occurred with the aim to reduce the spread of disease (AHA 2011). Routine vaccination for AIV is not permitted in Australia.

Aviary birds, caged birds and backyard birds are at little risk if simple biosecurity measures are adopted, such as preventing mixing with wild birds and protecting feed and water supplies (Department of Agriculture and Water Resources 2017). Most commercial poultry operations in Australia maintain high biosecurity protocols that would prevent the entry of avian diseases into their flocks, including AIVs.

In Australia, avian influenza is a nationally notifiable disease. The AUSVETPLAN outlines the response policy for the detection of AIVs. Under the Emergency Animal Disease Response Agreement (EADRA), cost-sharing arrangements between government and industry bodies are in place for certain AIV detections. Under the AUSVETPLAN (AHA 2011), a detection of:

- HPAI or LPAI (H5 or H7 subtype) in poultry would result in an eradication response that would include control measures such as stamping out, possible pre-emptive slaughter, quarantine and movement controls, decontamination, tracing and surveillance.
- LPAI (H5 or H7 subtype) in captive birds other than poultry would result in control measures that may include tracing and surveillance, quarantine and movement controls and decontamination of facilities.
- Non-H5 or H7 LPAI AIVs in poultry or captive birds would not result in a response unless an assessment of the risks to animal and public health indicated otherwise.
- HPAI in wildbirds may result in a response if indicated by a risk assessment.
- LPAI in wild birds warrants no further action.

Current biosecurity measures

Imports into Australia of other avian commodities including hatching poultry eggs, live pigeons, poultry meat, egg products for human consumption, and live pet birds from New Zealand all require specific measures to manage the biosecurity risk associated with AIV. Risk management for these commodities includes country freedom from HPAI, pre-export and post-entry quarantine and testing for AIV (serology and virus isolation), and freedom from clinical signs of AIV in the consignment, the source flocks and in a 40 km radius of source flocks, quarantine facilities and premises affiliated with the source flocks.

The OIE has recommendations for the safe trade in live birds other than poultry. These recommendations are found in [Article 10.4.6](#) of the OIE Code (OIE 2019a). Risk management includes a requirement for the absence of clinical signs of infection, pre-export quarantine, and pre-export testing of a sample of the birds to show freedom from infection.

4.1.3 Conclusion

- Avian influenza is potentially present in all exporting countries.
- Infection of pet and aviary psittacine birds with avian influenza is considered rare.
- Australia has been free from HPAI and LPAI in poultry since 2013. Australia practices a stamping out policy for outbreaks of HPAI and LPAI (H5 or H7) in poultry.
- No HPAI viruses have been identified in Australian wild birds; LPAI viruses of various subtypes, including LPAI H5 and H7 subtypes, have been detected in wild birds.
- In Australia, avian influenza is a nationally notifiable disease and control measures are in place.
- An outbreak in commercial poultry originating from an infected psittacine bird is highly unlikely due to high biosecurity practices maintained in most commercial operations. High mortalities due to HPAI have been reported in psittacine birds. It is therefore likely that infection would be diagnosed quickly and reported to state authorities.
- Australia has biosecurity measures for the importation of commodities that carry a risk of AIV introduction into Australia. Avian influenza (HPAI and LPAI in poultry) is an OIE-listed disease agent and there are recommendations in the OIE Code on measures for safe trade, including for live birds other than poultry.

Therefore, the department concluded that further risk assessment for AIV was not required, however, certification will be required in accordance with the OIE Code for AIV for the import of psittacines.

The OIE Code recommendations for live birds other than poultry (OIE 2019e) include isolation and testing. As routine vaccination against avian influenza is not permitted in Australia, animals are not permitted to have been previously vaccinated.

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4.2 Avian orthoavulavirus 1

4.2.1 Background

Avian orthoavulavirus 1 (AOAV-1), formerly known as Avian paramyxovirus 1 (APMV-1), is a highly contagious virus that causes generalised viral disease of birds worldwide; notably causing significant economic impact on domestic poultry production (AHA 2014; Dimitrov et al. 2016). Under the OIE Code, virulent AOAV-1 infection in poultry is referred to as Newcastle disease (ND). AOAV-1 infection in other avian species is often referred to as ND in the scientific literature, although this does not meet the OIE definition.

Due to several name changes in recent years, the terms APMV-1 and Newcastle disease virus (NDV) still appear in the majority of scientific literature.

AOAV-1 belongs to the genus *Orthoavulavirus* of the family Paramyxoviridae. Strains of AOAV-1 vary greatly in their virulence and tissue tropism, and in susceptible birds infection induces a wide range of clinical signs and pathological lesions (Brown & Bevins 2017). AOAV-1 strains are further classified into 5 pathotypes based on clinical signs seen in infected chickens:

- 1) **viscerotropic velogenic**: highly pathogenic/virulent, haemorrhagic intestinal lesions are frequently seen
- 2) **neurotropic velogenic**: highly pathogenic/virulent, high mortality usually following respiratory and nervous signs
- 3) **mesogenic**: moderately pathogenic, respiratory signs, occasional nervous signs, but low mortality
- 4) **lentogenic or respiratory**: lowly pathogenic, mild or subclinical respiratory infection
- 5) **asymptomatic**: usually subclinical enteric infection (AHA 2014; OIE 2019f).

Due to the broad variation in virulence and clinical signs produced by AOAV-1 strains, not all are considered to cause ND for the purpose of classification as an emergency animal disease (AHA 2014). Of the above, only velogenic or mesogenic pathotypes fulfil the OIE definition of ND, for which there are specific criteria for either in vivo intracerebral pathogenicity index (ICPI) testing, or in vitro amino acid sequence determination of the virus, that must be met (OIE 2019f).

At least 250 avian species can be infected with AOAV-1 naturally or experimentally (Wang et al. 2015). Psittacine birds are very susceptible to AOAV-1 and infection can result in acute respiratory, gastrointestinal, and/or nervous disease, death, or chronic illness (AHA 2014). Subclinical infections occur and some species can act as a reservoir of virulent AOAV-1, excreting virus for at least one year (AHA 2014; Erickson et al. 1977). Pools of highly virulent AOAV-1 are thought to occur in psittacines in endemically-infected countries (AHA 2014). Infected exotic psittacine species have been responsible for numerous outbreaks of AOAV-1 globally via human-assisted movement, both legal and illegal (Diel et al. 2012; Eaves & Grimes 1978; Mase et al. 2002; Seal, King & Bennett 1995; Utterback & Schwartz 1973).

AOAV-1 is a major concern to the Australian poultry industry and a detailed description of the disease in chickens can be found in the generic import risk analysis report for chicken meat (Biosecurity Australia 2008).

Infection with Newcastle disease virus is OIE-listed and infection with virulent Newcastle disease virus is nationally notifiable in Australia (Department of Agriculture 2017; OIE 2018a). The virus is zoonotic and human infection may result in transient conjunctivitis and/or flu-like signs (AHA 2014; Evans 2011; OIE 2019f).

4.2.2 Technical information

Epidemiology

AOAV-1 viruses are a diverse group of enveloped viruses with single-stranded, non-segmented, negative sense RNA genomes (Dimitrov et al. 2016; Kim, Suarez & Afonso 2008; Miller et al. 2015). Phylogenetic analysis of isolates has separated AOAV-1 into 2 clades; class I and class II. Class I AOAV-1 are of a single genotype, are generally avirulent to chickens and are most often isolated from waterfowl, shorebirds, live market birds and occasionally captured wild birds (Miller et al. 2015; Samal et al. 2011). However, an outbreak of high virulence AOAV-1 of a class I strain occurred in poultry in Ireland in 1990 (Alexander et al. 1992). Nineteen genotypes, some with sub-genotypes, comprise Class II AOAV-1. Several of these have been responsible for significant economic damage to poultry industries worldwide (Miller et al. 2015). Evidence suggests that AOAV-1 viruses are constantly evolving and increasing in diversity (Miller et al. 2015). Immune failure following vaccination is considered related to genetic variation of strains (Wang et al. 2015). Additionally, there is evidence that very few point mutations are required for low virulence strains to become virulent. However, there is suggestion that this process may not be simple or frequent (Collins, Bashiruddin & Alexander 1993).

Hosts/susceptible species

AOAV-1 has a wide host range, with at least 27 of the 50 orders of birds reported to be capable of supporting viral replication (Alexander 2011; Seal, King & Sellers 2000). Susceptibility to and subsequent severity of disease varies between virus strain and host species, and also between breed and genetic line within a species. Low virulence strains can induce severe respiratory disease in the presence of other potentially pathogenic organisms and/or adverse environmental conditions (OIE 2019f; Seal, King & Sellers 2000). Reported susceptibility of psittacine birds to AOAV-1 varies, however, it is generally considered to be high (AHA 2014; Hirai et al. 1981).

As noted previously, human infection can result in transient infections. However, this has been primarily reported in people exposed to large quantities of virus, such as members of vaccination teams and laboratory workers exposed during workplace accidents (MacLachlan & Dubovi 2017).

Vectors

Rodents, insects and humans may act as mechanical vectors, as can fomites such as feed, water, implements, clothing, boots, sacks, egg trays and crates. The presence of faeces, such as on soiled egg shells, prolongs survival of the virus (Evans 2011; Falcon 2004; OIE 2013).

Modes of transmission

AOAV-1 is highly contagious and is transmitted via inhalation, ingestion or mucous membrane exposure to virions excreted in faeces and respiratory secretions of infected birds (Falcon 2004). The carcass can also be a source of infection with virus remaining viable well into decomposition (AHA 2014; OIE 2013). The significance of airborne spread is debated, and may be associated

with the severity of respiratory signs, population density of susceptible hosts and climatic conditions (AHA 2014; Alexander 2000a). Some strains of AOAV-1 can infect hatching chicks through an intact egg shell (AHA 2014; OIE 2013). Vertical transmission within eggs is also possible, with lentogenic strains isolated more frequently than more virulent strains (AHA 2014). Frozen contaminated meat products have been a significant source of spread in overseas outbreaks (AHA 2014).

Psittacine and other pet birds are often implicated in the spread of AOAV-1 throughout the world and highly virulent strains have been isolated from birds in quarantine stations and pet stores (Dimitrov et al. 2016). A widespread pandemic of viscerotropic velogenic AOAV-1 in the 1960s was traced to importation of salmon-crested cockatoos, originally from Indonesia, from Singapore into the United Kingdom (Eaves & Grimes 1978). AOAV-1 panzootics across Europe and North America from 1969 to 1973 were linked to imported parrots from South American countries. These parrots were thought to have contracted AOAV-1 whilst in local collecting centres/holding stations in their country of origin (Kaleta & Baldauf 1988).

Three outbreaks of virulent AOAV-1 in southern England in 1975 were attributed to 20 psittacines imported from the 'Far East' by a poultry flock owner who had placed them in a shed alongside their battery hen house (Ashton 1984). In Australia, during the 1970s, AOAV-1 capable of causing severe respiratory disease in young chickens was isolated from a cockatoo illegally imported from Indonesia; control measures including slaughter of direct/indirect-contact poultry flocks were initiated (Eaves & Grimes 1978). In Japan in 1980, a consignment of cockatoos imported from Indonesia was responsible for an outbreak of AOAV-1 in cockatiels, rosellas, parakeets and Amazon parrots at a bird dealer's premises; 225 of 345 housed psittacines died, while 200 finches (Order passeriformes) also housed in the facility were unaffected. Velogenic AOAV-1 was isolated the previous year in cockatoos imported from the same location, and the same year a velogenic AOAV-1 was isolated from diseased love birds from a department store pet shop (origin unknown) (Hirai et al. 1981).

Detection of AOAV-1 in New York, US, in 1970 was traced to quaker parrots imported from Paraguay. AOAV-1 was again introduced by imported Paraguayan parrots the following year in Connecticut. In 1971 in Florida, 5,000 pet birds were destroyed at an importer's premises following introduction of AOAV-1 via a Mynah bird (Order Passeriformes) imported from Thailand. Investigations revealed the owner, a pet bird geneticist, had shipped an additional 176 consignments of AOAV-1-exposed birds to pet shops in 35 US states and internationally (Walker, Heron & Mixson 1973). A larger AOAV-1 outbreak in 1972 affecting poultry in California, was traced to illegally imported Mexican double yellow-headed parrots from South America. This introduction resulted in the destruction of 12 million birds at a cost of US\$56 million (Panigrahy et al. 1993; Walker, Heron & Mixson 1973). Further AOAV-1 introductions occurred in numerous US states throughout 1972 via parrots and other bird species imported from Colombia, Ecuador, Mexico, Thailand and elsewhere (Walker, Heron & Mixson 1973). That same year, US government restrictions were placed on importation of psittacine birds and 2 species of Mynah bird, requiring pre-export and post-entry quarantine in order to prevent further introductions of AOAV-1 (Walker, Heron & Mixson 1973). From 1974 to 1981, 2,274 lots of birds (over 2.8 million individual birds) were imported into the United States and underwent 30 days post-entry quarantine. AOAV-1 was isolated from 173 lots, a virulent strain being detected in 147 of these. The majority (72%) of virulent AOAV-1-affected lots consisted of psittacine birds, and clinical disease was frequently reported. In 1991, AOAV-1 was isolated in only 3 of 160 lots of

imported quarantined birds, 2 held in United States Department of Agriculture (USDA)-supervised private facilities and one in a USDA-operated facility; all 3 groups contained psittacine species (Panigrahy et al. 1993).

The number of virulent AOAV-1 detections in birds imported into the United States decreased from 1974 to 1981. Senne et al. (1983) attribute this decrease to importer discrimination on species of birds and countries of origin; pre-export quarantine; a 90 day ban on issuance of permits for countries following a virulent AOAV-1 isolation in birds; and population dynamics in the countries of origin including reduced numbers of wild birds and decreasing perpetuation of infectious diseases. Illegally imported (smuggled) pet birds remain a potential source of virulent AOAV-1.

Although human infections can occur, they are uncommon and there is no evidence of human-to-human transmission (OIE 2019f).

Incubation period

The incubation period of AOAV-1 is 2–28 days, averaging 5–6 days. It is shorter in younger birds and has a maximum of 21 days for the purposes of the OIE Code (AHA 2014; OIE 2019f; Ritchie 1995a). Experimental infection of a group of psittacine birds with a viscerotropic velogenic AOAV-1 strain demonstrated an incubation period of 3–14 days, while the range in a naturally infected group was 5–16 days (Ritchie 1995a). In humans, the reported incubation period is 6–7 days (AHA 2014), however, conjunctivitis can develop within 24 hours of viral exposure to the eye (OIE 2019f).

Persistence of agent

Psittacines may be subclinically infected with virulent AOAV-1 and may shed virus intermittently for over a year (Erickson et al. 1977). This carrier-like state has been associated with introduction of the virus into poultry (AHA 2014; OIE 2013).

AOAV-1 is relatively heat stable, requiring heat treatment of 70°C for 8.2 minutes to achieve a 6 log reduction of virus in homogenized chicken meat with a 15% skin and fat content (Alexander 1997; Alexander & Manvell 2004). Direct sunlight will inactivate AOAV-1 within 30 minutes (AHA 2014). The virus is sensitive to ether and is inactivated by formalin, phenolics and oxidising agents, such as 6% sodium hypochlorite, chlorhexidine, acids with pH ≤ 2 and alkalis such as sodium hydroxide and sodium carbonate anhydrous (AHA 2014; FAO 2001; OIE 2019f).

In slaughtered infected chickens, AOAV-1 can remain infectious in bone marrow and muscle for up to 4 months at refrigerator temperatures, and at least 6 months at –20°C (AHA 2014). Eggs laid by infected chickens can harbour infectious virus for months at room temperature and for more than a year at 4°C. Similar survival times have been observed on contaminated feathers and in contaminated premises (AHA 2014).

AOAV-1 is environmentally stable with prolonged infectivity under favourable environmental conditions (Davis-Fields et al. 2014; Dimitrov et al. 2016; OIE 2013). Studies indicate that AOAV-1, even at low titres, may remain infective for years in 17°C water (Davis-Fields et al. 2014). Contaminated water has been suggested as a possible environmental reservoir of virus, a facilitator of interspecies transmission and a means of possible spill-over from wild birds to domestic poultry (Davis-Fields et al. 2014; Dimitrov et al. 2016; Snoeck et al. 2013). The virus also survives for long periods in faeces (OIE 2013).

Distribution and prevalence

Lentogenic strains of AOAV-1 are distributed worldwide (OIE 2013). Virulent strains are endemic in areas of Mexico, Central and South America, many parts of Asia, the Middle East and Africa, and in wild birds in the United States and Canada. AOAV-1 is also endemic in all of Indonesia, East Timor and South-East Asia, with West Papua being the closest infected area to Australia (AHA 2014). Following exposure, AOAV-1 is shed during incubation, clinical disease and for a limited time during recovery (OIE 2013). Wild gulls, waterfowl and shorebirds may be reservoir hosts for lentogenic pathotypes, which can become virulent following mutation in domestic poultry (Dimitrov et al. 2016; OIE 2013; Vidanović et al. 2011). There is evidence on the basis of strain similarity that successive outbreaks of AOAV-1 from 1996 to 2005 in Denmark, Finland, Sweden and the United Kingdom were due to multiple virus introductions from the same pool of wild birds (Snoeck et al. 2013). Wild cormorants in Canada and the United States have maintained virulent AOAV-1 infections over many years, with spill-over into domestic turkeys in some instances (Alexander 2000a). An outbreak of velogenic AOAV-1 in little owls in an Israeli zoo in 2011 was suspected to have been introduced from migratory birds (e.g. waterfowl) or passeriformes shedding virulent virus on zoo grounds, although the feeding of infected chicks was also considered (Haddas et al. 2013). It has been shown that captive caged birds are frequently infected with virulent AOAV-1 viruses; in some instances these have caused outbreaks of disease in commercial and backyard poultry (AHA 2014).

It is worth noting that there are widespread mesogenic pathotypes of variant virulent AOAV-1 strains circulating, such as the highly pigeon-specific pigeon paramyxovirus (PPMV-1) (Aldous et al. 2012; OIE 2013). PPMV-1 strains exhibit a broad range of pathogenicities for poultry, which may increase following serial passages in chickens (Snoeck et al. 2013). In many instances these variant AOAV-1 strains do not appear to readily infect other avian species. However, there are examples of PPMV-1 causing outbreaks of ND in domestic poultry and game birds across Europe (Aldous et al. 2012; Alexander 2011; Seal, King & Sellers 2000; Snoeck et al. 2013).

Australian status

Australia experienced outbreaks of ND in commercial poultry in Victoria in 1930 and 1932, both of which were successfully eradicated. An avirulent strain of AOAV-1 identified in Queensland in 1966, rapidly spread across Australia and since then a number of avirulent and lentogenic strains have emerged. Outbreaks of ND from 1998 to 2002 in New South Wales and Victoria were a result of mutations in one or more of these strains. Australia is currently free from ND, with vaccination, in accordance with the OIE definition of an ND-free country (OIE 2018b).

PPMV-1 is present in domestic and wild pigeons in Australia and is notifiable in Victoria, New South Wales and Western Australia. However, as clinical signs are comparable to ND, any signs of disease should be notified in all states and territories (Agriculture Victoria 2018; NSW DPI 2015; WA Department of Primary Industries and Regional Development 2017).

Pathogenesis

The progression of AOAV-1 infections varies between different avian species and factors influencing pathogenicity are not completely understood (Ritchie 1995a; Senne et al. 2009).

At a molecular level, post-translation cleavage of F protein (F₀) into F₁ and F₂ is required for AOAV-1 virus particles to be infectious (OIE 2019f; Senne et al. 2009). The sequence of amino acids at the cleavage site is a major determinant of virulence (Senne et al. 1983; Snoeck et al.

2013). Aside from the F0 cleavage site, the amino acid sequence of both HN and V proteins have been shown to contribute to differences in virulence and, in the case of HN, differences in tissue tropism (Huang et al. 2003; Huang et al. 2004; Senne et al. 2009).

Virulent strains will bind to erythrocytes and spread rapidly; in chickens, virus is detectable in most tissues by 24 hours post exposure (Ritchie 1995a). AOAV-1 damages vascular endothelial cells, commonly resulting in haemorrhage. Some strains preferentially infect the intestinal tract; others infect respiratory tract mucosal cells (from the nasal passage to lungs), with the central nervous system is generally invaded later in the disease process (Ritchie 1995a).

Diagnosis

Clinical signs

AOAV-1 produces a wide range of clinical signs which are dependent on the strain/pathotype (including virulence and tissue tropism factors), host species, route of exposure and magnitude of infecting dose, host factors (age, breed, immune status and condition), co-infection with other organisms, and environmental/husbandry conditions (AHA 2014; Alexander 2011; Kommers, King & Brown 2003; OIE 2013; Seal, King & Sellers 2000).

Clinical signs in psittacine species are usually predominately neurological. However, respiratory, gastrointestinal, ocular, and more generalised signs such as lethargy, weight loss and ruffled plumage, can occur (AHA 2014; Erickson et al. 1977; Harcourt-Brown 2000). Some cases that survive acute disease may have persistent neurological signs including ataxia, tremors, torticollis, opisthotonus, head bobbing, chorea and paralysis (Erickson et al. 1977; Harcourt-Brown 2000; Harrison & Lightfoot 2006).

Psittacines can also act as reservoirs of AOAV-1 viruses, including virulent pathotypes, shedding virus without showing clinical signs (AHA 2014; Erickson et al. 1977; Harrison & Lightfoot 2006; Ritchie 1995a).

In chickens, velogenic strains commonly cause severe disease, primarily of respiratory and/or nervous systems (OIE 2013). Initial signs may include lethargy, prostration, inappetence, ruffled feathers, oedema and injection of conjunctiva; progressing to diarrhoea, dyspnoea, and head/neck inflammation (often with cyanosis). Neurological signs may appear including tremors, tonic/clonic spasms, paresis, paralysis, torticollis and circling. There is usually a sharp decrease in egg production and eggs may appear malformed with abnormally coloured, rough or thin shells (AHA 2014). In some infections, sudden death may occur with few or no clinical signs (OIE 2013) and flock mortality may reach 100% (AHA 2014; Alexander 2011).

Compared to chickens, clinical signs in turkeys are usually less severe, however, effects on egg production are comparable (OIE 2013). Quail tend to be very susceptible to disease. Signs in ducks, geese, peafowl, guinea fowl, pheasants and canaries are usually mild (or absent altogether), although there are a few reports of clinical disease in these species. Ratites appear to be fairly resistant to clinical disease, as do raptors, however, acute disease in some species has been reported (AHA 2014; OIE 2013).

Wild birds, including waterfowl and passeriform species can harbour and shed both avirulent and virulent AOAV-1 viruses, with or without clinical signs (AHA 2014; Miller et al. 2015; OIE 2013). These infections can spill-over into other wild or domestic bird populations and cause clinical disease, at times resulting in outbreaks of economic importance (Miller et al. 2015).

Pigeons and doves can also carry and disseminate AOAV-1 (and PPMV-1) without displaying clinical signs (Seal, King & Sellers 2000). However, infection in these species can result in clinical signs similar to those outlined for chickens (NSW DPI 2015).

Vaccination may greatly diminish the severity of clinical signs seen in relation to the level of antibody achieved, and may mask clinical disease (AHA 2014; Alexander 2011). In the United States, vaccination of imported birds is prohibited as it does not eliminate the carrier state and hampers viral detection during quarantine (Hoppes 2016).

Pathology

Numerous lesions can be associated with AOAV-1 but none are considered pathognomonic (OIE 2013). In young birds or in the case of a strain that causes rapid death, no gross lesions may be evident (AHA 2014).

Following infection with the viscerotropic form of AOAV-1, clinical signs can include swelling of the periorbital area or head, oedema of neck tissues (particularly at the thoracic inlet), pharyngeal/tracheal mucosal congestion, haemorrhage and the presence of diphtheric membranes, proventricular mucosal petechiae and ecchymoses, ovarian oedema, haemorrhage or degeneration, petechiae on heart and breast muscle, adipose tissue and serosal surfaces, and damage to respiratory and digestive lymphoid tissue including haemorrhage, oedema, necrosis or ulceration (often involving Peyer's patches) (AHA 2014; OIE 2019f).

Erickson et al. (Erickson et al. 1977) reported that following exposure of a number of avian species to a viscerotropic velogenic AOAV-1, gross lesions in budgerigars and parrots were not comparable to those induced in chickens. Conures, like chickens, developed haemorrhagic visceral lesions, however the conures' lesions were restricted to the proventriculus, proventricular junction and small intestine (rather than the proventriculus, ventriculus, and lymphoid aggregates of the upper and lower intestinal tract, as seen in chickens).

The neurotrophic form may cause severe haemorrhagic tracheal inflammation with minimal damage to the alimentary tract other than occasionally in the proventriculus. Partial immunity will minimise the severity of gross lesions, proportional to the degree of immunity (AHA 2014).

Histopathological examination of brain lesions may find hyperplasia of vascular endothelium (often characteristic), neuronal degeneration, gliosis and perivascular lymphocytic infiltration. Other organs may display necrosis of vascular endothelial lining, thrombosis, oedema and haemorrhage. The submucosa of the nasal tract, trachea, lungs and air sacs may have pronounced oedema and cellular infiltration (AHA 2014).

Testing

The wide variation in clinical signs and lesions observed in affected birds means that neither can be used as a reliable basis for diagnosis of AOAV-1 (OIE 2013). Virus isolation is the prescribed test for international trading purposes. Molecular techniques that determine the characteristic F0 cleavage site sequence of a virulent strain is one criterion the OIE recommends to define an outbreak of ND. The other that can be used is *in vivo* intracerebral pathogenicity index testing in day old chickens (where a value of 0.7 or greater confirms ND, and which must be performed if molecular testing fails to demonstrate the characteristic amino acid sequence) (OIE 2019f).

Virus identification techniques such as the haemagglutination assay, HI and polymerase chain reaction (PCR) based techniques can be employed. However, cross-reactivity with other paramyxoviruses may result in mistyping of isolates (with HI testing) and genetic variability may lead to false negatives in genetic-based tests (OIE 2019f). Mouse monoclonal antibodies directed against specific AOAV-1 strains or variant isolates have been used in HI tests to reduce cross-reactions (OIE 2019f). Real-time PCR has been employed to eliminate the need for post-amplification processing (as is required with standard PCR systems). However, this should only be used as a screening test as it does not discriminate between lentogenic, mesogenic and velogenic strains (OIE 2019f). In addition, some highly divergent AOAV-1 strains may escape detection by PCR due to significant heterogeneity in their genomes (Kim et al. 2007).

AOAV-1 can be used as an antigen in serological tests including neutralisation or ELISA and HI, for assessing antibody levels in birds (OIE 2019f). The diagnostic value of these tests is therefore related to the expected immune status of birds tested. Titres in chickens are detectable 6–10 days following infection, peak after 3–4 weeks, and are undetectable by 8–12 months (AHA 2014). Expected titres (response and duration) in aviary birds following natural infection with lentogenic pathotypes are unknown (AHA 2014). Serology can be used to monitor vaccinated flocks, however, it should be noted that infections with other paramyxoviruses in AOAV-1-vaccinated birds may increase AOAV-1 titres substantially (OIE 2019f).

Control

Birds may be vaccinated for AOAV-1 with live or inactivated vaccines, usually at no earlier than 1–2 weeks of age to reduce interference by maternal immunity (AHA 2014). Vaccine-induced immunity lasts 10–12 weeks and repeat vaccinations are required to maintain adequate protection (AHA 2014).

Most commercial chicken flocks in Australia vaccinate against AOAV-1 using live and inactivated lentogenic-strain vaccines. Additionally, some Australian flocks have partial immunity from natural exposure to non-pathogenic strains of AOAV-1. These, and vaccinated, flocks can be a risk for maintenance of virulent AOAV-1 virus, as they can be infected and excrete virulent virus intermittently, particularly when subjected to stresses (e.g. transport, concurrent disease), while remaining subclinical (AHA 2014).

Any vaccination of birds may mask clinical disease and hamper viral detection techniques.

Current biosecurity measures

Infection with virulent AOAV-1 is notifiable throughout Australia and the department monitors for the disease in birds across northern Australia through the Northern Australia Quarantine Strategy (NAQS) surveillance program. The department has in place restrictions on the import of goods into Australia that may pose a risk of AOAV-1 introduction. These are based on risk assessments that determine what, if any, measures are required to reduce the risk to Australia's appropriate level of protection (ALOP).

At present the import of live birds, other than pigeons, is only permitted from New Zealand (OIE-listed as ND free) under certain conditions. Pigeons can be imported from Canada, France, Germany, Ireland, Netherlands, New Zealand and United Kingdom and under more comprehensive conditions, including pre-export quarantine in approved premises, post-entry quarantine, source flock and premises ND-freedom certification and various tests and treatments.

Fertile bird eggs (domestic duck, turkey and hen only) can be imported from Canada, France, Germany, Ireland, Netherlands, New Zealand, United Kingdom and United States, again under conditions that manage the risk of AOAV-1 introduction. The process is lengthy, requiring pre-export quarantine of the source flock with testing and treatments, additional monitoring and testing of the source flock after egg collection, and at least 12 weeks post-entry quarantine with additional testing and sentinel bird surveillance. The specific requirements vary between commodities and whether or not vaccination of the source flock against AOAV-1 has been performed (permissible for domestic hen and turkey only).

The import of food and other products that may pose a risk of AOAV-1 introduction (e.g. poultry meat, products containing egg) are also subject to biosecurity measures that achieve Australia's ALOP. For example, imported chicken meat from AOAV-1 infected countries must be treated in a commercial heating process using moist heat to a minimum core temperature of 70°C for at least 8.2 minutes, or equivalent time and temperature (Biosecurity Australia 2008).

The OIE has recommendations for the safe trade in live birds other than poultry. These recommendations are found in [Article 10.9.5](#) of the OIE Code (OIE 2019a). Risk management includes a requirement for the absence of clinical signs of infection, pre-export quarantine, and pre-export testing of a sample of the birds to show freedom from infection.

Conclusion

- Virulent AOAV-1 is not present in Australia; avirulent strains of AOAV-1 are endemic.
- Virulent AOAV-1 is endemic in many areas of the world, including parts of Mexico, Central and South America; many parts of Asia, the Middle East and Africa; and in wild birds in the United States and Canada.
- Virulent AOAV-1 is a nationally notifiable disease in Australia.
- AOAV-1 can cause clinical disease and mortalities in psittacine birds and they are considered highly susceptible to infection.
- Psittacine imports have been linked to outbreaks of AOAV-1 in commercial poultry.

Therefore, the department concluded that further risk assessment of virulent AOAV-1 was required.

Treatment

Treatment of birds with ND is ineffective (AHA 2014).

4.2.3 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of virulent AOAV-1 being present in imported psittacine birds:

- All psittacine species are considered highly susceptible to infection.
- Virulent AOAV-1 is present in many overseas countries.
- Pools of highly virulent AOAV-1 are likely to occur in psittacines in endemically-infected countries.
- The incubation period varies from days to weeks and clinical signs can be severe, although subclinical infections may occur. Sub-clinical carrier birds shed virus intermittently.

- Vaccination can reduce clinical signs of disease.
- The importation of psittacine birds has been associated with AOAV-1 outbreaks.

Conclusion: based on this information the likelihood of importation of virulent AOAV-1 associated with psittacine birds was estimated to be **moderate**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to virulent AOAV-1 via an infected imported psittacine bird:

- Transmission is via aerosols and the faecal-oral route. Rodents, insects, humans and other animals can act as mechanical vectors. AOAV-1 can be spread by fomites including contaminated feed, dust and feathers.
- AOAV-1 is highly contagious.
- Poultry and other avian species are susceptible to infection with AOAV-1 from psittacine birds. Mild human infection is possible.
- The exposure group is considered to be captive birds, wild birds, and low biosecurity poultry (backyard and free-range commercial poultry).
- AOAV-1 is susceptible to environmental inactivation, however, prolonged infectivity exists under favourable conditions.

Conclusion: based on this information the likelihood of susceptible species in exposure groups being exposed to virulent AOAV-1 associated with psittacine birds was estimated to be **high**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of virulent AOAV-1 in imported psittacine birds was estimated to be **moderate**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible species to virulent AOAV-1 has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to virulent AOAV-1 was considered to be a **widespread outbreak**, whereby virulent AOAV-1 establishes in directly exposed populations (captive birds, wild birds, and/or low biosecurity poultry), spreads to other populations of wild birds and/or low biosecurity poultry, and becomes endemic in Australian wild birds.

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario:

- AOAV-1 is highly contagious.
- AOAV-1 can persist in the environment and be spread by mechanical vectors and on fomites.
- Psittacine birds can be a subclinical reservoir of AOAV-1 infection and shed virus.
- Psittacine birds are likely to spread the virus to wild bird populations including Passeriformes which may contribute to the virus becoming endemic.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of virulent AOAV-1 through populations of captive birds, wild birds and/or poultry was **moderate**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of virulent AOAV-1 were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of virulent AOAV-1.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Infection with AOAV-1 can cause clinical disease and mortalities in susceptible species.
- There would be a loss of production in low biosecurity commercial poultry establishments.
- Humans can be mildly affected and minimal public health impacts.
- Based on these considerations, the effect of the establishment and/or spread of AOAV-1 for this criterion was estimated to be of minor significance at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- High mortalities in wild bird populations are expected.
- There would be some environmental contamination concerns relating to carcase disposal of wild birds and as part of eradication program in commercial poultry flocks.
- Based on this consideration, the effect of the establishment and/or spread of AOAV-1 for this criterion was estimated to be of minor significance at the national level.

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Control strategies and programs initiated would be as per the AUSVETPLAN manual including stamping out, movement controls, surveillance and vaccination.
- Associated costs in commercial poultry would be shared in accordance with the EADRA.
- Both commercial producers and owners of backyard poultry may have to vaccinate as part of the response policy or may choose to vaccinate if they do not already, at an additional expense.
- Based on this consideration, the effect of the establishment and/or spread of AOAV-1 for this criterion was estimated to be of minor significance at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- There would likely be negative effects on poultry producers, supplying industries (feed, litter, etc.) and those using outputs (eggs, meat, etc due to impacts on supply and price).
- Vaccine manufacturers may see increased demand.
- Movement controls associated with control and eradication programs may interfere with trading patterns.
- Based on this consideration, the effect of the establishment and/or spread of AOAV-1 for this criterion was estimated to be of minor significance at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- The effect on international trade would depend on the species infected and the export health certification agreed with trading partners for each commodity impacted.
- Infection of poultry with virulent AOAV-1 is notifiable to the OIE and would likely result in trade restrictions. There would be cessation of exports in the short term, with recommencement after a period of review by, and negotiations with, trading partners. Some markets may not return to normal trade for some time following ND eradication in poultry.
- Virulent AOAV-1 detected in pet, aviary or wild psittacines only should not result in trade restrictions in poultry or other commodities.
- Based on this consideration, the effect of the establishment and/or spread of AOAV-1 for this criterion was estimated to be significant at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- Outbreak in wild birds may cause a reduction in biodiversity if high mortalities are encountered. It is likely that any negative effects on the environment would be minor.

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- An outbreak in commercial poultry would impact communities in affected areas, depending on the scale of the outbreak. A large outbreak may impact supplies of poultry and poultry products.
- Movement restrictions and suspension of community activities due to control/eradication measures (if the outbreak occurs in an area close to commercial poultry farms) could result in significant levels of community concern.
- Based on this consideration, the effect of the establishment and/or spread of AOAV-1 for this criterion was estimated to be significant at the national level.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **moderate**.

Estimation of the likely consequences

The estimate of the likelihood of establishment and/or spread for the scenario (**moderate**) was combined with the overall effect associated with the outbreak scenario (**moderate**) using Figure 4 to obtain an estimation of likely consequences of **moderate**.

Risk estimation

Using Figure 5, the likelihood of entry and exposure (**moderate**) was combined with the likely consequences of establishment and/or spread (**moderate**), which resulted in a risk estimation of **moderate**.

Therefore, as the unrestricted risk estimate does not achieve Australia's ALOP, specific risk management is considered necessary for AOAV-1.

Chapter 4.11 proposes a combination of risk management measures to reduce the above likelihood of entry and exposure from **moderate** to **very low** in order to result in an overall risk estimate of **very low** and achieve Australia's ALOP.

Key features of virulent AOAV-1 to address include: the presence of pools of highly virulent AOAV-1 in psittacines in endemically-infected countries; an incubation period ranging from days to weeks; clinical signs that vary from nil to severe; difficulty in clarifying a bird's disease status via diagnostic testing; and a carrier state where birds (particularly psittacines) may only shed virus intermittently.

The OIE Code prescribes an incubation period for AOAV-1 of 21 days. However, as described above, the literature reviewed recognises that the incubation period may be up to 28 days. The OIE Code also prescribes a range of pre-export measures including pre-export quarantine for 21 days and diagnostic testing within 14 days prior to export. The measures prescribed in the OIE Code were assessed as insufficient to meet Australia's ALOP. This is because they do not account for the potentially longer incubation period recognised in the literature, they allow for testing before a full incubation period has passed in pre-export quarantine, and they do not adequately recognise the potential for psittacines to act as subclinical reservoirs of infection. Proposed measures to achieve Australia's ALOP therefore include:

- A requirement for the bird to undergo suitable laboratory testing to confirm freedom from AOAV-1 both before export and after arrival in Australia.
- A requirement for the bird to spend at least 28 days in pre-export quarantine before sampling for laboratory testing is carried out. This allows sufficient time for disease expression to occur in the event that a bird is infected immediately prior to pre-export quarantine, and so maximises the potential for laboratory testing to identify an infection.
- A total pre-export quarantine period of at least 35 days immediately before export. This allows sufficient time for clinical signs of disease to manifest and be recognised and also provides an allowance for laboratory testing results and official certification to be obtained ahead of the scheduled export.
- A post-entry quarantine period of at least 15 days. This provides time to identify clinical illness in imported birds and it accounts for the fact that infection can be subclinical and can revert to clinical illness following a stressful event (such as international transport). It also provides a period of time for laboratory testing to be completed and reports finalised, and for official import documentation to be assessed prior to release.

4.2.4 References

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4.3 Avian paraavulavirus 3

4.3.1 Background

Avian paraavulavirus 3 (APAV-3), formerly known as avian paramyxovirus 3 (APMV-3), was first isolated from turkeys in Canada in 1967, followed by another isolation in the United States in 1968 (Tumova, Robinson & Easterday 1979). APAV-3 is a member of the genus *Paraavulavirus*, in the Paramyxoviridae family. There are 2 broad groups of APAV-3 isolates—one group of isolates are mainly found in turkeys and the other in psittacine and passerine birds (Anderson et al. 1987). APAV-3 infection has been associated with mild respiratory signs and decreased egg production in infected turkey flocks (as reviewed in Mundt 2013). In psittacine and passerine birds, APAV-3 infection can range from subclinical infection to severe disease, especially in smaller sized birds (Jung et al. 2009; Schmidt, Reavill & Phalen 2015).

APAV-3 is not an OIE-listed disease, is not a nationally notifiable disease in Australia and is not known to be a human pathogen. APAV-3 has not been isolated from any avian species in Australia (Department of Agriculture and Water Resources 2014).

4.3.2 Technical information

Epidemiology

APAV-3 isolates show considerable diversity—turkey APAV-3 isolates exhibit antigenic differences from APAV-3 viruses isolated from psittacine and passerine birds (Anderson et al. 1987). Even within turkey isolates, there are distinguishable differences between the viruses isolated from different geographical regions (as reviewed in Mundt 2013).

Hosts/susceptible species

It appears that within poultry, natural APAV-3 infection is restricted to turkeys, although chickens have been shown to be fully susceptible to experimental infection (Kumar et al. 2010). In psittacine birds, disease is reported most frequently in *Neophema* species, lovebirds, cockatiels and Amazon parrots, although recently the disease also has been seen in African grey parrots (as reviewed in Phalen, 2006). Infection in passerine birds can be occasionally seen, particularly in small species that are in contact with infected psittacine birds (Schmidt, Reavill & Phalen 2015; Shihmanter et al. 1998).

While APAV-3 has been isolated from wild birds, there have been no reports of disease associated with APAV-3 in wild birds (Maldonado et al. 1994).

Modes of transmission

Similar to other paramyxoviruses, the primary routes of transmission appear to be via the faecal-oral and respiratory routes (as reviewed in Mundt 2013). APAV-3 viruses appear to spread slowly between animals and between flocks (as reviewed in Alexander 2000a).

Incubation period

Earlier reports on APAV-3 in turkeys describe the onset of respiratory signs from day 2 after infection of adult turkeys (Tumova, Robinson & Easterday 1979). In another report of infection in captive psittacine birds in Germany, clinical signs of disease were first seen 2 weeks after the introduction of 10 new birds (Jung et al. 2009).

Persistence of agent

In the absence of any specific reports on the inactivation of APAV-3, it is assumed that its spectrum of sensitivity is similar to AOAV-1, which is most sensitive to phenols and glutaraldehyde but resistant to quaternary ammonium compounds (refer to 4.2.2 for further information).

Distribution and prevalence

APAV-3 viruses have been documented in turkeys in the Canada, France, Germany, Spain, United Kingdom and United States (Anderson et al. 1987; Andral & Toquin 1984; Maldonado et al. 1994; Schemera et al. 1987; Smit & Rondhuis 1976; Tumova, Robinson & Easterday 1979).

Infection in captive birds has been documented in Germany, Israel, Netherlands, United Kingdom and United States (Alexander 2000a; Jung et al. 2009; Shihmanter et al. 1998). However, as reviewed by Alexander (2000a), it appears that APAV-3 is probably widespread as it has been isolated from captive caged birds in all countries that monitor such birds.

Australian status

APAV-3 has not been isolated from any avian species in Australia (Department of Agriculture and Water Resources 2014).

Pathogenesis

Little is known about the pathogenesis of APAV-3. APAV-3 infection appears to affect different host species differently, and it is likely that different isolates exhibit different pathogenicity (as reviewed in Mundt 2013).

Experimental infection by Kumar and colleagues (2010) in young chickens and turkeys has demonstrated systemic infection without clinical disease. Virus was detected in the brain, lung, spleen, trachea, pancreas and kidney. Another recent study confirmed the neurotropism of APAV-3 in day-old chicks and 2-week-old chickens following experimental intranasal infection (Kim et al.). Results show that despite neuroinvasion, APAV-3 infection in chickens is not neurovirulent (Kim et al. 2012).

Diagnosis

Clinical signs

In turkeys, reports indicate that clinical signs are mainly related to egg production problems, which may be preceded by mild respiratory disease (Alexander 2000a; Tumova, Robinson & Easterday 1979). Recent experimental studies have shown mild respiratory and gastrointestinal clinical signs in day-old chicks and turkeys, and an absence of clinical signs in 2-week-old chickens and turkeys (Kumar et al. 2010). It is likely that older poultry birds are more resistant to developing clinical disease (Kumar et al. 2010).

In captive birds, signs of infection can range from subclinical to severe disease. Some birds may develop encephalitis and display severe central nervous system signs (Jung et al. 2009). Chronic infections, particularly in *Neophema* species, can result in chronic pancreatitis. These birds produce voluminous stools with undigested fats (as reviewed in Phalen 2006). Non-specific clinical signs such as weakness, anorexia, vomiting and sneezing have also been noted and some birds may die within 24-48 hours of developing clinical signs (Phalen 2006; Shihmanter et al. 1998).

Pathology

Experimental infection of young poultry birds by Kim and colleagues (2012) identified mild pathological changes mainly in the respiratory tract, including a mild tracheitis and bronchitis. Another experimental infection of young birds produced focal pancreatic necrosis, but no lesions in the respiratory tract (Kumar et al. 2010).

In captive birds, gross lesions are rarely seen. In birds with encephalitis, inflammatory changes may not always be seen. In some birds, histopathological changes due to chronic pancreatitis can be observed (particularly in *Neophema* species) and the liver may be grossly enlarged (Schmidt, Reavill & Phalen 2015).

Testing

Diagnostic test types for APAV-3 are identical to those for AOAV-1 (as reviewed in Mundt 2013). For virus isolation, samples from live birds should include both tracheal or oropharyngeal and cloacal swabs, the latter should be visibly coated with faecal material. Samples from dead birds should be taken from grossly affected tissues, namely from lung, kidneys, intestine (including contents), caecal tonsils, spleen, brain, liver and heart tissues. Virus can be isolated through inoculation of embryonated SPF eggs or through cell culture (OIE 2019f).

Identification of virus can be performed through HI testing or PCR of bacteriologically sterile fluids harvested from inoculated eggs. Due to the known cross reactivity in HI testing between AOAV-1 and APAV-3 (especially with psittacine isolates), the risk of mistyping the isolate can be greatly reduced by using a panel of reference sera or specific monoclonal antibodies (OIE 2019f).

Serological testing for APAV-3 antibodies should be interpreted with caution as antibodies for APAV-3 may be detected in turkeys and chickens showing high vaccine-induced titres to AOAV-1 (Nayak et al. 2012). Newcastle disease vaccinated birds infected with APAV-3 show a rise in HI titre to both viruses (Nayak et al. 2012). In captive birds chronically infected with APAV-3, the HI test may not detect antibodies (as reviewed in Phalen 2006).

Treatment

There is no treatment for APAV-3 infection.

Control

A high level of biosecurity is seen as the main mechanism of control. However, good hygiene, disinfection and allowing time before restocking does not always prevent infection in subsequent flocks (as reviewed in Alexander 2000a). Vaccination with inactivated APAV-3 virus (psittacine isolate) appears to provide protection in psittacines—such a vaccine does not currently appear to be commercially available (Beck et al. 2003).

Current biosecurity measures

Currently, the only live psittacine birds that may enter Australia are pet birds from New Zealand, and there are no specific biosecurity measures related to APAV-3 associated with these imports (New Zealand has not detected APAV-3 in psittacines or turkeys). There are no OIE recommendations regarding APAV-3.

There are biosecurity measures relating to APAV-3 in place for the importation of hatching eggs of domestic hens and turkeys. These include:

- Poultry flocks producing eggs for export to Australia (source flocks) must be certified as free from APAV-3 during the 90 days prior to egg collection.
- Eggs must undergo fumigation or disinfection after collection and again after arrival in Australia.
- Source flocks must be serologically tested for APAV-3 within 21 days before the first day of collection of eggs. It is recognised that cross-reactions between APAV-3 and AOAV-1 will occur.
- Unvaccinated flocks—a random sample of sufficient size is tested to give 99% confidence of detecting the agent if there is 5% prevalence in the source flocks. Positive serology may indicate active infection of APAV-3 within the source flocks.
- Vaccinated flocks—a random sample of 100 individually identified birds must be tested with individual titres recorded for each bird sampled. The test is repeated on these same birds not less than 14 days after the collection of the last eggs for the consignment. A rise in titre may indicate an active infection of APAV-3 within the source flocks.

OR

- Using a validated PCR test, a random sample of sufficient size is tested to give 99% confidence of detecting the agent if there is 5% prevalence in the source flocks.
- This testing must be carried out within 21 days before the first day of collection of eggs, and then not less than 14 days after the collection of the last eggs for the consignment.

4.3.3 Conclusion

- APAV-3 viruses have been reported from many countries all over the world. The prevalence of APAV-3 in countries from which psittacine birds may be imported is unknown.
- APAV-3 is not an OIE listed disease agent, there are no recommendations in the OIE Code on measures for safe trade, it has not been detected in Australia, and it is not notifiable in Australia.
- There are 2 broad groups of APAV-3 viruses, one which primarily affects turkeys, and the other affecting psittacine and passerine birds.
- Infection with the psittacine APAV-3 viruses can cause significant disease and mortality in psittacine birds, particularly in small species. The *Neophema* genus (an Australian genus) are reported to have a high susceptibility to APAV-3 infections.
- There have been no reports of severe disease associated with APAV-3 in wild birds, however, many of the species that are highly susceptible are only found in the wild in Australia.
- It is unclear whether the psittacine APAV-3 viruses cause clinical disease in naturally infected poultry.

Therefore, the department concluded that further risk assessment of APAV-3 was required.

4.3.4 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of APAV-3 being present in imported psittacine birds:

- Many psittacine species are considered susceptible to infection.

- APAV-3 viruses have reportedly been isolated from captive birds in the majority of countries that monitor such animals.
- The exact prevalence APAV-3 in psittacine birds is unknown, however, based on reports, infection does not appear to be uncommon.
- Subclinical disease, particularly in psittacine birds, has been reported.

Conclusion: based on this information the likelihood of importation of APAV-3 associated with psittacine birds was estimated to be **moderate**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to APAV-3 via an infected imported psittacine bird:

- Transmission appears to occur via the faecal-oral and respiratory routes. APAV-3 can be reasonably expected to behave similarly to AOAV-1, although the lower rate of detection world-wide may indicate that APAV-3 does not spread as readily as AOAV-1.
- *Neophema* species, lovebirds, cockatiels and Amazon parrots appear to be particularly susceptible to APAV-3, many of which are native to Australia.
- Passerines appear to be susceptible, particularly small species which are in contact with affected psittacine birds.
- Turkeys are susceptible hosts. Spread to other avian species including poultry may be possible, although natural infection in chickens has never been identified.
- APAV-3 is not zoonotic.

The exposure group is considered to be captive birds and wild birds.

Conclusion: based on this information the likelihood of susceptible species in exposure groups being exposed to APAV-3 associated with psittacine birds was estimated to be **moderate**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of APAV-3 in imported psittacine birds was estimated to be **low**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible species to APAV-3 has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to APAV-3 was considered to be a **widespread outbreak**, whereby APAV-3 virus establishes in directly exposed populations (captive birds and wild birds), spreads to other populations of wild birds and becomes endemic in Australian wild birds.

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario:

- Many of the susceptible bird species are native wildbirds of Australia and commonly kept captive birds. Considering the epidemiology of APAV-3, it is unlikely that this disease would be self-limiting once introduced to susceptible populations.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of APAV-3 through Australian captive bird and wild bird populations was **moderate**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of APAV-3 were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of APAV-3.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Effects are likely to be limited to psittacine and passerine birds.
- No vaccination for APAV-3 is currently available in Australia.
- Based on these considerations, the effect of the establishment and/or spread of APAV-3 for this criterion was estimated to be of minor significance at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- There have been no reports of severe disease associated with APAV-3 in wild birds overseas, however, high mortalities may occur in infected birds of the *Neophema* genus which are native wildbirds of Australia.
- Based on these considerations, the effect of the establishment and/or spread of APAV-3 for this criterion was estimated to be of minor significance at the national level.

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- APAV-3 is not covered by the EADRA and there are no established response plans in place. It is unlikely that government led response will be undertaken. Individual owners are likely to be responsible for managing the impact of the disease on their birds.
- Based on these considerations, the effect of the establishment and/or spread of APAV-3 for this criterion was estimated to be of minor significance at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- The disease may negatively affect industries involved in breeding and selling pet/aviary psittacine birds (and related feed and equipment), and people buying birds from affected aviaries.
- Movement restrictions or other effects on domestic trade or industry are not expected.
- Based on these considerations, the effect of the establishment and/or spread of APAV-3 for this criterion was estimated to be indiscernible at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- APAV-3 is not OIE listed and it is not likely to cause any international trade effects.
- Based on these considerations, the effect of the establishment and/or spread of APAV-3 for this criterion was estimated to be indiscernible at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- Negative effects may be seen in populations of birds of the *Neophema* genus, if infected.
- Based on these considerations, the effect of the establishment and/or spread of APAV-3 for this criterion was estimated to be of minor significance at the national level.

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Effects on communities are likely to be minimal. Control measures are unlikely.
- Based on these considerations, the effect of the establishment and/or spread of APAV-3 for this criterion was estimated to be indiscernible at the national level.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **low**.

Estimation of the likely consequences

The estimate of the likelihood of establishment and/or spread for the scenario (**moderate**) was combined with the overall effect associated with the outbreak scenario (**low**) using Figure 4 to obtain an estimation of likely consequences of **low**.

4.3.5 Risk estimation

Using Figure 5, the likelihood of entry and exposure (**low**) was combined with the likely consequences of establishment and/or spread (**low**), which resulted in a risk estimation of **very low**.

Therefore, as the unrestricted risk estimate achieves Australia's ALOP, no specific risk management is considered necessary for APAV-3.

4.3.6 References

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4.4 Avian metaavulavirus 5

4.4.1 Background

Avian metaavulavirus 5 (AMAV-5), formerly known as avian paramyxovirus 5 (APMV-5), is a member of the *Metaavulavirus* genus, in the Paramyxoviridae family. The virus was first isolated in 1974 during an epizootic outbreak involving budgerigars in Japan. The disease was characterised by depression, dyspnoea, torticollis, diarrhoea and up to 100% mortality (Nerome et al. 1978). There were reports of 2 subsequent disease outbreaks, where affected budgerigars displayed clinical signs suggestive of AMAV-5 infection, and the causative virus had properties consistent with the paramyxovirus group. The first case occurred in Brisbane, Australia, in 1974 and caused an acute fatal enteritis among immature budgerigars. The isolated viral agent had the properties of a paramyxovirus, but was distinct from Newcastle disease virus, and was suggested for tentative inclusion into the paramyxovirus group (Mustaffa-Babjee, Spradbrow & Samuel 1974). The second case caused vomiting, diarrhoea and death in budgerigars in the United Kingdom in 1993. Haemagglutination inhibition and virus neutralisation tests showed that only antisera against AMAV-5 inhibited and neutralised the isolated virus (Gough et al. 1993).

More recently, a virus isolated from a subclinical wild musk lorikeet in 2014 in Victoria, Australia was suggested to represent a novel genotype of genetic subgroup within the AMAV-5 serotype (Amery-Gale et al. 2018).

AMAV-5 is not an OIE-listed disease, is not a nationally notifiable disease in Australia and is not known to be a human pathogen.

4.4.2 Technical information

Epidemiology

AMAV-5 has only been definitively isolated from budgerigars (Muzyka et al. 2014). It has different properties from the other avian paramyxoviruses in that it lacks a virion hemagglutinin and does not grow in the allantoic cavity of embryonated chicken eggs. AMAV-5 may be cultured in the amniotic cavity of embryonated chicken eggs (Mustaffa-Babjee, Spradbrow & Samuel 1974; Muzyka et al. 2014).

Of the avian paramyxoviruses, only AOAV-1 and AMAV-5 have been associated with 100% mortality, with AMAV-5 being associated with mortalities in budgerigars (AQIS 1999; Gogoi, Ganar & Kumar 2017; Samuel et al. 2010).

Hosts/susceptible species

AMAV-5 has been definitively isolated from budgerigars and is associated with up to 100% mortality in affected birds (Muzyka et al. 2014; Nerome et al. 1978). Mustaffa-Babjee (1974) observed 60-70 free living rainbow lorikeets die of acute enteritis 2 months prior to a 1974 outbreak at a bird sanctuary near Brisbane where budgerigars experienced enteritis prior to high mortalities. The causative virus was found to be consistent with what would later be identified as AMAV-5. Mustaffa-Babjee (1974) suggested that given the evidence for pathogenicity of the virus in budgerigars, its possible role in the production of enteritis in rainbow lorikeets should also be investigated.

In 2014, Basic Local Alignment Search Tool (BLAST) analysis on a virus isolated from a wild musk lorikeet in Victoria, Australia showed a gene sequence that did not match any known paramyxovirus, but had highest (77.4–77.6%) nucleotide sequence identity with strains of AMAV-5. Phylogenetic analysis suggests that this virus strain belongs to the serotype AMAV-5, and perhaps represents a novel genotype or genetic subgroup within this serotype (Amery-Gale et al. 2018).

Wild budgerigars are naturally found throughout much of mainland Australia, but are absent from the far south-west, the north of the Northern Territory, Tasmania and the majority of the east coast (Australian Museum 2018).

Experimental infection of chickens and pigeons with AMAV-5 has not caused disease (Mustaffa-Babjee, Spradbrow & Samuel 1974).

Modes of transmission

Similar to other paramyxoviruses, the primary routes of transmission are faecal-oral and respiratory (Mundt 2013).

Incubation period

The incubation period for AMAV-5 is likely to be similar to that for AOAV-1 which averages around 5–6 days (Mundt 2013).

Persistence of agent

In the absence of any specific reports on the inactivation of AMAV-5, it is assumed that its spectrum of sensitivity is similar to AOAV-1, which is most sensitive to phenols and glutaraldehyde but resistant to quaternary ammonium compounds (refer to section 4.2.2 for further information).

Distribution and prevalence

AMAV-5 has thus far been described exclusively in budgerigars (Mundt 2013), with cases reported from Australia, Japan and the United Kingdom (Gough et al. 1993).

Australian status

Literature on the Australian status of AMAV-5 is limited. AMAV-5 was implicated in an outbreak in Brisbane in 1974 (Gogoi, Ganar & Kumar 2017; Samuel et al. 2010) and a novel genotype was thought to have been detected in a single subclinical wild musk lorikeet in Victoria in 2014 (Amery-Gale et al. 2018).

Pathogenesis

Compared to AOAV-1, little is known about the clinical significance and pathogenicity of the other avian paramyxoviruses including AMAV-5 (Kumar & Samal 2012; Meulemans, Rauw & van den Berg 2015; Samuel et al. 2010).

Diagnosis

Clinical signs

AMAV-5 infection in budgerigars is associated with depression, dyspnea, vomiting, diarrhoea and torticollis before death (Gogoi, Ganar & Kumar 2017; Mundt 2013).

Pathology

Infected budgerigars show haemorrhages in the proventriculus, duodenum, jejunum and rectum. Occasionally, discoloration of the liver and splenomegaly are also observed (Mundt 2013).

Gross lesions can be minimal and may include pulmonary oedema, pale myocardium and pancreatic atrophy. Histologic lesions can include encephalitis, myocarditis and pancreatitis, associated with intranuclear and intracytoplasmic inclusion bodies (Beck et al. 2003).

Histologic lesions can include extensive loss of mucosal epithelium, intestinal wall oedema, vascular engorgement and the production of intranuclear inclusion bodies (Mustaffa-Babjee, Spradbrow & Samuel 1974). Nerome (1978) reported that the virus could be isolated from the brain, kidney, lung, spleen, liver and blood of experimentally infected budgerigars.

Testing

Diagnosis of AMAV-5 infection can be assisted by serology and confirmed by virus isolation and identification. Unlike the other avian paramyxoviruses (from the genera *Metaavulavirus*, *Orthoavulavirus* and *Paraavulavirus*), isolation is by inoculation of samples into the amniotic cavity of 9–11 day old specific pathogen free embryonated eggs (inoculation is into the allantoic cavity for the other avian paramyxoviruses). A passage via the yolk sac may also help in virus isolation. Samples from live birds include droppings, tracheal, cloacal and faecal swabs. Samples from dead birds include intestinal content, lungs, air sacs, spleen, brain, liver and heart, and should reflect lesion distribution where relevant. The inoculated eggs are incubated for no more than 7 days, and identification is then conducted by haemagglutination inhibition testing with antisera specific for AMAV-5 (Alexander 2000a; Alexander & Senne 2008; Meulemans, Rauw & van den Berg 2015; Mundt 2013; Muzyka et al. 2014).

Treatment

There are no known treatments for birds infected with AMAV-5 (Mundt 2013).

Control

A high level of biosecurity is seen as the main mechanism of control (Mundt 2013). However, good hygiene, disinfection and allowing time before restocking does not always prevent infection in subsequent flocks (Alexander 2000a; Fraser et al. 1991).

Current biosecurity measures

There are no specific biosecurity measures in place for AMAV-5. Currently, the only live psittacine birds that may enter Australia are pet birds from New Zealand. AMAV-5 has not been reported in New Zealand. There are no OIE recommendations for AMAV-5.

Conclusion

- AMAV-5 has been associated with disease events in budgerigars in Japan and the United Kingdom. There are limited reports of the disease in Australia but the prevalence is unknown.
- Experimental infection of chickens and pigeons with AMAV-5 has not caused disease.
- The prevalence of AMAV-5 in countries from which psittacine birds will be imported is unknown.

- AMAV-5 is not an OIE-listed disease agent and there are no recommendations in the OIE Code on measures for safe trade. AMAV-5 is not notifiable in Australia.
- AMAV-5 disease events in budgerigars have been associated with mortality rates of up to 100%.

As such, the department concluded that further risk assessment of AMAV-5 was required.

4.4.3 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of AMAV-5 being present in imported psittacine birds:

- Susceptibility to natural infection appears to be restricted to budgerigars.
- There are limited recorded disease events associated with AMAV-5 from Australia, Japan, and the United Kingdom, with all being restricted to budgerigars.
- Based on the limited reports of AMAV-5, prevalence appears to be low.
- The incubation period varies from days to weeks. Clinical signs may vary from nil to severe.

Conclusion: based on this information the likelihood of importation of AMAV-5 associated with psittacine birds was estimated to be **low**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to AMAV-5 via an infected psittacine bird:

- Transmission appears to be faecal-oral and respiratory. Based on few reports, AMAV-5 appears to be highly contagious in budgerigars.
- Recorded disease events confirmed to be associated with AMAV-5 are limited to budgerigars. Experimental infection of chickens and pigeons with AMAV-5 has not caused disease. The distribution of wild budgerigars in Australia is extensive, but is mainly located away from major human (and therefore captive bird) populations. It is not zoonotic.
- The exposure group is considered to be captive birds, primarily budgerigars.

Conclusion: based on this information the likelihood of susceptible species in exposure groups being exposed to AMAV-5 associated with imported psittacine birds was estimated to be **very low**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of AMAV-5 in imported psittacine birds was estimated to be **very low**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible avian or non-avian species to AMAV-5 has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to AMAV-5 was considered to be **local (limited) outbreak**, whereby AMAV-5 virus establishes in directly exposed populations of

captive birds, spreads to other populations of captive birds and becomes endemic in captive birds, but does not spread to populations of wild birds.

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario:

- Considering the epidemiology of AMAV-5, it is unlikely that this disease would be self-limiting once introduced to susceptible populations. It is, however, considered more likely to become endemic in the captive aviculture collections than wild bird populations.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of AMAV-5 through Australian captive bird populations was **low**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of AMAV-5 were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of AMAV-5.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Effects are likely to be limited to captive budgerigars, primarily captive aviary birds.
- No vaccination for AMAV-5 is currently available in Australia.
- Based on these considerations, the effect of the establishment and/or spread of AMAV-5 for this criterion was estimated to be indiscernible at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- Effects are likely to be limited to affected birds only. High mortalities are not expected, but may occur in the unlikely event of an outbreak in wild budgerigars.
- Based on these considerations, the effect of the establishment and/or spread of AMAV-5 for this criterion was estimated to be indiscernible at the national level.

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- AMAV-5 is not covered by the EADRA and there are no established response plans in place. It is unlikely that government led response will be undertaken. Individual owners are likely to burden the cost of the outbreak.

Based on these considerations, the effect of the establishment and/or spread of AMAV-5 for this criterion was estimated to be indiscernible at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- The disease may negatively affect industries involved in breeding and selling pet/aviary budgerigars (and related feed and equipment), and people buying birds from affected aviaries. Effects on other psittacine breeders/buyers would be negligible.
- Movement restrictions or other effects on domestic trade or industry are not expected.
- Based on these considerations, the effect of the establishment and/or spread of AMAV-5 for this criterion was estimated to be indiscernible at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- AMAV-5 is not OIE listed and it is not likely to cause any international trade effects.
- Based on these considerations, the effect of the establishment and/or spread of AMAV-5 for this criterion was estimated to be indiscernible at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- Negative effects may be seen in wild populations of budgerigars, if infected.
- Based on these considerations, the effect of the establishment and/or spread of AMAV-5 for this criterion was estimated to be indiscernible at the national level.

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Effects on communities are likely to be minimal. Control measures are unlikely.
- Based on these considerations, the effect of the establishment and/or spread of AMAV-5 for this criterion was estimated to be indiscernible at the national level.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **very low**.

Estimation of the likely consequences

The estimate of the likelihood of establishment and/or spread for the scenario (**low**) was combined with the overall effect associated with the outbreak scenario (**very low**) using Figure 4 to obtain an estimation of likely consequences of **negligible**.

Risk estimation

Using Figure 5, the likelihood of entry and exposure (**very low**) was combined with the likely consequences of establishment and/or spread (**negligible**), which resulted in a risk estimation of **negligible**.

Therefore, as the unrestricted risk estimate achieves Australia's ALOP, no specific risk management is considered necessary for AMAV-5.

4.4.4 References

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4.5 Internal and external parasites (excluding protozoa)

4.5.1 Background

Internal and external parasites of psittacine birds include helminths, arthropods and protozoa. These parasites may be either primary or opportunistic pathogens. Most parasites do not cause disease, and with the improvement of husbandry and nutrition of captive psittacines, parasitic infestations have become less common. Serious parasitic infestations generally indicate an underlying problem, such as ill health or poor husbandry practices (as reviewed in Doneley 2009).

Due to the ubiquitous occurrence and numerous species of avian parasites, only the most common and/or harmful psittacine parasites have been assessed in this chapter. Avian protozoal parasites have not been assessed in this chapter as they are already present in Australia and have been ruled out in the hazard identification chapter of this report. Furthermore, as most original research on parasites is dated and, in many cases inaccessible, relevant review papers have been cited instead of original publications.

Internal and external parasites of concern to avian health are not OIE listed and are not notifiable in Australia nor under official control. Some parasites have zoonotic potential, where humans are accidental hosts and infection is self-limiting (as reviewed in Evans 2011).

4.5.2 Technical information

Internal parasites

Internal parasites of concern in psittacine birds mainly affect the gastrointestinal tract and include nematodes (roundworms), cestodes (tapeworms) and trematodes (flukes). These helminths have 3 main life-cycle stages: eggs, larvae and adults. Adults infect definitive hosts (those in which sexual development occurs) whereas larval stages may be free-living or parasitise invertebrate vectors, intermediate or paratenic hosts.

Nematodes produce eggs that embryonate in utero or outside the host. The emergent larvae undergo 4 metamorphoses (moult) before they mature as adult male or female worms. Cestode eggs released from gravid segments embryonate to produce 6-hooked embryos (hexacanth oncospheres) which are ingested by intermediate hosts. The oncospheres penetrate the intermediate host's tissues and become metacestodes (encysted larvae). When ingested by definitive hosts, the larvae excyst and form adult tapeworms. Trematodes have more complex life-cycles where 'larval' stages undergo asexual amplification in snail intermediate hosts. Eggs hatch to release free-swimming miracidia which actively infect snails and multiply in sac-like sporocysts to produce numerous rediae. These stages mature to cercariae which are released from the snails and either actively infect new definitive hosts or form encysted metacercariae on aquatic vegetation which is ingested by definitive hosts.

Due to the lifecycles of helminth parasites, internal parasitism is common in captive psittacine birds that are housed in outdoor aviaries with access to the ground (Doneley 2009; Mawson 1982). Such birds are exposed to a higher re-infection rate which leads to a higher parasite load and increased severity of disease, especially under stress or poor husbandry practices (Globokar, Fischer & Pantchev 2017).

Within nematode parasites, ascarid worms are a common parasite of captive parrot collections with access to the ground (Mawson 1982). Commonly affected species include budgerigars, cockatiels, quaker parrots and princess parrots (Doneley 2016; Mawson 1982). Species of *Ascaridia* identified in psittacine birds include *Ascaridia hermaphrodita*, *A. columbae*, *A. galli*, and *A. platycercii* (Doneley 2016; Mawson 1982). These worms have a direct life cycle where ingestion of contaminated food, water and faeces causes infection. They are found in the small intestine and cause clinical signs of lethargy, poor body condition and diarrhoea, with death ensuing in severe infestations (Doneley 2016; Mawson 1982).

Capillaria (thread worm or hair worm), a genus of small nematode affecting the upper gastrointestinal tract, is found in many species of birds, including psittacines (Globokar, Fischer & Pantchev 2017). It is a pathogenic parasite and lesions described include hyperaemic tracts in the mucosa caused by worms tunnelling through the mucosa of the oral cavity, oesophagus and crop (Levine 1938). Ulceration and secondary bacterial infection may cause diphtheritic membranes (Levine 1938). *Capillaria annulata* and *C. obsignata* have been reported in parrots (as reviewed in Doneley 2009). These worms can have a direct or indirect life cycle through insect vectors. Birds may present with no clinical signs, or may have dysphagia, anorexia, weight loss and diarrhoea (Greenacre 2004).

Cestode (tapeworm) parasites occur occasionally in psittacine birds (Globokar, Fischer & Pantchev 2017), and are generally seen in wild caught birds or birds that are kept in dirt enclosures (as reviewed in Clyde & Patton 1996). Old World parrots, including African grey parrots, cockatoos, lorikeets and eclectus parrots appear to be particularly susceptible (as reviewed in Doneley 2016). Tapeworm genera reported in psittacine birds include *Raillietiaenia*, *Choanataenia*, *Gastronemia*, *Idiogenes*, and *Amoebataenia* (as reviewed in Doneley 2016). Ingestion of infected intermediate hosts including grasshoppers, beetles, ants, horseflies, earthworms, slugs, snails and crayfish, leads to infection in birds (as reviewed in Doneley 2016). Affected birds may be unthrifty, have diarrhoea or strain to defecate. They may also shed proglottids (tapeworm segments) infrequently in their droppings (as reviewed in Clyde & Patton 1996).

Digenetic liver trematode (flake) infections in psittacine birds are rare; cases are occasionally seen in imported, wild caught Old World parrots (as reviewed in Clyde & Patton 1996). Various species of liver fluke from the Dicrocoelidae family have been described in parrots (Kazacos et al. 1980; Quesenberry et al. 1986). Only severe infestations appear to affect hepatic function and cause disease (Kazacos et al. 1980; Quesenberry et al. 1986). Clinical signs include depression and anorexia (as reviewed in Greenacre 2004). Birds with severely compromised hepatic function with other co-morbidities may also die (Kazacos et al. 1980; Quesenberry et al. 1986). Ingestion of an infected snail intermediate host leads to infection in birds (Greenacre 2004).

Diagnosis and testing

Diagnosis of most internal helminth parasites of the gastrointestinal system is through a faecal float test. Most internal parasites, including those described above, pass eggs or other life stages in the host animal's faeces which can be detected through a faecal float once the pre-patent period has passed. The pre-patent period varies for each parasite species – testing the faeces prior to the end of the pre-patent period will lead to false negative results. The morphology of helminth eggs varies markedly. Diagnostic characteristics for each type of helminth egg have been described well in the literature (Greiner 1989). Faecal float tests can be easily performed

in-house and are generally sufficient for a diagnosis. Definitive identification can be made by sending whole worms for identification at a veterinary laboratory. For nematode infestations, a nematode larval culture can also be performed for definitive identification.

Treatment and control

Treatment and control of internal parasites requires treatment of affected birds as well as removing sources of re-infection.

Anthelmintics with broad spectrum activity are generally effective against roundworms. Most anthelmintics, such as ivermectin, pyrantel pamoate and fenbendazole, are effective against ascarid worms (Doneley 2016; Greenacre 2004). Ivermectin, moxidectin and levamisole have been recommended for use against capillaria worms (Doneley 2009). Resistance to anthelmintic treatment has been noted in capillaria and response to treatment should be monitored through repeat faecal float tests (Doneley 2016; Greenacre 2004). For ascarid infections, the environment of the bird should be cleaned to prevent reinfection. This can be done by steaming, flaming or desiccating as ascarids are resistant to disinfectants. For *Capillaria* species infections, access to insect vectors should be controlled in addition to environmental disinfection.

The drug of choice for treating tapeworm and fluke infections is praziquantel (Greenacre 2004). Access to intermediate hosts should be controlled to prevent re-infection.

Preventative deworming once or twice a year with a broad spectrum anthelmintic formulation containing several active ingredients is a cheap and effective way of managing internal parasitism, particularly if the infection is subclinical. However, the best control mechanism to prevent internal parasitism is through aviary design and hygiene. Psittacines housed in outdoor aviaries should be kept off the ground to prevent contact with infected faeces. Captive birds should also be prevented from co-mingling with free-ranging birds (as reviewed in Doneley 2009).

External parasites

Arthropod parasites, including mites and lice, are uncommon in psittacine birds (Gill 2001). Many mites are considered to be non-pathogenic to their host species. Of the pathogenic mites, scaly face or leg mite (*Knemidocoptes pilae*) is typically seen in budgerigars, kakarikis, and parrots (*Neophema* and *Polytelis* species) (as reviewed in Doneley 2009). Clinical signs include pathognomonic hyperkeratotic encrustations on the face, beak, cere and legs. It is thought that clinical disease is associated with poor nutrition, stress, immunosuppression and/or genetic factors (as reviewed in Doneley 2009).

Air sac mites appear to mainly infect canaries and finches, but have been reported in budgerigars and cockatiels (as reviewed in Doneley 2009). These mites live in the respiratory tract of affected birds and cause clinical signs including dyspnea, coughing and sneezing (Stephan, Kaschula & Canham 1950). It appears that chicks become infected in the nest from their parents (as reviewed in Doneley 2009). Heavy infestations can cause tracheitis, bronchitis and death by asphyxiation (Stephan, Kaschula & Canham 1950).

Other mites of lesser significance include roost mites (*Dermanyssus gallinae*), primarily a poultry parasite, which may occasionally parasitise parrots (Sparagano et al. 2014). These blood-sucking mites can cause skin irritation and anaemia, especially in small or juvenile birds (as reviewed in Doneley 2009). These mites can cause localised pruritic lesions on human skin which are usually

self-limiting (as reviewed in Evans 2011). Feather mites are obligatory permanent ectoparasites of birds; they typically inhabit the skin, inside the quills and on the surface of feathers (Dabert & Mironov 1999). Psittacine birds have their specific feather mite fauna, however, disease can occur in non-host adapted species or immunocompromised birds (Rubinstein & Lightfoot 2014). Similarly, quill mites (eg, *Syringophilus* and *Dermoglyphus* species) are non-pathogenic, but can cause horizontal barring, haemorrhage, and fracture of feather shafts with subsequent feather loss (Rubinstein & Lightfoot 2014).

Biting (chewing) lice are extremely host-specific and most birds appear to harbour lice without showing any disease (Clayton, Gregory & Price 1992). Two suborders of lice, Amblycera and Ischnocera, are known to parasitise birds and mostly inhabit the feathers of the birds they parasitise (Clayton, Adams & Bush 2008). Heavy infestations may lead to pruritus and poor feather quality (as reviewed in Doneley 2009).

Diagnosis and testing

Diagnosis of mite and louse infestations can be made easily with a thorough physical exam to identify parasites directly. In-house cytological exam under a light microscope can also be used to identify parasites provisionally from skin scrapings and tape preparations which is sufficient for a diagnosis. Definitive identification of species from a veterinary laboratory is generally not warranted.

Skin scrapings from lesions can be used to identify scaly leg mite. Air sac mites and their eggs can be visualised through a tracheal wash, and sometimes can be seen directly within the trachea with a focal intense light. Feather mites can be identified directly or through cytology on a tape preparation of affected feathers. Quill mites can be found by examining the pulp material within a developing or affected feather. Lice and their eggs can be readily seen with the naked eye – eggs can be found glued to feather shafts (as reviewed in Doneley 2009).

Treatment and control

Treating and controlling parasite infestations requires an understanding of the parasite's life cycle and properties. Treatment measures generally include use of a parasiticide and disinfecting the bird's environment to reduce re-infestation. For the aforementioned mites and lice, all except the roost mite spend their entire life cycle on the avian host. Roost mites feed on avian hosts during the night and remain secluded in the environment during the day (Sparagano et al. 2014). Treatment recommendations include use of oral ivermectin to treat scaly leg mite, air sac mite, feather mite and quill mites (as reviewed in Doneley 2009). Repeated treatment in 10–14 days is necessary for scaly legs mites or until signs resolve. Lice can be treated with topical carbaryl dusting powder or pyrethrin sprays (as reviewed in Doneley 2009; Greenacre 2004). For roost mites, both the bird and its housing/environment must be treated. Birds can be given oral ivermectin or topical carbaryl dusting powder and the environment should be treated concurrently with a residual insecticide (as reviewed in Doneley 2009).

Distribution and prevalence

Parasites are ubiquitous in nature. There is a wide geographic variation in the type and occurrence of parasites, however, the specific distribution of most avian parasites is unknown.

Australian status

A wide variety of avian parasites already exist in Australia. Details of the genus and species of avian parasites present in Australia and their prevalence and distribution is unknown. It is likely that there are some avian parasites overseas that are exotic to Australia.

Current biosecurity measures

Australia currently has no biosecurity measures in place for internal and external parasites in psittacines, including pet psittacines imported from New Zealand.

However, the current conditions for the importation of live pigeons require each pigeon to be examined by a government approved veterinarian for evidence of infectious or contagious disease and/or external parasites. During the pre-export quarantine period, each pigeon is subjected to the following treatments:

- An external parasiticide effective against ticks, lice and mites, applied twice as a powder/wash at an interval of 10 days with the first treatment being within 24 hours after the commencement of the quarantine period.
- A broad spectrum anthelmintic effective against nematodes administered twice at an interval of 21 days with the final treatment being within 3–7 days prior to the scheduled date of export.
- A broad spectrum anthelmintic effective against cestodes administered twice at an interval of 21 days, with the final treatment being within 3–7 days prior to the scheduled date of export.

There are also current biosecurity measures for internal and external parasites for other live animal species.

4.5.3 Conclusion

- Some species of internal and external parasites are present in Australia.
- There are limited studies on psittacine parasites and those that have been done infrequently identify parasites that cause clinical disease.
- In Australia there are no control measures in place for internal or external parasites of psittacines and none are nationally notifiable.
- There are no recommendations for psittacine internal or external parasites found in the OIE Code.
- Treatment of both internal and external parasites is effective, simple, cheap and safe to the live animal.

Therefore, the department concluded that further risk assessment was not necessary. Instead, general risk management measures in the form of pre-export broad-spectrum parasiticide application and anthelmintic treatment for internal and external parasites is considered appropriate for psittacine birds. If there is evidence of internal or external parasites on arrival or during post-entry quarantine, the department will require that the bird(s) be treated with a registered broad spectrum anthelmintic(s) and/or parasiticide(s).

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4.6 Infection with *Salmonella* spp.

4.6.1 Background

Salmonellosis is an important disease of both humans and animals worldwide. *Salmonella enterica* subspecies *enterica* serotype Typhi, the cause of typhoid fever, was first isolated from a human patient in 1884 and *S. enterica* subspecies *enterica* serotype Choleraesuis was first isolated from pigs in 1886.

Salmonellae are gram negative, non-spore forming, facultative anaerobic bacteria belonging to the family *Enterobacteriaceae*. The genus *Salmonella* is divided into 2 species, *S. enterica*, which cause diseases in humans, mammals and birds, and *S. bongori*, which is most frequently associated with reptiles. Several subspecies, and more than 2,500 serotypes exist. Salmonellosis can manifest in 5 ways; enteric fever, gastroenteritis, bacteraemia, extra-intestinal focal infection and a carrier state. *Salmonella* can also be classified according to their host ranges. Generalists are able to cause disease (usually acute gastroenteritis) in a wide range of human and animal hosts. Host-adapted *Salmonella* usually cause systemic infections in certain species but can also infect a limited number of other species. Host-restricted *Salmonella* are almost exclusively associated with severe systemic infections (enteric fever) in a single host species (Sanderson & Nair 2013).

While many *Salmonella* species and serotypes are present in Australia, a number of *Salmonella* serotypes that are exotic to Australia or subject to official controls could be transmitted by psittacine birds. This chapter and risk assessment focuses on the following Salmonellae that are of biosecurity concern and that have been detected in psittacine birds:

- *Salmonella enterica* subspecies *enterica* serotype Gallinarum biovar Gallinarum (*S. Gallinarum*)
- *Salmonella enterica* subspecies *enterica* serotype Gallinarum biovar Pullorum (*S. Pullorum*)
- *Salmonella enterica* subspecies *enterica* serotype Enteritidis (*S. Enteritidis*)
- *Salmonella enterica* subspecies *enterica* serotype Typhimurium (*S. Typhimurium*)
- *Salmonella enterica* subspecies *arizonae* (*S. arizonae*)

S. Pullorum and *S. Gallinarum* affect poultry and are listed in the OIE Code (OIE 2018b). The OIE also recognises that multiple antibiotic resistant *Salmonella* serotypes are of increasing concern in both public health and primary production (OIE 2018b, 2019g).

4.6.2 Technical information

Epidemiology

Salmonellae generally function as primary pathogens in psittacines, causing gastrointestinal and systemic infections. Salmonellosis in psittacine birds has been reported as a significant problem in wild-caught birds closely confined in quarantine stations and in aviaries with significant rodent infestations (Schmidt, Reavill & Phalen 2015).

Salmonella Typhimurium is the most common isolate from clinically affected psittacine birds but other salmonellae including those important to poultry (*S. arizonae*, *S. Enteritidis*, *S. Gallinarum* and *S. Pullorum*) have occasionally been identified in parrots (Deem et al. 2005; Karesh et al. 1997; Marietto-Gonçalves et al. 2010; Orós et al. 1998; Tunca et al. 2012). A distinct multi-drug resistant strain of *S. Typhimurium* that displays resistance to ampicillin, chloramphenicol,

streptomycin, sulphonamides and tetracycline (ACSSuT) is present in many countries. It is known as *S. Typhimurium* definitive type 104 R-ACSSuT and commonly abbreviated to *S. Typhimurium* DT104 (Helms et al. 2005). *S. Typhimurium* DT104 was isolated from 2 pet psittacine birds in south-eastern United States (Hudson et al. 2000), no other reports of *S. Typhimurium* DT104 in psittacine birds were identified.

Hosts/susceptible species

The potential host range for avian salmonellosis appears to be unlimited (Daoust & Prescott 2007). *S. Typhimurium* is the most common cause of salmonellosis in psittacine birds. *S. Typhimurium* has been shown to cause disease in a range of parrots including African grey parrots, Senegal parrots, Amazon parrots, cockatiels, cockatoos, conures, macaws, lorries and lorikeets (Hudson et al. 2000; Panigrahy & Gilmore 1983; Phillips & Hatkin 1978; Piccirillo et al. 2010; Shima & Osborn 1989; Vigo et al. 2009; Ward et al. 2003). Host-adapted salmonellae are known to occur in some birds such as *S. Pullorum* and *S. Gallinarum* in chickens and a host adapted serotype of *S. Typhimurium* which causes paratyphoid in pigeons (Pasmans, Boyen & Haesebrouck 2013). No host-adapted or host-restricted *Salmonella* have been identified in psittacine birds. The host adapted *S. Pullorum* and *S. Gallinarum* have been isolated from a wide range of bird and mammal species. Despite this wide host range, *S. Pullorum* and *S. Gallinarum* have been eradicated from commercial poultry flocks in Australia, Canada, the United States and Western Europe. The successful eradication demonstrates that other birds and mammals are of little importance in the epidemiology of these diseases in chickens (Snoeyenbos & Williams 1991).

Modes of transmission

Salmonellae are primarily enteric bacteria and the main mode of transmission is the faecal-oral route. Vertical transmission of some salmonellae occurs in chickens, but has not been shown to occur in psittacine birds. Possible sources of infection include:

- direct transmission from other birds, including wild birds
- contaminated feed, water and equipment
- humans
- other animals, especially reptiles and rodents.

Salmonellae can persist for long periods in the environment and contaminated environments constitutes a continuous source of reinfection for birds (Daoust & Prescott 2007; Pasmans, Boyen & Haesebrouck 2013).

Incubation period

No specific studies were identified that investigated the incubation period of salmonellosis in psittacine birds. Case reports described clinical disease or death occurring within days or weeks of probable exposure to salmonellae (Shima & Osborn 1989; Ward et al. 2003). In humans, the incubation period is typically 6–72 hours; in atypical cases illness has been documented 14 days after exposure (Healy & Bruce 2019).

Persistence of agent

Clinically infected birds shed salmonellae in faeces. No specific studies were identified that investigated the persistence of shedding in psittacine birds following clinical infection. However,

chronic infection with generalist *Salmonella* spp. is possible and could result in shedding of salmonellae in faeces over weeks or months after clinical disease appears to have resolved. Psittacine birds exposed to salmonellae may passively shed the organism without becoming infected, but this is true of all animals including humans.

Host-restricted *Salmonella* spp. can cause a carrier state in some animals and carrier animals can shed salmonellae intermittently without showing clinical signs of disease (Uzzau et al. 2000). No direct evidence of a carrier state or intermittent shedding in psittacine birds was identified, however, many authors have suggested that this is possible based on extrapolation from similar findings in poultry (Daoust & Prescott 2007; Pasmans, Boyen & Haesebrouck 2013).

In general, salmonellae are relatively resistant to environmental conditions and survive well in the presence of moisture. Salmonellae do not sporulate and are destroyed by disinfectants and inactivated by heat and sunlight (Gast 2013). Salmonellae proliferate between 5.2°C and 46.2°C (FSANZ 2017) and can survive freezing in some circumstances (Manios & Skandamis 2015). Salmonellae will grow at a broad pH range of 3.8 to 9.5 (FSANZ 2017). Salmonellae are resistant to desiccation (Margas et al. 2014) and low water activity conditions (Mattick et al. 2000). This makes them capable of prolonged survival in dried faeces, dust, feedstuffs and other organic substrates (Radostits et al. 2007).

Distribution and prevalence

Salmonellae are ubiquitous in the environment and have worldwide distribution.

The prevalence of *Salmonella* infection in clinically healthy psittacine birds has been investigated. Marietto-Gonçalves and colleagues (Marietto-Gonçalves et al. 2010) collected blood and cloacal swabs from 103 psittacine birds seized from the illegal wildlife trade; *S. Enteritidis* was isolated from 3 (2.9%) blue-fronted Amazon parrots that also tested positive serologically. Allgayer (Allgayer et al. 2008) tested cloacal swabs from 280 captive psittacine birds of 13 species using PCR. *Salmonella* DNA was detected in 13.2% of birds although none of the samples were positive when tested using standard culture techniques. Deem and colleagues (Deem et al. 2005) surveyed captive and free-ranging blue fronted Amazon parrots in Bolivia. Significantly more captive than free-ranging birds demonstrated antibodies to *Salmonella* (85.7% cf. 53.6%). Karesh and colleagues (Karesh et al. 1997) conducted a similar survey of macaws in Peru and found higher seropositivity (7/10 positive) in sub-adult birds compared to nestlings (1/15 positive). It was not clear if the microagglutination test used for the Deem et al. (2005) and Karesh et al. (1997) studies was validated for use in parrots or whether cross reaction with other salmonella antibodies was likely to occur with this test. Grimes and Arizmendi (1992) tested sera from 2,470 psittacine birds and found 1.8% to be positive for *S. Typhimurium* with higher seropositivity in African grey parrots (9.5% positive). A further study by these authors in 1995 tested sera from 3,915 birds of 8 psittacine species and found 1.4% to be positive for *S. Typhimurium* and again found higher seropositivity in African grey parrots (6.8% positive) (Grimes & Arizmendi 1995). De Souza Lopes et al. (2014) collected cloacal swabs from 182 captive psittacine birds housed in commercial and conservation establishments in Brazil. Salmonellae were isolated from 3 birds, including *S. Saintpaul*, *S. Lexington* and *S. Newport*. In a further study by these authors in 2015, cloacal swabs were collected from 167 birds housed in a wildlife rehabilitation centre. One bird tested positive for *S. Saintpaul*. Akhter et al (2010) collected cloacal, faecal and oral swabs from 15 psittacine birds housed at Dhaka zoo, Bangladesh. Nine of the birds tested positive for *Salmonella*. The isolate was identified as

S. Pullorum based on a slide agglutination test, however, this is not conclusive as cross-reaction between *S. Typhimurium* and *S. Pullorum* specific serum has been shown to occur (Sumithra et al. 2013).

The proportion of birds with positive *Salmonella* test results varied widely between studies, most likely due to the differences in testing method and the bird populations sampled. Only one study was identified that conclusively isolated *Salmonella* of biosecurity concern from apparently healthy psittacine birds. Based on the studies reviewed, the prevalence of faecal shedding of viable *Salmonellae* of biosecurity concern in healthy psittacine birds is likely to be very low.

A number of case reports of clinical salmonellosis in psittacine birds were also identified. Oros et al. (1998) reported a case of *S. arizonae* in a sulphur crested cockatoo that died without showing clinical signs of disease 2 days after the introduction of a group of iguanas to the pet shop in the Canary Islands where the bird was kept. Tunca et al. (2012) investigated 3 outbreaks of salmonellosis in budgerigar flocks in Turkey. Three separate flocks of 245, 450 and 600 birds, experienced high morbidity (45–90%) and mortality (35–80%) in adult and young birds. *Salmonella* was isolated from tissue samples and identified in several organs using immunohistochemistry. The authors concluded that the causative agent was *S. Gallinarum*, however, the isolate was reported to be motile (a trait not generally associated with *S. Gallinarum*) so it is unclear if the causative agent was accurately identified. Piccirillo et al. (2010) reported on the clinical presentation and pathology of *S. Typhimurium* in 2 Moluccan cockatoos. One bird died suddenly without showing clinical signs and the other died 20 days later after showing clinical signs of depression, anxiety and diarrhoea. Shima and Osbourne (1989) described an outbreak of salmonellosis caused by *S. Typhimurium* in a collection of lorries and lorikeets in the San Diego Zoo. The outbreak occurred following the introduction of birds imported from Australia, although it was not clear if the imported birds were the source of the infection. Unusually warm weather and poor husbandry contributed to the outbreak. Vigo et al. (2009) reported cases of *S. Typhimurium* in 2 blue and gold macaw chicks that died after showing signs of depression, poor appetite, delayed crop emptying, laboured breathing and diarrhoea. The source of the infection was not identified. Ward et al. (2003) described an outbreak of salmonellosis in a closed flock of 45 lorries and lorikeets that had previously been tested for *Salmonella* with negative results. Ten birds died in the outbreak which occurred shortly after a group of birds were observed to attack and kill a snake. All cases were acute or per-acute and *S. Typhimurium* was isolated from samples collected post-mortem.

Australian status

A number of *Salmonella* spp. of importance to poultry production are nationally notifiable and subject to control in Australian commercial poultry including *S. Enteritidis*, *S. Gallinarum* and *S. Pullorum* (Department of Agriculture 2017).

Salmonella Typhimurium, the most common *Salmonella* serotype isolated from parrots, is present in Australia. The Australian Salmonella Reference Centre (ASRC) (Australian Salmonella Reference Centre 2017) annual report showed that *S. Typhimurium* was the most common *Salmonella* isolated from humans and a common isolate from non-human sources. The 2016 ASRC report listed 1,174 confirmations of *S. Typhimurium* from a range of sources including human infections, human food, animal food, alpaca, horses, sheep, environment samples, poultry and other birds (Australian Salmonella Reference Centre 2017). In addition to this, a study by

Iveson et al. (2014) found that *S. Typhimurium* was also the most common *Salmonella* isolated from wildlife in areas of Western Australia with dense human populations.

Salmonella Typhimurium DT104 has not been reported in Australian livestock nor products derived from Australian livestock (Barlow & Gobius 2008). In addition, there is a low incidence of human *S. Typhimurium* DT104 infection in Australia. When present, it is often associated with imported food or overseas travel (Fisher et al. 2001; Helms et al. 2005). Salmonellosis due to *S. Typhimurium* DT104 is not a nationally notifiable animal disease in Australia (Department of Agriculture 2017). However, it is a serious zoonosis (OIE 2019g) and salmonellosis is a nationally notifiable disease in humans (Department of Health 2018).

Pathogenesis

The pathogenesis of salmonellosis in psittacine birds is less well described than in poultry. Salmonellosis in psittacine birds is most commonly caused by the generalist, *S. Typhimurium* and usually occurs when the birds are subject to stressful conditions such as introduction of new birds, change of diet or housing, transport, poor hygiene and management, overcrowding, and concurrent disease (Piccirillo et al. 2010). However, disease can occur in healthy birds exposed to sufficient numbers of virulent *Salmonella*. Variable susceptibility to infection has been reported in different strains of mice and breeds of chicken, but no information was available about susceptibility of different species of psittacine birds (Daoust & Prescott 2007).

Diagnosis

Diagnosis of clinical disease in psittacine birds is usually based on clinical presentation and culture of faeces or tissues collected during post-mortem examination (Pasmans, Boyen & Haesebrouck 2013).

Clinical signs

Salmonellosis in psittacine birds can present as gastroenteritis, systemic infection or acute death without previous clinical signs. Clinical signs in birds with gastroenteritis include diarrhoea, poor appetite, poor body condition, crop stasis, lethargy, laboured breathing and ruffled feathers. Birds with systemic infections can show these clinical signs as well as subcutaneous granulomas, conjunctivitis, arthritis and neurological signs (Daoust & Prescott 2007; Pasmans, Boyen & Haesebrouck 2013).

Pathology

On post-mortem examination findings are variable; there can be few gross lesions in birds that die acutely and more extensive lesions following chronic cases. Lesions commonly found during post-mortem examination include enteritis and lesions consistent with systemic bacterial infection. These can include an enlarged congested liver with or without randomly distributed necrotic or granulomatous foci, an enlarged friable spleen, congested lungs and kidneys, enteritis, granulomatous lesions in other organs including the brain, muscle and subcutis, encephalitis, fibrinopurulent inflammation of the pericardium, peritoneum and air sacs, and arthritis (Daoust & Prescott 2007).

Testing

Living birds can be tested serologically or cloacal swabs can be collected for identification of *Salmonella* by culture or molecular methods. Samples of liver and other organs collected at post-mortem can also be tested for *Salmonella* by these methods (OIE 2019g). Some researchers have

used immunohistochemistry to identify *Salmonella* in tissue sections collected during post-mortem examinations (Orós et al. 1998; Tunca et al. 2012). Serological testing is widely used in *Salmonella* surveillance programs in commercial poultry. The OIE manual recommends *Salmonella* isolation by culture as the preferred test for diagnosis in individual animals. Numerous culture methods are described in the OIE manual (OIE 2019g).

Treatment

Recommended treatment for clinically affected psittacine birds includes isolation of affected birds, antimicrobial treatment of individual birds based on culture and sensitivity results, and supportive care (Daoust & Prescott 2007).

Control

While a number of *Salmonella* serotypes are subject to control in commercial poultry production systems, including official *Salmonella* accreditation programs and autologous vaccination of birds (specific to challenge strains), there are no official control programs in place for psittacine birds. There are no *Salmonella* vaccines available for psittacine birds. A number of authors have described good husbandry practices that can assist in control and prevention of salmonellosis in captive psittacine birds, including hygienic feeding practices and quarantine of newly acquired birds (Daoust & Prescott 2007).

Current biosecurity measures

Current biosecurity measures are in place for relevant salmonellae in a number of avian commodities, including fertile poultry (chicken, duck and turkey) eggs, pigeons and chicken meat.

Biosecurity measures for fertile poultry eggs require the source flock to be free from clinical signs of salmonellosis for 90 days prior to collection of the eggs. The source flock must also be part of a government monitoring program for *S. Enteritidis*, *S. Gallinarum*, *S. Pullorum* and *S. arizonae* or be tested for these salmonellae prior to egg collection. After arrival in Australia, the fertile eggs must be incubated and hatched in a high biosecurity government post-entry quarantine facility or Approved Arrangement site. In post-entry quarantine, hatchery waste, pipped embryos and birds that die in the first 10 days after hatching are sampled and tested for *Salmonella*. In addition to this, specific pathogen free (SPF) sentinel chickens are raised with the imported birds and tested serologically for *Salmonella* at 6 weeks of age.

Biosecurity measures for pigeons require the birds to be held in pre-export quarantine for at least 55 days prior to import and be tested for *S. Enteritidis*, *S. Gallinarum*, *S. Pullorum* and *S. arizonae* by culture of cloacal swabs at least 28 days after the commencement of quarantine. In addition, the birds must undergo post-entry quarantine at a high biosecurity government post-entry quarantine facility where they are housed with SPF sentinel chickens. The SPF chickens are inoculated with faeces from the imported pigeons and tested serologically for *Salmonella* 21 days after commencement of post-entry quarantine.

Chicken meat must originate from a zone or country free from *S. Enteritidis*, *S. Gallinarum*, *S. Pullorum* and *S. Typhimurium* DT104 or be treated (heated to core temperature of 70°C for 2.5 minutes) to address the biosecurity risk.

4.6.3 Conclusion

- There are over 2,500 serotypes of *Salmonella*.
- *Salmonella* spp. are ubiquitous in the environment and have worldwide distribution including in Australia.
- *S. Pullorum* and *S. Gallinarum* are OIE-listed and *S. Typhimurium* DT104 is a serious zoonosis recognised by the OIE.
- *S. Pullorum*, *S. Gallinarum*, *S. Enteritidis* are nationally notifiable in Australia and subject to control.
- Salmonellosis in psittacine birds caused by any other *Salmonella* is not notifiable in Australia.
- Current biosecurity measures are in place for other avian species and commodities for *S. Pullorum*, *S. Gallinarum*, *S. Enteritidis*, *S. arizonae* and *S. Typhimurium* DT104.
- Most documented clinical cases of salmonellosis in psittacine birds have presented as acute and often fatal gastroenteritis.
- There is no evidence to show that a *Salmonella* latent carrier state exists in psittacine birds.
- The most common cause of salmonellosis in psittacine birds is the generalist, *S. Typhimurium*, one of the most common *Salmonella* serotypes isolated from human and non-human sources in Australia.
- Other salmonellae (*S. arizonae*, *S. Enteritidis*, *S. Gallinarum*, *S. Pullorum* and *S. Typhimurium* DT104) have occasionally been identified in psittacine birds.
- Non-poultry birds and mammals are of little importance in the epidemiology of *S. Pullorum* and *S. Gallinarum* in chickens.

Therefore, the department concluded that further risk assessment for specific *Salmonella* of biosecurity concern (*S. Pullorum*, *S. Gallinarum*, *S. Enteritidis*, *S. arizonae* and *S. Typhimurium* DT104) was required.

4.6.4 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of a *Salmonella* of biosecurity concern being present in imported psittacine birds:

- All psittacine species are considered susceptible to infection.
- Salmonellae are ubiquitous in the environment and have worldwide distribution.
- Only 1 study was identified that definitively isolated *Salmonella* of biosecurity concern from apparently healthy psittacine birds. The prevalence of faecal shedding of viable salmonellae of biosecurity concern in healthy psittacine birds is likely to be extremely low.
- The incubation period may vary from days to weeks. Clinical signs may vary depending on the body system affected.

Conclusion: based on this information the likelihood of importation of a *Salmonella* of biosecurity concern associated with psittacine birds was estimated to be **very low**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to *Salmonella* of biosecurity concern via an infected imported psittacine bird:

- Transmission is primarily via faecal-oral route and *Salmonella* are easily transmitted via direct contact and contaminated food, water and equipment.
- The potential host range for salmonellosis appears to be unlimited - both humans and other animals are susceptible.
- *Salmonella* survive well in the environment.

Conclusion: based on this information the likelihood of susceptible species in all exposure groups being exposed to *Salmonella* of biosecurity concern associated with psittacine birds was estimated to be **moderate**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of a *Salmonella* of biosecurity concern in imported psittacine birds was estimated to be **very low**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of a susceptible avian or non-avian species to a *Salmonella* of biosecurity concern has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to a *Salmonella* of biosecurity concern was considered to be a **local (limited) outbreak**, where by the *Salmonella* of biosecurity concern establishes in a directly exposed population only (captive birds, wild birds, poultry and/or non-avian species), is identified and is eradicated (or is self-limiting in wild bird populations).

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario (in addition to those outlined in section 2.3.4 and relevant entry and exposure factors outlined above):

- *Salmonella* of biosecurity concern are a major concern to poultry production industries.
- Infected birds could potentially spread *Salmonella* of biosecurity concern between captive bird populations or contaminate feeding areas for wild birds. However, infection with salmonella in birds usually occurs when the birds are subject to stressful conditions or concurrent disease. Exposed but otherwise healthy animals are unlikely to become infected and further spread the disease.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of a *Salmonella* of biosecurity concern in captive birds, wild birds, poultry and/or non-avian species is **low**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of salmonellosis were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of *Salmonella* of biosecurity concern.

*Direct effects**The effect on the life or health (including production effects) of susceptible animals*

- Effects are likely to be limited to a small number of infected animals.
- Many poultry producers vaccinate for this disease via inactivated autologous vaccines (specific to challenge strains).
- Based on these considerations, the effect of the establishment and/or spread of *Salmonella* of biosecurity concern for this criterion was estimated to be indiscernible at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- High mortalities in wildlife are not expected, but cannot be completely discounted.
- Based on these considerations, the effect of the establishment and/or spread of *Salmonella* of biosecurity concern for this criterion was estimated to be indiscernible at the national level.

*Indirect effects**The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs*

- *Salmonella* of biosecurity concern are not covered by the EADRA. Individual owners would have to bear the costs of any strategies or programs they implement.
- Based on these considerations, the effect of the establishment and/or spread of *Salmonella* of biosecurity concern for this criterion was estimated to be indiscernible at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- Any effects are likely to be limited to a very local level to individual owners. Effects on domestic trade or industry are unlikely.
- Based on these considerations, the effect of the establishment and/or spread of *Salmonella* of biosecurity concern for this criterion was estimated to be indiscernible at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- A limited outbreak of a *Salmonella* of biosecurity concern is not likely to cause any international trade effects, other than for affected premises. Export requirements are limited to requirements for individual premises of origin.
- Based on these considerations, the effect of the establishment and/or spread of *Salmonella* of biosecurity concern for this criterion was estimated to be indiscernible at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- It is not likely that there will be any negative effects on the environment.
- Based on these considerations, the effect of the establishment and/or spread of *Salmonella* of biosecurity concern for this criterion was estimated to be indiscernible at the national level.

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Effects on communities are likely to be minimal. Control measures are unlikely.
- Based on these considerations, the effect of the establishment and/or spread of *Salmonella* of biosecurity concern for this criterion was estimated to be indiscernible at the national level.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **very low**.

Estimation of the likely consequences

The estimate of the likelihood of establishment and/or spread for the scenario (**low**) was combined with the overall effect associated with the outbreak scenario (**very low**) using Figure 4 to obtain an estimation of likely consequences of **negligible**.

4.6.5 Risk estimation

Using Figure 5, the likelihood of entry and exposure (**very low**) was combined with the likely consequences of establishment and/or spread (**negligible**), which resulted in a risk estimation of **negligible**.

Therefore, as the unrestricted risk estimate achieves Australia's ALOP, no specific risk management is considered necessary for *Salmonella* spp.

4.6.6 References

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4.7 Parrot bornavirus

4.7.1 Background

Proventricular dilatation disease (PDD) is a fatal disease mainly affecting captive psittacine birds. It is considered to be one of the greatest threats to the avicultural industry and to endangered psittacine species, such as the last significant population of Spix's macaw (WHA 2013a). PDD was first reported in the 1970s in imported macaws in Europe and North America (as reviewed in Hoppes, Tizard & Shivaprasad 2013). Various alternative names exist in literature, including macaw wasting disease, macaw fading syndrome, myenteric ganglioneuritis, infiltrative splanchnic neuropathy and neuropathic gastric dilation.

A viral aetiology had been long suspected and in 2008, 2 research teams independently reported the isolation of a novel bornavirus from PDD affected birds, provisionally named avian bornavirus (ABV), with isolates assigned genetic subgroups/genotypes (Kistler et al, 2008; Honkavuori et al, 2008). The virus is a single-stranded, negative-sense RNA virus belonging to the family Bornaviridae. After the taxonomic reorganisation of the Bornaviridae family in 2018 ABV isolates from psittacines were renamed parrot bornavirus (PaBV) and classified into 2 species, named *Psittaciform 1 orthobornavirus* and *Psittaciform 2 orthobornavirus*. To date, a total of 8 genetically variant PaBV viruses have been identified in psittacine birds; PaBV-1/2/3/4 and 7 are grouped within *Psittaciform 1 orthobornavirus* and PaBV-5 is grouped within *Psittaciform 2 orthobornavirus*. The more recently discovered PaBVs (PaBV-6 and PaBV-8) currently remain unclassified (ICTV, 2015).

PDD has been documented in over 80 species of parrots. Similar lesions have been described in other species of birds including passerines, falconiformes, piciformes, and anseriformes, resulting from infection with other ABV isolates (Daoust et al. 1991; Delnatte et al. 2011; Perpiñán et al. 2007; Weissenböck et al. 2009). This chapter will focus on PDD infection caused by strains of PaBV.

PDD is characterised by extensive dilatation of the proventriculus, caused by inflammatory damage to the enteric nervous system of the upper and middle gastrointestinal segments (Berhane et al. 2001). Clinically, birds exhibit gastrointestinal signs or central nervous system dysfunction, or a combination of both (Berhane et al. 2001). PDD is a progressive and terminal disease, eventually leading to the death of the animal due to emaciation.

PaBV is not an OIE listed disease agent, is not notifiable in Australia nor under official control, and is not considered to be zoonotic.

4.7.2 Technical information

Epidemiology

The epidemiology of PaBV in the development of PDD in parrots appears to be complex, with many knowledge gaps yet to be filled. PaBV infection can cause PDD or be limited to subclinical infection with no evidence of disease. The presence of PaBV in healthy, disease-free birds is well documented in the scientific literature (De Kloet & Dorrestein 2009; Lierz et al. 2009; Villanueva et al. 2010). Subclinically infected birds may shed the virus for years and shedding may occur continuously, intermittently or rarely (Payne et al. 2011). It is highly likely that a combination of host and viral factors contribute to the development of PDD, especially considering the broad host range and the numerous genetic variants of the virus. Furthermore, studies have

demonstrated differences in the pathogenicity of different isolates; experimental infection of 2 groups of cockatiels with PaBV-2 and PaBV-4 showed higher morbidity associated with PaBV-2 infection (Piepenbring et al. 2016).

Mixed infection with 2 different parrot bornavirus genotypes has been reported in naturally infected parrots with PDD (Nedorost et al. 2012), suggesting that some birds may require simultaneous infection with more than one genotype to develop PDD. In another study, birds naturally infected with PaBV-4 (and subclinical for PDD) were inoculated experimentally with a second dose of PaBV-4. These birds developed unusually severe clinical lesions upon second inoculation (Payne et al. 2011). The authors hypothesised that the unusual severity of disease may be due to the long period of time taken to develop PDD, or the birds may have mounted a type IV hypersensitivity reaction following the second exposure.

Hosts/susceptible species

PaBVs show marked genetic diversity and are capable of crossing species barriers within the order Psittaciformes, with infection being reported in over 80 different species of parrots (as reviewed in WHA 2013a). Wildlife Health Australia has determined it is highly likely that many native Australian parrot species are susceptible to infection (WHA 2013a).

Modes of transmission

Transmission of PaBV is poorly understood and the exact route of infection remains unknown. Despite the fact that PDD may spread to in-contact birds and occur as an outbreak, the evidence to support a horizontal route of transmission is limited. Horizontal transmission to in-contact birds in both natural and experimental conditions appears to be inefficient in adult immunocompetent birds (Rubbenstroth et al. 2014). More recently, a study investigating the different routes of infection has demonstrated that oral and nasal inoculation, which were the presumed routes of natural infection, do not cause infection in cockatiels (Heckmann et al. 2016). Invasive parenteral routes including intravenous, intra-cerebral and intramuscular routes have been shown to produce persistent infection (Gray et al. 2010; Piepenbring et al. 2012). Furthermore, vertical spread of the virus has been hypothesised as a possible pathway, as viral RNA has been detected in the eggs of some infected parrots (Monaco et al. 2012), however, active infection of the embryos has not been confirmed.

Incubation period

The incubation period is highly variable; some infected birds may not develop PDD at all, and in birds that develop disease, the incubation period may range from a few weeks to many years (as reviewed in WHA 2013a).

Persistence of agent

As PaBV is an enveloped virus, it is assumed to be susceptible to destruction by commonly employed disinfectants including chlorhexidine, phenolics, quaternary ammonium products and bleach (as reviewed in Hoppes, Tizard & Shivaprasad 2013).

Distribution and prevalence

Prevalence of PaBV infection in captive populations is highly variable and may be masked by subclinical infections. PaBV has been reported in Africa, Austria, Australia, Canada, Denmark, Germany, Hungary, Italy, Japan, Spain, Switzerland, United Kingdom and the United States

(Encinas-Nagel et al. 2014; Heffels-Redmann et al. 2011; Weissenböck et al. 2009). Based on these reports, it is likely that the virus is present worldwide due to global trade of parrots.

A survey study undertaken in 5 European countries found certain genera of parrots to have higher infections rates including *Amazona*, *Ara*, *Cacatua*, *Eclectus*, *Poicephalus* and *Psittacus*. Whether these genera are more susceptible to infection remains to be proven (Heffels-Redmann et al. 2011).

Furthermore, in the experience of some investigators it appears that PaBV varies considerably amongst flocks and with eradication efforts across aviaries in Europe, prevalence may be decreasing (Dennis Rubbenstroth [Institut für Virologie Universitätsklinikum Freiburg] 2017, pers. comm., 4 April).

Australian status

In Australia, PDD was first diagnosed in an imported captive green-winged macaw shortly after its release from quarantine in 1993. This case was prior to the identification of PaBV as the causative agent of the disease, and the diagnosis was based on the finding of lympho-plasmacytic ganglioneuritis in the gizzard and proventriculus, considered pathognomonic for PDD (Sullivan et al. 1997). In 2007, another 4 cases of PDD were diagnosed in south-east Queensland (Doneley, Miller & Fanning 2007). Of the 8 genetically variant PaBVs identified, only PaBV-2 and PaBV-4 are known to have been detected in Australia (Weissenböck et al. 2009). The presence of other PaBV variants in Australia is unknown. There are no reports of PDD or PaBV in Australian wild birds (WHA 2013a). In the experience of avian veterinarians in Australia, only a handful of other cases have been diagnosed in Australia and, as such, prevalence is estimated to be extremely low (David Phalen [The University of Sydney] 2016, pers. comm., 18 September).

Pathogenesis

Many aspects of the pathogenesis of PDD remain unknown. As mentioned before, the routes of infection and transmission are unknown and the sequence of disease after experimental inoculation of PaBV-2 in cockatiels has only recently been studied. It appears that after intramuscular inoculation, the virus spreads centripetally to the spinal cord then invades the brain and spinal segments (de Araujo et al. 2017). After reaching the central nervous system, the virus spreads centrifugally to the ganglia in the gastrointestinal system, adrenal gland, heart and kidneys (de Araujo et al. 2017). In clinically affected birds, inflammatory damage occurs in the ganglia and the myenteric plexus of the gastrointestinal tract, brain, spinal cord and major nerves (Berhane et al. 2001). Due to the resulting inflammation of the myenteric ganglia, a functional loss of gastrointestinal organs occurs (Berhane et al. 2001). At late points of infection, the virus is detectable in the smooth muscle and/or scattered epithelial cells of tissues such as the crop, intestines, proventriculus, kidneys, skin, and blood vessels (Berhane et al. 2001).

De Araujo and colleagues (2017) have hypothesised that PaBV reaches the central nervous system through retrograde axonal transport and migrates back to the periphery via ante-retrograde axonal transport, similar to another neurotropic negative sense single-stranded RNA virus, the rabies virus.

Diagnosis

Clinical signs

The majority of clinical signs in PDD affected birds result from gastrointestinal dysfunction. These include emaciation, intermittent regurgitation, passing of partially digested or completely undigested feed, and eventually death from starvation (as reviewed in Phalen 2014). Inflammatory damage to the central nervous system causes some birds to develop neurological signs which are typically slow to develop and are progressive. These include changes in mentation, ataxia, progressive weakness turning into paralysis, and rarely seizures. Blindness, although rare, can occur as the result of ocular or neurologic disease (as reviewed in Phalen 2014). Diagnosis of PaBV infection should not be made on evidence of PDD alone.

Pathology

Histopathological changes associated with PDD have been well described in the literature. PDD affected birds usually develop pathognomonic lymphoplasmacytic inflammatory lesions in the peripheral or central nervous system, often including nerves of the gastrointestinal tract, brain and spinal cord (Berhane et al. 2001).

Testing

Diagnosis in live birds is problematic due to the remarkable genetic variability of PaBV and consequent challenge in developing reliable diagnostic tests, in addition to the potential for subclinical infection to confuse diagnosis. Currently, PCR tests for particular PaBV viruses are commercially available in limited laboratories around the world. These include sensitive qPCR for detecting specific PaBVs or conventional PCRs called Mcon, Ncon and Ccon which employ degenerate primers for detecting a broad range of bornaviruses (Dennis Rubbenstroth [Institut für Virologie Universitätsklinikum Freiburg] 2017, pers. comm., 7 April). In the live bird, the urofaeces is the best sample for viral detection as the greatest amount of virus is shed in the urine (Heatley & Villalobos 2012). Despite this, viral shedding in the urine is intermittent and thus requires pooling of multiple samples collected from a single bird over several days (Guo et al. 2014). Samples from multiple birds can be pooled if detecting the presence of the virus in a flock or an aviary. Blood and oral swabs can also be used for PCR detection but are less sensitive (Guo et al. 2014). PCR has also been performed on feather calami, however, the reliability of positive results is questionable due to possible environmental contamination (Guo et al. 2014). Necropsy samples can be taken from most major organs as the virus can be detected in a wide range of tissues, with the highest prevalence in the cerebrum, followed in order by the cerebellum, optic nerves, spinal cord, heart, liver, proventriculus and kidney (Guo et al. 2014).

Serological antibody detection can be performed through ELISA, indirect immunofluorescence or western blotting (Guo et al. 2014). Designing an assay that is both sensitive and specific is a major challenge. Zimmerman and colleagues (2014) found that there is cross reactivity between sera from PaBV infected birds and sera from birds infected with related bornaviruses, and that assay sensitivity may vary considerably amongst different viral genotypes. Therefore, it is recommended that diagnostic laboratories design their serological assays based on the genotype that is expected in the birds being tested (Dennis Rubbenstroth [Institut für Virologie Universitätsklinikum Freiburg] 2017, pers. comm., 7 April).

The confirmatory test for PDD is histopathology. In the live patient, biopsies of the serosal surface of the proventriculus and/or ventriculus are ideal as these sites are most commonly

affected by PDD (as reviewed in Phalen 2014). However, these procedures are technically challenging and increase patient morbidity and, as such, crop biopsies are preferred from a practical standpoint. The crop biopsy should include a major blood vessel because this increases the chance of obtaining nerve sections (as reviewed in Phalen 2014). The sensitivity of crop biopsies for detecting PDD is controversial as the reported prevalence of ganglioneuritis in crops ranges from 22 to 76% (as reviewed in Gancz et al. 2009).

The virus can be isolated from brain tissue and successfully grown in multiple cell lines, including chicken, quail and duck embryo fibroblast cells lines. Since the virus is non-cytopathic, confirmation of infection in cell lines is by PCR, western blotting or immunofluorescence assays (Guo et al. 2014).

In advanced cases in live birds, survey radiographs may demonstrate dilation of the proventriculus and ventriculus, with dilation of the intestines occurring less frequently. Contrast studies using repeated radiographs or fluoroscopy can reveal changes in gastric motility. However, imaging alone is not sufficient to guarantee a diagnosis of PDD as other diseases have a similar clinical appearance, for example, zinc and lead intoxication, gastrointestinal obstructions and bacterial/fungal infections of the upper gastrointestinal tract.

The current recommendation for molecular diagnosis of PaBV infection in live birds is a combination of PCR and serological antibody testing. However, a small proportion of birds which neither shed the virus nor seroconvert may still be missed and thus repeated testing is necessary (Dennis Rubbenstroth [Institut für Virologie Universitätsklinikum Freiburg] 2017, pers. Comm., 7 April).

Treatment

There is no current treatment that can cure PDD. Treatment strategies are often prolonged and are aimed at reducing the clinical signs of PDD. This is most effective during the earlier stages of disease (as reviewed in Hoppes, Tizard & Shivaprasad 2013).

General supportive care is a big part of treatment for PDD affected birds. Due to impaired gastrointestinal motility, birds often develop secondary bacterial and fungal gastrointestinal infections which need to be managed. Furthermore, in cases of reduced intestinal motility or stasis, administration of metoclopramide (0.5 mg/kg every 12 hours by mouth or intramuscularly) is beneficial. Birds with PDD often become anaemic and hypoproteinaemic, and supplementation of vitamins, especially B complex vitamins, is helpful (Gancz, Clubb & Shivaprasad 2010).

Runge and colleagues (2017) report the development of an experimental vaccine using Newcastle disease virus and modified vaccinia virus Ankara vectors expressing PaBV-4 antigens. Subsequent challenge with a heterologous PaBV-2 infection in vaccinated cockatiels and control non-vaccinated cockatiels was successful; the vaccine appeared to offer immunity to the vaccinated birds which did not develop microscopic lesions of PDD that were seen in the non-vaccinated control cockatiels (Runge et al. 2017).

Control

Many overseas aviaries infected with PDD have been attempting to eradicate the virus from their flocks through repeated testing of all individuals (with PCR and antibody testing) and removal or isolation of all birds testing positive. Such rigorous efforts have been successful in

completely eradicating the virus in some aviaries (Dennis Rubbenstroth [Institut für Virologie Universitätsklinikum Freiburg] 2017. pers. Comm., 7 April).

Current biosecurity measures

Currently, there are no biosecurity measures in place for PaBV.

4.7.3 Conclusion

- PaBV-2 and PaBV-4 are the only genotypes identified in Australia. All other genotypes are considered to be exotic to Australia.
- The prevalence of PaBV infection in Australia is considered to be extremely low compared to European countries.
- Subclinically infected birds may be imported and may serve as a source of introduction of exotic PaBV genotypes.
- PDD is considered to be one of the greatest threats to the avicultural industry and to endangered psittacine species.
- PDD is not an OIE-listed disease and there are no recommendations in the OIE Code on measures for safe trade.
- PaBV is not notifiable in Australia.

Therefore, the department concluded that further risk assessment of exotic strains of PaBV was required.

4.7.4 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of exotic strains of PaBV capable of causing PDD being present in imported psittacine birds:

- Over 80 species of psittacine birds are considered susceptible to infection and certain genera appear to have higher infection rates (e.g. *Ara*, *Amazona*, *Poicephalus*, *Psittacus*, *Eclectus*, *Cacatua*).
- Based on reported cases, PaBV is widely distributed throughout the world.
- The incubation period varies from weeks to years. Clinical signs may vary from nil to severe. Birds may be carriers and shed intermittently and this complicates diagnosis.

Conclusion: based on this information the likelihood of importation of exotic strains of PaBV associated with psittacine birds was estimated to be **moderate**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to exotic strains of PaBV via an infected imported psittacine bird:

- Transmission is poorly understood and may involve horizontal and vertical routes. PaBV does not appear to be highly contagious.
- Susceptibility to infection with PaBV appears to be limited to psittacines. The virus is not zoonotic.
- PaBV is susceptible to inactivation using commonly used disinfectants.

- Only psittacine birds are susceptible to infection. The exposure group would include captive and wild psittacine birds.

Conclusion: based on this information the likelihood of susceptible species in exposure groups being exposed to exotic strains of PaBV associated with psittacine birds was estimated to be **low**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of exotic strains of PaBV in imported psittacine birds was estimated to be **low**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible species to exotic strains of PaBV has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to exotic strains of PaBV was considered to be a **widespread outbreak**, whereby PaBV establishes in directly exposed populations (captive birds and wild birds), spreads to other populations of captive and wild birds and becomes endemic in Australian captive and wild birds.

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario:

- Birds can have subclinical infection with PaBV and be a source of infection to susceptible birds.
- PaBV is limited to psittacines and not reported to affect other avian species.
- Considering the epidemiology of PaBV, it is unlikely that this disease would be self-limiting once introduced to susceptible populations.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of exotic strains of PaBV through Australian captive bird and wild bird populations was **moderate**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of exotic strains of PaBV were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of exotic strains of PaBV.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Effects are likely to be limited to psittacine birds; primarily captive aviary birds. Co-infection with endemic and exotic strains may increase the risk of developing PDD.
- No vaccination for PaBV is currently available in Australia.
- Based on these considerations, the effect of the establishment and/or spread of PaBV for this criterion was estimated to be of minor significance at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- High mortalities in wild birds are not expected, but cannot be completely discounted. Many Australian parrots are highly likely to be susceptible to PaBV so an outbreak in wild birds may have significant impacts.
- Based on these considerations, the effect of the establishment and/or spread of PaBV for this criterion was estimated to be of minor significance at the national level.

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- PaBV is not covered by EADRA. Individual owners would have to bear the costs of any strategies or programs they implement.
- Based on these considerations, the effect of the establishment and/or spread of PaBV for this criterion was estimated to be of minor significance at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- The disease may negatively affect industries involved in breeding and selling pet/aviary psittacine birds (and related feed and equipment), and people buying birds from affected aviaries.
- Movement restrictions or other effects on domestic trade or industry are not expected.
- Based on these considerations, the effect of the establishment and/or spread of PaBV for this criterion was estimated to be of minor significance at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- PaBV is not OIE listed and it is not likely to cause any international trade effects.
- Based on these considerations, the effect of the establishment and/or spread of PaBV for this criterion was estimated to be indiscernible at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- If endangered species of parrots become affected, this may have a significant impact on the species' conservation status. Many Australian parrots are susceptible to PaBV and an outbreak may impact biodiversity in the affected areas.
- Based on these considerations, the effect of the establishment and/or spread of PaBV for this criterion was estimated to be significant at the national level.

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Effects on communities are likely to be minimal. Control measures are unlikely.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **moderate**.

Estimation of the likely consequences

The estimate of the overall effect associated with the outbreak scenario (**moderate**) was combined with the likelihood of establishment and/or spread for the scenario (**moderate**) using Figure 4 to obtain an estimation of likely consequences of **moderate**.

4.7.5 Risk estimation

Using Figure 5, the likelihood of entry and exposure (**low**) was combined with the likely consequences of establishment and/or spread (**moderate**), which resulted in a risk estimation of **low**.

Therefore, as the unrestricted risk estimate does not achieve Australia's ALOP, specific risk management is considered necessary for PaBV.

Chapter 4.11 proposes a combination of risk management measures to reduce the above likelihood of entry and exposure from **low** to **very low** in order to result in an overall risk estimate of **very low** and achieve Australia's ALOP.

PaBV is not an OIE-listed disease and therefore there are no risk management measures recommended by the OIE.

Key features of PaBV to address include: an incubation period ranging from weeks to years; clinical signs that vary from nil to severe; and a carrier state where birds may only shed virus intermittently. Proposed measures therefore include:

- Suitable laboratory testing in both pre-export and post-entry quarantine. Repeat testing, including after a period of stress (international travel) increases the likelihood of identifying any birds that are subclinically infected and shedding virus intermittently.
- A pre-export quarantine period of at least 7 days immediately before export. This allows sufficient time for clinical signs of disease to be recognised and also provides an allowance for laboratory testing results and official certification to be obtained ahead of the scheduled export.
- A post-entry quarantine period of at least 15 days. This provides time to identify clinical illness in imported birds and it accounts for the fact that infection can be subclinical and viral shedding can be intermittent, but may be more likely following a stressful event (such as international transport). It also provides a period of time for laboratory testing to be completed and reports finalised, and for official import documentation to be assessed prior to release.

4.7.6 References

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4.8 Psittacid alphaherpesvirus 1 and psittacid herpesvirus 2

4.8.1 Background

Pacheco's disease (PD) is an acute and fatal disease of parrots that was first reported in Brazil in 1920 and was later found to be caused by psittacid alphaherpesvirus-1 (PsHV-1) (Schröder-Gravendyck et al. 2001; Simpson, Hanley & Gaskin 1975). Prior to 2015, PsHV-1 was referred to as psittacid herpesvirus-1. Four genotypes (1 to 4) corresponding to at least 3 (possibly 5) serotypes have been discovered, comprising at least 12 genetic variants (Tomaszewski, Kaleta & Phalen 2003). PsHV-1 has also been identified as the causative agent of a common, more chronic and latent form of disease known as internal papillomatous disease (IPD). IPD results in the formation of wart like lesions (papillomas) in the oral cavity, cloaca and sometimes in the upper gastrointestinal tract leading to clinical signs related to gastrointestinal or sometimes upper respiratory obstructive disease (as reviewed in Phalen 2006).

Psittacid herpesvirus-2 (PsHV-2) is phylogenetically related to PsHV-1 but sufficiently different to be considered a distinct psittacid herpesvirus (Styles, Tomaszewski & Phalen 2005). PsHV-2 remains unclassified by the International Committee on Taxonomy of Viruses (ICTV). PsHV-2 has been identified in African grey parrots and a single blue and golden macaw (Styles et al. 2004; Tomaszewski, Wigle & Phalen 2006). Evidence suggests that PsHV-2 is relatively non-pathogenic, however, it may induce mucosal and cutaneous papillomas. There is no evidence to suggest that PsHV-2 causes PD (Styles et al. 2004).

PD and IPD are not OIE-listed diseases and are not considered to be zoonotic.

4.8.2 Technical information

Epidemiology

The outcome of infection with PsHV-1 appears to be the result of a complex interaction between the genotype of the infecting virus, the species of parrot infected and other unidentified factors (Styles et al. 2004). It is thought that all genotypes are capable of causing disease, with the most pathogenic variants of PsHV-1 appearing to be genotypes 1 and 4 (Tomaszewski, Kaleta & Phalen 2003). It is hypothesised that the viral genotypes of PsHVs have co-evolved with different species of parrots and disease results when infection occurs in non-adapted susceptible species (Styles, Tomaszewski & Phalen 2005). With PsHV-1, some susceptible birds will develop PD, while others will develop subclinical infection (as reviewed in Phalen 2006).

Investigators have observed PsHV-1 infection to result in mass mortality events in parrot collections, however, instances of only a single bird being affected in a flock have also been seen (Phalen 2006).

Hosts/susceptible species

The natural hosts of PsHV-1 are psittacine birds, with some reports in the literature noting infections in passerine birds (Tomaszewski et al. 2004). Observations suggest that PsHV-1 infection is seen more commonly in imported birds, birds that have been parent-raised and birds that have survived a PD outbreak (Phalen, Tomaszewski & Styles 2004). In particular, Amazon parrots, conures (especially Patagonian conures and *Aratinga* species), and macaws appear to be more susceptible to latent PsHV-1 infection (Phalen, Tomaszewski & Styles 2004; Styles et al. 2004).

PsHV-2 appears to be primarily confined to the African grey parrot and Styles et al (2005) speculated that the virus co-evolved with this species. PsHV-2 has also been identified in a single blue and gold macaw (Tomaszewski, Wigle & Phalen 2006).

Repeated testing of infected parrots strongly suggests that they remain latently infected for life (Styles et al. 2004).

Differences in morbidity and mortality across species have been noted. For example, infection with PsHV-1 genotype 3 usually results in fatal infection in Amazon parrots but rarely in macaws (Styles et al. 2004). Genotype 4 appears to be pathogenic for both macaws and conures, whilst genotypes 1 and 2 may not cause disease at all in these species (Tomaszewski, Kaleta & Phalen 2003). Cockatiels, cockatoos and other Pacific species of birds are relatively resistant to PD, but when they do develop disease, any of the 4 genotypes may be responsible (Tomaszewski, Kaleta & Phalen 2003).

The potential range of carrier species is unknown. The majority of subclinical infections are seen in neotropical parrots, however, subclinical infection has also been reported in African and Australian parrots (Tomaszewski, Wigle & Phalen 2006).

Reports of natural infection in poultry species have not been found in the scientific literature. However, early studies demonstrated that experimental infection in day-old chicks and chick embryos was possible (Randall et al. 1979; Simpson, Hanley & Gaskin 1975).

Modes of transmission

It is widely assumed that parrots latently infected with PsHV-1 persistently shed the virus at low levels and are sources of infection for other parrots (Tomaszewski, Wigle & Phalen 2006). The route of transmission appears to be through ingestion of oral secretions and droppings from an infected bird (Tomaszewski, Wigle & Phalen 2006).

Incubation period

Experimental infections have shown the incubation period for PD to be 5-14 days (Simpson, Hanley & Gaskin 1975). Mucosal papillomas can occur as a late sequelae of subclinical infections, taking up to several months to develop (Styles et al. 2004). The incubation period of PsHV-2 is unknown.

Persistence of agent

As PsHVs are enveloped viruses, they are readily inactivated by commonly used disinfectants. Disinfectants registered for virucidal, fungicidal and bacteriocidal activity or sodium hypochlorite (bleach) solution (800 ppm) are effective for most herpesviruses.

Distribution and prevalence

A study has found that all 4 PsHV-1 genotypes are present in both the United States and Europe (Tomaszewski, Kaleta & Phalen 2003). PsHV-1 infection presumably has a worldwide distribution due to international bird trade (Tomaszewski, Kaleta & Phalen 2003). Cases have been reported in Europe, Japan, Kenya, New Zealand, North America the Middle East (in imported birds in quarantine), South Africa and Spain (as reviewed in Katoh et al. 2010). Anecdotal evidence suggests that PD outbreaks in the United Kingdom and the United States are becoming uncommon (David Phalen [The University of Sydney] 2019, pers. Comm., 1 March).

Wild-caught parrot species are known to harbour PsHV-1 infections, however, the prevalence is unknown (Tomaszewski, Wigle & Phalen 2006).

PsHV-2 has only been found in African grey parrots and a single blue and gold macaw (Styles, Tomaszewski & Phalen 2005).

Australian status

In Australia, PsHV-1 has not been reported in wild bird populations. However, in 1997 two cases of cloacal papillomatosis were diagnosed in macaws imported from the United Kingdom between 1990 and 1995 (Gallagher & Sullivan 1997; Roe 1997). This was prior to PsHV-1 being identified as a causative agent for IPD and, therefore, diagnosis was based on clinical findings alone.

In 2004, a mating pair of green-winged macaws that presented for veterinary investigation into infertility issues were diagnosed with PsHV-1 infection via PCR. The male, who exhibited IPD, was positive for PsHV-1 genotype 2, while the clinically normal female was positive for PsHV-1 genotype 3. Both birds had been imported in 1993 and were acquired by their owner in 1995. Both birds had been bred and raised numerous chicks. A third macaw, an offspring of the pair, was also positive. Attempts at sequencing the virus in this bird were unsuccessful due to the presence of 2 sequences, thought most likely to be genotypes 2 and 3. The rest of the 26 neotropical parrots in the collection were also tested via PCR and were found to be negative (Vogelneust et al. 2005).

There have been no reported cases of PD in Australia and anecdotal evidence suggests that although macaws in Australia have been diagnosed with IPD, incidence is extremely rare (Vogelneust et al. 2005).

PsHV-2 has not been detected in Australia.

Pathogenesis

In PD, viral replication occurs in a number of organs and birds develop a viremia. The most affected organ is the liver, resulting in an acute fatal hepatitis (Godwin, Jacobson & Gaskin 1982; Schmidt, Reavill & Phalen 2015). The pathogenesis of PsHV-2 infection has not been described.

Diagnosis

Clinical signs

PD is an acute and fatal disease, often without any premonitory signs and in natural infections birds are found dead (Godwin, Jacobson & Gaskin 1982). If clinical signs develop, they are mostly non-specific including lethargy, depression and anorexia (Godwin, Jacobson & Gaskin 1982). Sulphur coloured urates (biliverdinuria), indicating liver failure, has been reported to be the most consistent non-specific sign in PD (as reviewed in Phalen 2005). In experimental infections, death usually occurs within 6–10 days of inoculation and mortality reaches 100% (Godwin, Jacobson & Gaskin 1982).

In IPD, birds develop papillomas in the oral cavity, cloaca and, less frequently, in the upper gastrointestinal tract (as reviewed in Phalen 2006). These typically appear raised and pink with a cauliflower-like surface, and lesions may wax and wane (Schmidt, Reavill & Phalen 2015). Birds with disseminated papillomas in the oesophagus, crop or proventriculus may develop chronic wasting disease (as reviewed in Phalen 2006). Frequently, birds with IPD as a result of

infection with PsHV-1 genotype 3 will develop biliary and pancreatic duct carcinomas, and will often exhibit clinical signs of chronic liver disease such as weight loss, overgrown beak and poor feather quality (Graham 1991; Hillyer et al. 1991b).

There is one report in the literature of a cockatiel with an atypical presentation of PSHV-1 infection, initially diagnosed with diabetes mellitus. Histological examination revealed a chronic active pancreatitis and PsHV-1 DNA was isolated from the pancreatic lesions (Phalen, Falcon & Tomaszewski 2007).

Pathology

On gross post-mortem examination, the liver may be enlarged and friable (Schmidt, Reavill & Phalen 2015). In cases where the disease had progressed rapidly, the liver may appear normal or have diffuse colour changes resembling hepatic lipidosis (Schmidt, Reavill & Phalen 2015). Histologically, extensive hepatic and splenic necrosis is present in the vast majority of cases (Godwin, Jacobson & Gaskin 1982; Schmidt, Reavill & Phalen 2015). Necrotising lesions in multiple organs, including the pancreas and crop, are commonly seen. Intestinal lesions are relatively uncommon and a necrotising tracheitis may be rarely observed (Schmidt, Reavill & Phalen 2015). Intranuclear inclusion bodies (Cowdry Type A) are most common in the liver, but have been demonstrated in the kidneys, spleen, pancreas and small intestines (as reviewed in Phalen 2006).

In IPD, the papillomas show characteristic papillary changes to the mucosa on histopathology (Schmidt, Reavill & Phalen 2015).

Testing

PCR-based assays are available for the detection of PsHV. Studies confirm that PCR of combined oral mucosal and cloacal swabs can consistently detect viral DNA of PsHV-1 in both subclinically infected birds and those with PD (Tomaszewski, Wigle & Phalen 2006). PCR on whole blood samples is also possible but is less sensitive. Viral shedding in the majority of subclinically infected birds is constant, however, in some birds it may be intermittent and thus may lead to false negative results (Tomaszewski, Wigle & Phalen 2006).

Testing for PD in live birds is difficult due to the lack of a premonitory signs in most birds. Affected birds are strongly positive on PCR but usually die before the samples are analysed (Godwin, Jacobson & Gaskin 1982). Aspartate transaminase (AST) elevation has been reported in birds that develop clinical signs (Godwin, Jacobson & Gaskin 1982).

Treatment

Birds that develop PD will die without treatment (Phalen 2006). Treatment with acyclovir (off-label; not registered for animal use in Australia), an antiviral agent which rapidly inhibits viral replication, is effective during the early stage of infection (Phalen 2006). In an outbreak of PD, administration of acyclovir has been shown to reduce severity of disease signs and incidence of death, and mortalities usually cease within 24 hours after the commencement of flock treatment (Phalen 2006).

In IPD, avian practitioners may elect to undertake surgical removal of papillomas in extreme cases as a palliative treatment (Phalen 2006).

An earlier study by Gaskin and colleagues (1980) reports the success of an experimental vaccine for PD. In the United States, a commercial monovalent vaccine derived from a single unreported serotype was available, however, the extension of protection to other genotypes was unknown and the vaccine appears to be no longer available (as reviewed in Stegeman 2013). There is one report in the literature that documents the use of an autogenous vaccine in an outbreak of PD in a zoological garden in Italy (Kaleta & Brinkmann 1993).

Control

Controlling the spread of this virus is mainly through good management, housing sanitation and testing of birds in avicultural collections (as reviewed in Phalen 2006). It is vital to test every bird for PsHV viruses prior to introduction into a collection due to the potential for a carrier state. Any subclinically affected birds must be kept in isolation and prevented from exposure to susceptible birds.

Current biosecurity measures

Currently, there are no biosecurity measures in place for PsHV.

4.8.3 Conclusion

- PsHV-2 has not been detected in Australian captive or wild psittacine populations and is considered to be exotic to Australia.
- PsHV-2 is extremely rare overseas and there is no evidence to suggest that infection causes PD.
- Pathogenic PsHV-1 genotypes 1 and 4 are not present in Australian captive or wild parrot populations, and the prevalence of existing PsHV-1 genotypes is extremely low.
- There have been no reported cases of PD in Australia.
- Subclinically infected birds may serve as a source of introduction of exotic PsHV-1 genotypes.
- PD is not an OIE-listed disease and there are no recommendations in the OIE Code on measures for safe trade.
- PsHV-1 is not notifiable in Australia.

Therefore, the department concluded that further risk assessment of PsHV-2 was not required, however, risk assessment of PsHV-1 (genotypes 1 and 4) was required.

4.8.4 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of PsHV-1 (genotypes 1 and 4) being present in imported psittacine birds:

- All psittacine species are considered susceptible to infection.
- PsHV-1 has been reported in many countries, and presumably has a global distribution based on global trade in birds.
- The incubation period is dependent on the course of infection. PD can develop within days of infection whereas IPD may take several months to develop. Clinical signs may vary from nil to severe. Birds may be carriers and shed virus. Occasionally diagnostics tests may yield false negative results if a bird is shedding virus intermittently.

- Incursion into Australia through imported psittacines has occurred before.

Conclusion: based on this information the likelihood of importation of PsHV-1 (genotypes 1 and 4) associated with psittacine birds was estimated to be **moderate**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to PsHV-1 via an infected imported psittacine bird:

- Transmission appears to be mainly via the faecal-oral route. PsHV-1 appears to be moderately to highly contagious in some psittacine species.
- Susceptibility to infection with PsHV-1 is highest in psittacines, though passerines may be infected. Gallinaceous birds do not appear susceptible to natural infection and it is not zoonotic.
- PsHV-1 is susceptible to environmental inactivation.
- The exposure group is considered to be captive and wild birds.

Conclusion: based on this information the likelihood of susceptible species in exposure groups being exposed to PsHV-1 (genotypes 1 and 4) associated with psittacine birds was estimated to be **moderate**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of PsHV-1 (genotypes 1 and 4) in imported psittacine birds was estimated to be **low**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible avian or non-avian species to PsHV-1 has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to PsHV-1 (genotypes 1 and 4) was considered to be a **widespread outbreak**, whereby PsHV-1 establishes in directly exposed populations (captive birds and wild birds), spreads to populations of captive and wild birds and becomes endemic in Australian captive and wild birds.

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario:

- Latency/carrier states exist.
- Latency of PsHV-1 infection may delay the recognition of spread of virus into wild populations.
- Considering the epidemiology of PsHV-1, it is unlikely that this disease would be self-limiting once introduced to susceptible populations.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of PsHV-1 (genotypes 1 and 4) through Australian captive bird and wild bird populations was **moderate**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of PsHV-1 were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of PsHV-1.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Effects are likely to be limited to psittacine birds, primarily captive aviary birds.
- No vaccination for PsHV-1 is currently available in Australia.
- Based on these considerations, the effect of the establishment and/or spread of PsHV-1 for this criterion was estimated to be of minor significance at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- High mortalities are expected in a PD outbreak in wild birds.
- Based on these considerations, the effect of the establishment and/or spread of PsHV-1 for this criterion was estimated to be significant at the national level.

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- PaBV is not covered by EADRA. Individual owners would have to bear the costs of any strategies or programs they implement.
- Based on these considerations, the effect of the establishment and/or spread of PsHV-1 for this criterion was estimated to be of minor significance at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- The disease may negatively affect industries involved in breeding and selling pet/aviary psittacine birds (and related feed and equipment), and people buying birds from affected aviaries.
- Movement restrictions or other effects on domestic trade or industry are not expected.
- Based on these considerations, the effect of the establishment and/or spread of PsHV-1 for this criterion was estimated to be of minor significance at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- PsHV-1 is not OIE listed and it is not likely to cause any international trade effects.
- Based on these considerations, the effect of the establishment and/or spread of PsHV-1 for this criterion was estimated to be indiscernible at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- High mortalities associated with PD will impact local biodiversity in the affected area. Will likely have significant impacts on affected endangered species.
- Based on these considerations, the effect of the establishment and/or spread of PsHV-1 for this criterion was estimated to be significant at the national level.

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Effects on communities are likely to be minimal. Control measures are unlikely.
- Based on these considerations, the effect of the establishment and/or spread of PsHV-1 for this criterion was estimated to be indiscernible at the national level.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **moderate**.

Estimation of the likely consequences

The estimate of the likelihood of establishment and/or spread for the scenario (**moderate**) was combined with the overall effect associated with the outbreak scenario (**moderate**) using Figure 4 to obtain an estimation of likely consequences of **moderate**.

4.8.5 Risk estimation

Using Figure 5, the likelihood of entry and exposure (**low**) was combined with the likely consequences of establishment and/or spread (**moderate**), which resulted in a risk estimation of **low**.

Therefore, as the unrestricted risk estimate does not achieve Australia's ALOP, specific risk management is considered necessary for PsHV-1.

Chapter 5 proposes a combination of risk management measures to reduce the above likelihood of entry and exposure from **low** to **very low** in order to result in an overall risk estimate of **very low** and achieve Australia's ALOP.

PsHV-1 is not an OIE-listed disease and therefore there are no risk management measures recommended by the OIE. Key features of PsHV-1 to address include: an incubation period between days and several months; clinical signs that vary from nil to severe; and a carrier state where birds may only shed virus intermittently. Proposed measures therefore include:

- Suitable laboratory testing in both pre-export and post-entry quarantine. Repeat testing, including after a period of stress (international travel) increases the likelihood of identifying any birds that are subclinically infected and shedding virus intermittently.
- A pre-export quarantine period of at least 7 days immediately before export. This allows sufficient time for clinical signs of disease to be recognised and also provides an allowance for laboratory testing results and official certification to be obtained ahead of the scheduled export.
- A post-entry quarantine period of at least 15 days. This provides time to identify clinical illness in imported birds and it accounts for the fact that infection can be subclinical and viral shedding can be intermittent, but may be more likely following a stressful event (such as international transport). It also provides a period of time for laboratory testing to be completed and reports finalised, and for official import documentation to be assessed prior to release.

4.8.6 References

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4.9 Psittacine pox virus

4.9.1 Background

Avian pox viruses belong to the genus *Avipoxvirus* (family Poxviridae, subfamily Chordopoxvirinae) and are capable of causing cutaneous, diphtheritic or systemic changes in birds (ICTV 2018; Tripathy & Reed 2013). They are large, enveloped viruses containing a double-stranded DNA genome (Tripathy & Reed 2013; van Riper & Forrester 2007). Although all avian pox viruses are morphologically similar, they are antigenically and immunologically distinguishable from each other.

According to the ICTV (2018) there are currently 10 recognised types of avian pox: Canarypox virus, Fowlpox virus, Juncopox virus, Mynahpox virus, Pigeonpox virus, Psittacinepox virus (PsPoV), Quailpox virus, Sparrowpox virus, Starlingpox virus, and Turkeypox virus. Avian pox virus infections can cause significant economic losses in domestic poultry due to decreased egg production, reduced growth, and mortality (Tripathy & Reed 2013). Wild birds are also negatively affected by avian pox, with affected birds suffering increased predation, secondary infections, reduced mating success and death (Kane et al. 2012; Kleindorfer & Dudaniec 2006; Laiolo et al. 2007; Tsai et al. 1997).

Inter-species cross infection can occur with avian pox viruses and it is possible for psittacines to be infected with avian pox viruses other than PsPoV, as well as for PsPoV to infect other avian species (Gyuranecz et al. 2013). However, cross-infection is more common in passerine birds and in general the avian pox viruses are very host specific (Slocombe et al. 2013). This chapter will discuss avian pox viruses in general, focussing on PsPoV where specific information is available.

Avian pox viruses are not OIE-listed and are not nationally notifiable in Australia. While some species of avian pox virus are known to exist in Australia, PsPoV is considered exotic (WHA 2012).

There is no evidence of zoonotic transmission of any avian pox viruses.

4.9.2 Technical information

Epidemiology

Avian pox has a worldwide distribution, except there are no published reports from wild birds in the Arctic or Antarctic (van Riper & Forrester 2007). Various types of avian pox affect commercial poultry, domestic pets and wild birds. New isolates continue to be identified from a wide variety of avian species (Gyuranecz et al. 2013; Illera, Emerson & Richardson 2008; Zimmermann et al. 2011) and therefore the exact number of existing avian pox viruses, strains, and variants is unknown. Gyuranecz et al. (2013) and Jarmin et al. (2006) studied the phylogenetic analysis of the *Avipoxvirus* genus and describes an updated classification which differentiates avian pox viruses into 3 main clades (A to C) with further subclade differentiation. According to Gyuranecz et al. (2013) avian pox viruses tend to be host family or order specific, but ecological niche, habitat, and geography may modulate this pattern. Avian pox viruses are classified as mono-, bi-, or tri-pathogenic depending on their host range and specificity (van Riper & Forrester 2007).

The disease that develops from avian pox virus infection is influenced by the strain of virus, the route of exposure and host factors such as species, age and condition (Ritchie 1995b; van Riper & Forrester 2007). Prevalence of lesions can be as low as 0.5% and 1.5% (van Riper & Forrester 2007), but can vary, with host susceptibility reported to reach up to 25% (McClure 1989). In regions such as remote islands, where avian pox and its hosts have not had a long co-evolutionary history, prevalence is generally higher (Atkinson et al. 2005; van Riper et al. 2002; Vargas 1987).

Hosts/susceptible species

Avian pox viruses have been found to affect at least 232 species of birds from 23 Orders (Bolte, Meurer & Kaleta 1999; Gyuranecz et al. 2013), however, all avian species are considered susceptible to one or more strains of the virus (Gyuranecz et al. 2013). There are still many unknowns regarding host spectrums and complicating this is the fact that avian pox virus infections are often described without identifying the particular strain of virus involved, or describing the avian pox virus solely by the host species affected.

It is known that PsPoV can infect a wide range of psittacine birds and virulence differs depending on the host species. PsPoV has also been shown to infect and cause disease in chickens (even when vaccinated against fowlpox virus), however, the disease that develops is milder than in psittacines (Boosinger et al. 1982). Avian pox has been recorded in psittacine species including cockatiels, vernal hanging parrots, various lovebirds, Amazon parrots, red-fan parrots, pionus parrots, macaws, African grey parrots, red-winged parrots, grey-cheeked parrots, blue-bonnet parrots, lorries and lorikeets, red-rumped parrots, various parakeets and conures, rosellas and budgerigars (Bolte, Meurer & Kaleta 1999; Gyuranecz et al. 2013; Kirmse 1967; van Riper & Forrester 2007).

Amazon parrots (particularly the blue-fronted Amazon), pionus parrots, macaws, lovebirds, parakeets and conures appear to be more susceptible to avian pox viruses than other psittacine species. Cockatiels and cockatoos appear to be more resistant (Ritchie 1995b).

Vectors

Avian pox virus is most commonly transmitted by biting arthropods such as mosquitoes, mites, midges, and flies (Akey, Naya & Forrester 1981; Shirinov, Ibragimova & Misirov 1972; van Riper & Forrester 2007).

Mosquitoes are generally considered the primary vector, and the abundance of mosquito populations, often seasonally dependant, has been shown to play a large role in transmission and spread of avian pox viruses (Lee, Nenner & Lawrence 1958; van Riper & Forrester 2007). Several species of mosquitoes have been linked to transmission including those of the genera *Culex* and *Aedes* (DaMassa 1966; Lee, Nenner & Lawrence 1958; Matheson, Brunett & Brody 1931). Transmission occurs when a mosquito feeds on an infected bird and then feeds on an uninfected bird. Mosquitoes can retain infectious virus in their salivary glands for 2–8 weeks (Matheson, Brunett & Brody 1931) and following a single feeding can go on to infect a number of other birds (Tripathy & Reed 2013).

Modes of transmission

Transmission can occur directly, indirectly via contaminated fomites, or via mechanical vectors as described above. It has also been theorised that oral and respiratory tract infection may occur

following inhalation of aerosolised virus particles, such as those found in contaminated feathers, dried scabs, droppings or soil (van Riper & Forrester 2007).

The virus can also be transmitted via ingestion, for example when food and water sources, feeders, perches or cages are contaminated with the virus (The Department of Natural Resources n.d.). Transmission is facilitated in situations where large numbers of birds live in close proximity (van Riper & Forrester 2007).

Importantly, avian pox virus cannot penetrate unbroken skin and must enter via injured, abraded or lacerated skin, or alternatively via intact mucous membranes. Behaviour including territorial aggression, feather picking, aggressive preening or exuberant feeding can provide a route for virus entry (van Riper & Forrester 2007). Humans handling infected birds may carry the virus on their hands and clothing, leading to infection of other birds (van Riper & Forrester 2007).

Artificial insemination using semen from an infected bird may also transmit avian pox virus (Metz et al. 1985).

Incubation period

Depending on the strain of avian pox virus and host species affected, the incubation period varies from 4 days to over a month. Clinical changes have been observed in Amazon parrots 10–14 days post-infection with PsPoV, and in otherwise healthy birds with cutaneous disease only, lesions resolved within 2–6 weeks. Other infectious agents such as bacteria or fungi may invade damaged tissues, resulting in more severe disease and longer recovery. This is particularly the case when diphtheritic lesions cause defects in alimentary and respiratory mucosa (Boosinger et al. 1982; McDonald, Lowenstine & Ardans 1981; Ritchie 1995b).

Avian pox viruses can also display latency, reappearing at times of stress. Some birds that appear recovered may be persistently infected, intermittently shedding virions from the skin, feathers and gastrointestinal tract (Gartrell et al. 2003; Gerlach 1994; Ritchie 1995b)

Persistence of agent

Avian pox viruses are extremely stable in the environment, resistant to desiccation, and are able to survive for years on items such as perches and in dried organic debris such as feathers, faeces, scabs, blood and soil. The viruses are resistant to destruction with ether, and pigeonpox virus has been shown to be resistant to chloroform. Avian pox viruses can withstand 1% phenol and 1:1000 formalin for 9 days. The virus is inactivated when heated to 50°C for 30 minutes or 60°C for 8 minutes (Ritchie 1995b; Tripathy & Reed 2013; van Riper & Forrester 2007).

Distribution and prevalence

Avian pox has been observed worldwide except in more remote regions including the Arctic and Antarctic (van Riper & Forrester 2007). Accurate prevalence and geographical distribution of PsPoV is difficult to determine. Both a lack of reporting and reporting of avian pox events without identifying the particular strain of virus involved contribute to this. The disease is most commonly reported in psittacines that have been held in close proximity, such as in quarantine stations (McDonald, Lowenstine & Ardans 1981; Wheeldon, Sedgwick & Schulz 1985).

Generally, distribution and prevalence of avian pox virus is linked to 3 factors: climatic conditions (infection is most common in temperate and warmer parts of the world, and

following periods of extensive rainfall), vector numbers (in particular mosquitoes—outbreaks are often linked to the seasonal mosquito cycles), and host species density and susceptibility (Akey, Naya & Forrester 1981; Forrester & Spalding 2003; Ritchie 1995b; van Riper & Forrester 2007).

The prevalence of avian pox lesions in established wild bird populations is reported to be between 0.5 and 1.5%. In more naïve populations prevalence can rise to as high as 50% (van Riper & Forrester 2007).

Australian Status

Poultry in Australia and a number of native bird species (non-psittacine) have been reported to have pox virus infections. For example, Fowlpox is endemic and managed in the domestic poultry industry. Exact prevalence information on other strains is difficult to find as much of the information does not distinguish the species of avian pox, though some studies do so (Sarker et al. 2017).

PsPoV has never been identified and is considered exotic. A pox virus was identified in 2 wild-caught crimson rosellas in 2002 and 2008 in south-eastern Australia. DNA analysis from both cases concluded that the virus was a previously unrecognized avian pox virus endemic to this region of Australia, specific to this species and of low virulence (Slocombe et al. 2013).

Pathogenesis

Avian pox virus can cause cutaneous, diphtheritic or systemic lesions and the expression of disease varies according to strain of virus, route of infection, and the species of bird affected, its age and condition (Gartrell et al. 2003; Ritchie 1995b; Tripathy & Reed 2013). In the cutaneous form, after avian pox virus enters a host's dermal epithelial cells, viral DNA replication begins between 12 and 24 hours later, followed by an exponential rate of synthesis between 60 and 72 hours. A relatively long latent period follows in which the viroplasmic particles condense, acquire an outer membrane and become incomplete virions. A process whereby the virions gain a membrane coat and an additional outer membrane follows, producing the classical Bollinger bodies (inclusion bodies) that are observable by light microscopy (van Riper & Forrester 2007). The pathogenesis of the diphtheritic form of avian pox is unknown and may require prolonged incubation or other contributing factors to facilitate development of disease (Ritchie 1995b). Systemic infections are characterised by a primary viremia, transport of virus to and viral replication in the liver and bone marrow, which leads to a secondary viremia and more substantial lesions throughout the body (Ritchie 1995b).

Mortality is usually low in psittacine birds affected by the mild cutaneous form of the disease and affected birds tend to recover within 2–6 weeks. With diphtheritic or systemic forms of disease, or when complicated by factors such as poor environmental conditions or concurrent disease, mortality may increase (Ritchie 1995b; Tripathy & Reed 2013). Highly susceptible species of parrots may develop severe upper respiratory tract disease (as reviewed by Katoh et al. 2010).

Diagnosis

Clinical signs

Cutaneous

The cutaneous form of avian pox virus, in which discrete wart-like proliferative lesions develop on the skin, is the most commonly seen form of infection (van Riper & Forrester 2007).

Psittacines with the cutaneous form of PsPoV may develop nodules on the unfeathered parts of the skin, most commonly around the eyes, cere and feet (González-Hein, González & Hidalgo 2008). Serous ocular discharge, rhinitis and conjunctivitis followed by ulceration of eyelid margins and medial and lateral canthi of the eyes may also be present (Kato et al. 2010; Ritchie 1995b). Disease can resolve within a month or persist for more than a year (Gartrell et al. 2003). Permanent damage to the eyes and surrounding skin may occur and in rare cases ocular lesions may include ulcers or crystallisation of the cornea and anterior uveitis (Ritchie 1995b; Tripathy & Reed 2013).

Diphtheritic

The diphtheritic form of avian pox virus is less common in most species, however, it is frequent in psittacine birds with PsPoV. This form causes more severe disease, with development of moist, necrotic lesions on mucous membranes (Ritchie 1995b; van Riper & Forrester 2007). Disease in psittacines with the diphtheritic form of PsPoV is characterised by fibrino-necrotic lesions on oral, pharyngeal, oesophageal or crop mucosa (Ritchie 1995b; Tripathy & Reed 2013). If these lesions are damaged or removed, they tend to bleed profusely (Gartrell et al. 2003; Ritchie 1995b). If oral or nasal passages become occluded with plaques, affected birds may display rhinitis, dysphagia or dyspnoea, and subsequent starvation or suffocation may cause death. When defects in the mucosal lesions allow for invasion of bacterial or fungal agents, more severe disease can occur including pneumonia or airsacculitis (Ritchie 1995b; Tripathy & Reed 2013).

Systemic

In rare cases, avian pox virus infection may become systemic (van Riper & Forrester 2007). In these infections, birds may develop a fibrinous inflammation of serous membranes, liver degeneration or necrosis, oedema and hyperaemia of the lungs and fibrinous pneumonitis (McDonald, Lowenstine & Ardans 1981; Ritchie 1995b; Tripathy & Reed 2013).

Pathology

Localised proliferations of epithelial cells form the characteristic wart-like cutaneous pox lesions, and affected cells appear hyperplastic and hypertrophic. As the cells mature in layers of epithelium, large granular acidophilic intracytoplasmic inclusions appear. Avian pox lesions that are raised may predispose skin surfaces to trauma and subsequent invasion by bacteria or fungi, which can be seen histologically. However, in most instances the lesions are self-limiting and slough off without secondary infection (van Riper & Forrester 2007). Diphtheritic lesions may appear as white, opaque, slightly elevated nodules that often coalesce to form yellowish, cheesy, necrotic material that has the appearance of a pseudomembrane. These lesions may rapidly increase in size and become aggravated by the invasion of bacteria or fungi (Ritchie 1995b; van Riper & Forrester 2007). In systemic infections, there may be fibrinous inflammation of serous membranes. Lungs may appear oedematous and hyperaemic with a fibrinous pneumonitis, and liver degeneration or necrosis may also be present (van Riper & Forrester 2007).

Testing

Although the presence of gross lesions on a bird's body may indicate avian pox virus infection, there are a number of other avian diseases that present similarly. A definitive diagnosis is ideally obtained by isolating the virus via propagation on chorioallantoic membranes of chicken embryos. Not all strains of avian pox virus, particularly those that infect wild birds, grow readily in chicken embryos and, therefore, other avian embryos must be used. If virus isolation is not possible, histological examination demonstrating classical Bollinger bodies or electron microscopy demonstrating typical avian pox virus particles can also provide confirmation of infection (van Riper & Forrester 2007). Other diagnostic techniques including ELISA, hemagglutination inhibition, virus neutralisation and PCR may be used. PsPoV infection in particular can be confirmed by PCR (Kato et al. 2010), however, this test may only be reliable when lesions are present and may not detect subclinical carriers of the virus (Gartrell et al. 2003).

Treatment

Treatments exist both for signs of the disease and to prevent secondary infection. These include removing skin lesions and washing the area with sodium bicarbonate or iodine, bathing the eyes with a saline solution, removing diphtheritic membranes from the mouth and throat and applying iodine, and raising the environmental temperature (The Department of Natural Resources n.d.). In most cases, treating an infected bird spreads the infection to other parts of the skin and, if proper care is not taken, to other birds (Ritchie 1995b; The Department of Natural Resources n.d.; Tripathy & Reed 2013).

Control

Vaccines exist to control some avian pox virus strains including fowlpox and pigeonpox. No specific vaccine is available to control PsPoV infection and the existing vaccines for other avian pox viruses do not protect against PsPoV (Gyuranecz et al. 2013; Samanta & Bandyopadhyay 2017).

Vector reduction methods including control of adults and of breeding sites should be performed in outbreaks where vectors are contributing to transmission (van Riper & Forrester 2007). In captive settings, PsPoV can be controlled by limiting vector access to susceptible birds and isolating any bird with suspected infection (Ritchie 1995b). Regular sterilisation of feeders, waterers, bathers and perches in settings where birds are being artificially concentrated (e.g. aviaries) will assist in controlling spread of the disease (van Riper & Forrester 2007).

A PsPoV infection in a wild psittacine population is very difficult to control (WHA 2012).

Current biosecurity measures

Biosecurity measures exist for this disease for the importation of pet birds from New Zealand. The requirement is that the birds are examined by a New Zealand Government Veterinary Officer within one week of commencing the pre-export quarantine period and either no suspicion/evidence of any lesions suggestive of avian pox is found, or if lesions suggestive of avian pox in psittacine birds are detected, they are shown not to be caused by psittacine pox virus. The birds must have been completely isolated from insect vectors (including mosquitoes) during the pre-export quarantine period and during transport to Australia (Department of Agriculture and Water Resources 2018).

4.9.3 Conclusion

- PsPoV is present in other countries and is not present in Australia.
- Australia has suitable vectors present to propagate transmission.
- PsPoV can cause severe disease in psittacine birds.
- PsPoV is not a nationally notifiable disease in Australia and there are no control measures in place.
- PsPoV is not an OIE-listed disease agent and there are no recommendations in the OIE Code on measures for safe trade.

Therefore, the department concluded that further risk assessment of PsPoV was required.

4.9.4 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of PsPoV being present in imported psittacine birds:

- All psittacine species are considered susceptible to infection.
- The diphtheritic form of avian pox virus is frequent in psittacine birds with PsPoV. Whilst the pathogenesis of the diphtheritic form is unknown and may require prolonged incubation or other contributing factors, the disease is more severe with obvious clinical signs.
- Avian pox viruses have a global distribution.
- The exact prevalence of PsPoV is unknown. It is more commonly diagnosed in psittacines housed under stressful conditions.
- The incubation period varies from days to over a month. Clinical signs may vary from nil to severe. Birds may be carriers and shed intermittently and there are a number of limitations to reliable detection using diagnostic methods.

Conclusion: based on this information the likelihood of importation of PsPoV associated with psittacine birds was estimated to be **moderate**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to PsPoV via an infected imported psittacine bird:

- Transmission is primarily via mechanical vectors (biting insects) but also via inhalation or ingestion, indirectly via contaminated fomites, or via artificial insemination using semen from an infected bird. PsPoV appears to be highly contagious.
- Avian pox viruses have been found in 23 Orders of birds, however, strains tend to be host-specific. Poultry are susceptible to infection with PsPoV, however, they develop milder disease than psittacines. Avian pox viruses are not zoonotic.
- PsPoV survives extremely well in the environment – for years under favourable conditions.
- The exposure groups are considered to be captive birds, wild birds and low biosecurity poultry.

Conclusion: based on this information the likelihood of susceptible species in exposure groups being exposed to PsPoV associated with psittacine birds was estimated to be **high**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of PsPoV in imported psittacine birds was estimated to be **moderate**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible avian or non-avian species to PsPoV has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to PsPoV was considered to be a **widespread outbreak**, whereby PsPoV establishes in directly exposed populations (captive birds, wild birds and/or low biosecurity poultry), is readily transmitted by vectors, and becomes endemic in Australian captive and wild birds.

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario:

- Latency/carrier states exist.
- Potential for rapid spread via vector transmission - a single feed by a mosquito on an infected bird can lead to infection of multiple other birds. Due to their housing arrangements, household pet birds are less likely to contribute to vector transmission than aviary birds.
- As vectors play a major role in transmission, circulation and maintenance of infection in populations would be impacted by vector presence, distribution and density.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of PsPoV through captive (outdoor) and wild bird populations was **high**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of PsPoV were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of PsPoV (in addition to those outlined in section 2.3.4).

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Effects are likely to be mostly limited to psittacine birds.
- No vaccination for PsPoV is currently available in Australia.
- Based on these considerations, the effect of the establishment and/or spread of PsPoV for this criterion was estimated to be of minor significance at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- Overseas, introduction of avian pox virus has negatively impacted naïve native populations of wild birds. An outbreak in wild psittacine birds may have similar impacts.
- Based on these considerations, the effect of the establishment and/or spread of PsPoV for this criterion was estimated to be of minor significance at the national level.

*Indirect effects**The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs*

- PsPoV is not covered by EADRA. Individual owners would have to bear the costs of any strategies or programs they implement.
- Based on these considerations, the effect of the establishment and/or spread of PsPoV for this criterion was estimated to be of minor significance at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- The disease may negatively affect industries involved in breeding and selling pet/aviary psittacine birds (and related feed and equipment), and people buying birds from affected aviaries.
- Movement restrictions or other effects on domestic trade or industry are not expected.
- Based on these considerations, the effect of the establishment and/or spread of PsPoV for this criterion was estimated to be of minor significance at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- PsPoV is not OIE listed and it is not likely to cause any international trade effects.
- Based on these considerations, the effect of the establishment and/or spread of PsPoV for this criterion was estimated to be indiscernible at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- It is likely that any negative effects on the environment would be of minor significance at the national level.

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Effects on communities are likely to be minimal. Control measures are unlikely.
- Based on these considerations, the effect of the establishment and/or spread of PsPoV for this criterion was estimated to be of minor significance at the national level.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **low**.

Estimation of the likely consequences

The estimate of the likelihood of establishment and/or spread for the scenario (**high**) was combined with the overall effect associated with the outbreak scenario (**low**) using Figure 4 to obtain an estimation of likely consequences of **low**.

4.9.5 Risk estimation

Using Figure 5, the likelihood of entry and exposure (**moderate**) was combined with the likely consequences of establishment and/or spread (**low**), which resulted in a risk estimation of **low**.

Therefore, as the unrestricted risk estimate does not achieve Australia's ALOP, specific risk management is considered necessary for this agent.

Chapter 4.11 proposes a combination of risk management measures to reduce the above likelihood of entry and exposure from **moderate** to **low** in order to result in an overall risk estimate of **very low** and achieve Australia's ALOP.

PsPOV is not an OIE-listed disease and therefore there are no risk management measures recommended by the OIE.

Key features of PsPoV to address include: an incubation period between 4 to over 30 days and the presence of clinically detectable lesions; a state of latency in some birds that may cause lesions and/or viral shedding to reappear during times of stress; transmission by vectors especially mosquitoes; lack of reliable diagnostic tests. Proposed measures therefore include:

- A pre-export and post-entry quarantine period of at least 30 days and 14 days respectively, in order to identify clinically affected birds.
- A requirement that the bird undergo veterinary examination during a period of pre-export quarantine and either no lesions suggestive of avian pox are present or, if such lesions are present, they are comprehensively investigated and avian pox is ruled out as a cause.
- A requirement that the bird undergo veterinary examination during a period of post-entry quarantine (when the stress of transport may unmask subclinical disease or carrier states) and either no lesions suggestive of avian pox are present or, if such lesions are present, they are comprehensively investigated and avian pox is ruled out as a cause.

4.9.6 References

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4.10 Reovirus

4.10.1 Background

Avian reoviruses are members of the Orthoreovirus genus within the family Reoviridae (Mertens et al. 2005). There are various strains, but attempts to classify them according to their serological properties have been unsuccessful due to their high degree of antigenic heterogeneity and cross-reactivity in neutralisation tests (Benavente & Martínez-Costas 2007). Reoviruses may frequently exist as antigenic subtypes, rather than distinct serotypes and reassortment can occur (Robertson & Wilcox 1984). Because of the difficulty of sub-classification, different avian reovirus strains are referred to collectively as reovirus in this chapter.

Reovirus was first isolated by Fahey and Crawley (1954), from the respiratory tract of chickens with chronic respiratory disease, and became known as the 'viral arthritis agent' when Olson and Kerr (1966) demonstrated its association with viral arthritis lesions in chickens. Morphological data from electron microscopy studies conducted by Walker et al. (1972) allowed its further identification and classification as a reovirus. Reovirus is a non-enveloped, double stranded RNA virus (Benavente & Martínez-Costas 2007).

Reovirus has been isolated from domestic poultry, ducks, pigeons, psittacines and wild bird populations (Fahey & Crawley 1954; Graham 1987; McFerran, Connor & McCracken 1976; Styś-Fijoł, Kozdruń & Czekaj 2017). It is considered to be ubiquitous in poultry populations worldwide and has been isolated from birds displaying a variety of disease manifestations, although an etiological relationship with most of these diseases remains unestablished (as reviewed in Rosenberger, Olson & Van der Heide 1998). The exception to this is the causality that has been determined between reovirus and tenosynovitis in chickens and turkeys (Jones & Onunkwo 1978; Sharafeldin et al. 2014). Reovirus has been shown to cause significant economic losses in poultry breeding operations. This is due to conditions such as poor growth, uneven feathering, lack of bodyweight uniformity, leg swelling, swelling of hock and wing joints and increased flock mortality (Dobson & Glisson 1992).

The isolation of reovirus from psittacine birds imported into various overseas countries indicates that it is prevalent worldwide (Meulemans et al. 1983; Wilson et al. 1985).

There are various clinical signs consistently associated with reovirus infections in psittacines. These include hepatitis, enteritis, splenomegaly, pneumonia and air sacculitis (Conzo et al. 2001; Graham 1987; Meulemans et al. 1983). However, reports of reovirus being conclusively identified as the causative agent of disease are rare. In many cases, reovirus is isolated in birds with concurrent infections, and clinical signs have been attributed to reovirus where it has been the common agent among birds displaying similar lesions (Conzo et al. 2001; Sanchez-Cordon et al. 2002). In psittacine birds, reovirus is generally considered to be non-pathogenic due to its isolation from clinically healthy birds. However, it has been speculated that stress associated immunosuppression can cause viral shedding and transmission, and that reovirus may contribute to disease caused by other pathogens. Other factors thought to influence disease outbreaks are species susceptibility, secondary pathogens and the pathogenicity of the viral strain (Conzo et al. 2001; Van den Brand et al. 2007).

Reovirus is not zoonotic, not OIE-listed and is not notifiable or subject to official control or eradication in Australia. Currently, no avian reovirus-specific biosecurity measures exist for importation of products or live animals into Australia.

4.10.2 Technical information

Epidemiology

Avian reovirus affects a wide range of avian species, including poultry, and psittacines, and has been associated with various disease conditions. Of these conditions, only the link between reovirus infection and viral arthritis in chickens and turkeys has been well established (Jones & Onunkwo 1978; Sharafeldin et al. 2014).

There are multiple strains of reovirus, with varying pathogenicity, and concurrent infections with more than one reovirus strain with different levels of pathogenicity can occur (Conzo et al. 2001; Rosenberger et al. 1989). Younger birds have been shown to be more susceptible to infection and show more severe disease signs. This age-related resistance is thought to result from an improved ability to reduce viral dissemination throughout the body (Jones & Georgiou 1984a; Montgomery, Villegas & Kleven 1986; Roessler & Rosenberger 1989). The suppressive effects of reovirus on the immune system are discussed later in this chapter.

Factors that affect the severity of disease and clinical signs include age, species, pathogenicity, immune status and route of transmission (Montgomery, Villegas & Kleven 1986; Rosenberger et al. 1989; Sharma, Karaca & Pertile 1994).

Hosts/susceptible species

Reovirus has been isolated from multiple avian species, including chickens, pigeons, ducks, geese, turkeys, raptors, psittacines, quail and various wild bird populations (Fahey & Crawley 1954; Graham 1987; Lu et al. 2015; Magee et al. 1993; McFerran, Connor & McCracken 1976; Palya et al. 2003; Styś-Fijoł, Kozdruń & Czekaj 2017).

Studies on the characterisation of reovirus isolates from psittacines have shown these to be distinct from isolates affecting poultry (Meulemans et al. 1983; Van den Brand et al. 2007). Studies have shown some capacity for cross-species infectivity in experimental conditions, but there is a lack of evidence to suggest this occurs under natural conditions. (Styś-Fijoł, Kozdruń & Czekaj 2017).

Reovirus infections have been reported in multiple psittacine species, including African grey parrots, various parakeet varieties, budgerigars, Australian king-parrots, cockatoos, Amazon parrots, Senegal parrots, Indian ringnecks, hawk-headed parrots, lovebirds, and lorikeets (Conzo et al. 2001; Gaskin 1987; Meulemans et al. 1983; Perpiñán et al. 2010).

In an aviary environment, reovirus was found to affect both Old World psittacines and New World psittacines, but infection was twice as likely among Old World psittacines. This supports the speculation that Old World psittacines are more susceptible to reovirus infections than New World psittacines (Conzo et al. 2001; Van den Brand et al. 2007).

Modes of transmission

Most available literature on the transmission of avian reovirus have been conducted on chickens in experimental conditions.

The exact route of transmission of reovirus in aviary and companion birds remains speculative. However, the faecal-oral route, from contact with contaminated faecal dust, is thought to be the main source of infection in aviaries due to frequent reovirus recovery from the intestinal tract of

aviary psittacines (as reviewed in Ritchie 1995a). This is supported by the recovery of reovirus from the faeces of chicks inoculated with reovirus via the footpad and oral routes (Jones & Georgiou 1984a).

Horizontal transmission of reovirus was demonstrated in a study by Ni and Kemp (1995), with reovirus inoculated orally and via the footpad. Histopathological changes and virus replication were found in various visceral tissues, the bursa, hock joint and bone marrow by 8 days post-inoculation of day-old broilers. Vertical transmission has also been demonstrated. Reovirus was isolated from chicks hatched from eggs laid by hens inoculated with the virus via the oral, nasal and tracheal routes (Al-Muffarej, Savage & Jones 1996; Menendez, Calnek & Cowen 1975).

The persistence of reovirus in the caecal tonsils and hock joints of infected birds indicates that carrier birds might contribute to disease spread (Jones & Georgiou 1984a). Van der Brandt (2007) suggested this to explain 2 reovirus associated disease outbreaks in the EU—one in the United Kingdom and another in the Netherlands—caused by the same reovirus strain. Other factors thought to facilitate disease spread include migrating wild birds, attendance at bird shows and bird markets, and the introduction of new birds into existing collections.

Incubation period

The incubation period can vary depending on host age, virus pathotype and route of exposure (as reviewed in Jones & Georgiou 1984b; Van der Heide 1977).

The experimental incubation period in most birds is 2–9 days (as reviewed in Ritchie 1995a). Infection outcomes have been shown to vary in African grey parrots and cockatoo species, with some birds developing clinical disease followed by death, and others remaining subclinical (Gaskin 1989; Graham 1987). Symptomatic birds have been shown to develop similar clinical signs, viral shedding patterns and immunological responses to inoculations via the oral, intratracheal or intramuscular routes. Experimentally infected African grey parrots died on day 8 and 9 and had lesions consistent with natural disease (Gaskin 1989).

Intramuscular administration of reovirus in umbrella cockatoos and African grey parrots resulted in viral shedding 2–15 days post-inoculation and precipitating antibodies to reovirus antigens developed as early as 7 days post-inoculation (Gaskin 1989).

Persistence of agent

Avian reovirus is stable for up to 2 months at room temperature (Robertson & Wilcox 1984), more than 3 years at 4°C, and over 4 years at –20°C (Rosenberger 2003). It has been reported to persist for at least 10 days on the surface of egg shells when organic material is present, at least 10 days on feathers, wood shavings, chicken feed, metal, glass and rubber, and for at least 10 weeks in water (Jones 2000; Savage & Jones 2003).

Reovirus is heat resistant and able to survive at 60°C for 8–10 hours (Rosenberger 2003), and has been shown to be destroyed within 3 minutes in effluent heated to 82.2°C (Chmielewski et al. 2011). It is relatively resistant to disinfectants such as 2% formaldehyde at 4°C and 2% phenol at room temperature, but sensitive to 100% ethyl alcohol and chlorine disinfectants (Robertson & Wilcox 1984). Reovirus is stable over a wide pH range with studies reporting stability at pH 3.0 and pH 9.0 for 4 hours at 4°C, and at pH 3.0 and pH 7.0 for 3 to 5 hours at room temperature (Gershowitz & Wooley 1973; Glass et al. 1973; Robertson & Wilcox 1986).

Distribution and prevalence

Avian reovirus has a worldwide distribution and has been recognised to be ubiquitous in poultry in all major poultry producing areas (Jones 2013; Rosenberger et al. 1989).

Studies are lacking on the prevalence of reovirus infections in avian species other than domestic poultry. Reovirus isolations have been made from wild birds, but experimental work has not been conducted (as reviewed in Jones ; Magee et al. 1993; Malkinson 1981; Palya et al. 2003; Sanchez-Cordon et al. 2002; Vindevogel et al. 1982).

Isolation of reovirus from various imported psittacine species has indicated its worldwide distribution. Reovirus was isolated from 53% of psittacine bird consignments imported into Belgium over a 2-year period, with a total of 8,359 infected birds. Mortality rates ranged from 0.6 to 100%, and it was suggested that deaths were attributable also to the confounding effect of transport stress on existing viral infections. These birds originated from the African continent, South Asia, Malaysia and the Czech Republic (Meulemans et al. 1983).

In the United States, reovirus occurrence was studied over a 7 year period and isolated from 4.1% of psittacines tested during quarantine, although the origins of these birds were not described. There was no correlation between clinical disease and excessive mortality in reovirus affected birds (Senne et al. 1983).

A study conducted in Canada, isolated reovirus from 8% (22 out of 269) of groups of psittacine bird imports over a 3 year period (as reviewed in Ritchie 1995a). Conzo (2001) describes the isolation of reovirus from Australian king parrots imported into Italy from New Zealand. In this study, the deaths of 4 out of the 10 Australian king parrots was attributed exclusively to reovirus, as no other infectious agent was detected.

Australian status

There are distinct antigenic types of avian reovirus affecting poultry in Australia (Meanger et al. 1997). Vaccination of poultry for reovirus is not practiced in Australia as these strains appear to be of low virulence, isolated in concurrent infections with other disease agents and rarely determined to be the primary disease causing agent. However, there are reports of reovirus being associated with disease in young chickens, with hydropericardium as a distinctive lesion (Hussain, Spradbrow & MacKenzie 1981; Meanger et al. 1997).

Information about the presence or prevalence of reovirus in captive or wild Australian psittacine birds is lacking.

Pathogenesis

Avian reovirus is rarely identified as the primary disease causing agent, however, it is frequently recovered in conjunction with other pathogens, including *Salmonella* spp., *Aspergillus* spp., *Escherichia coli*, *Chlamydia psittaci*, *Mucor* spp. and paramyxoviruses (Conzo et al. 2001; Sanchez-Cordon et al. 2002; Van den Brand et al. 2007). Although associated with a variety of disease conditions in multiple avian species, this finding may be incidental as the only well established causative link between reovirus infection and disease is viral arthritis in chickens and turkeys (Jones & Onunkwo 1978; Sharafeldin et al. 2014).

This said, a necrotising hepatitis is reported to be a common lesion to psittacines with concurrent infections involving reovirus. This pathology has also been observed in psittacines

infected with reovirus only, and has been replicated in experimental conditions, suggesting the possibility of a primary pathogenic role of these viruses (Conzo et al. 2001; Graham 1987).

While some authors consider reovirus to be highly immunosuppressive, others have suggested that avian reovirus does not affect sufficient immunological parameters to cause generalised immunosuppression, and hence cannot be considered an immunosuppressive agent (Dohms & Saif 1984; Montgomery, Villegas & Kleven 1986; Sanchez-Cordon et al. 2002). Studies have shown avian reovirus to alter the function of the chicken immune system. The virus affects both B and T lymphocytes, and induces changes such as decreased Bursa of Fabricius weights (with consequent lymphopenia), increased splenic weights, elevated white cell counts and viral replication in macrophages (Kibenge, Jones & Savage 1987; Montgomery et al. 1986).

A role in immune compromise could explain the occurrence of reovirus outbreaks associated with stress factors such as post-transport quarantine, the introduction or mixing of birds and colder climates (Conzo et al. 2001; Meulemans et al. 1983; Van den Brand et al. 2007).

Avian reovirus replicates principally in the digestive tract, but can also replicate and persist in a variety of other tissue types. Following oral inoculation in chickens under experimental conditions, an initial (24-48 hour) replication in the intestinal mucosa was followed by viremia and dissemination to virtually all other organs, persisting for long periods in the lymphoid tissues and oviduct (Gaskin 1989).

Disease resistance appears to be associated with the development of neutralising antibodies, however, birds with and without titres have been shown to intermittently shed reovirus in their faeces (Gaskin 1989; as reviewed in Ritchie 1995a). In experimentally infected African grey parrots, viral shedding occurred from 2 to 15 days post-inoculation and precipitating antibody production was temporarily associated with a cessation in virus shedding. Precipitating antibodies developed as early as 7 days post-inoculation and persisted for 2-6 weeks. However, the development of precipitating antibodies was not proven to have a neutralising effect, with an African grey parrot still having reovirus recovered from the spleen (but not the liver or lung) (Gaskin 1989).

The persistence of reovirus isolated from caecal tonsils and hock joints of subclinical birds indicates that clinically recovered birds might develop latent infections and serve as carrier birds that contribute to viral spread (Jones 2000; Jones & Georgiou 1984b; Senne et al. 1983; Van den Brand et al. 2007).

In psittacines, there is evidence of widely dispersed disease outbreaks associated with reovirus strains of varying pathogenicity in aviaries and potentially wild bird populations. Reports of deaths in isolated companion birds, including birds in quarantine facilities, supports the hypothesis that persistent infections can occur (Conzo et al. 2001; Gaskin 1989; Meulemans et al. 1983; Van den Brand et al. 2007). Disease outbreaks associated with reovirus have also been reported to recur in large flocks every few months for years (Van den Brand et al. 2007).

Diagnosis

Clinical signs

Reovirus in psittacines has been isolated alone or in mixed infections with other disease agents. In reports of the latter, its association with pathology was hypothesised based on it being the agent common to infected birds and the consistency of lesions found in birds from which it was

isolated (Meulemans et al. 1983; Sanchez-Cordon et al. 2002; Van den Brand et al. 2007). There are some reports of reovirus causing mortalities with no other agents identified (Conzo et al. 2001; Meulemans et al. 1983).

In psittacines, reovirus infections have been associated with a variety of clinical signs including hepatitis, enteritis, splenomegaly and pneumonia. The association with necrotizing hepatitis in the absence of other pathogens in some cases suggests reovirus may have a primary pathogenic role (Conzo et al. 2001).

Graham (1987) reported subcutaneous haemorrhages, multiple necrotic foci in the liver, spleen, bone marrow and intestinal lamina propria, air sacculitis and epicarditis in an African grey parrot. Subsequent experimental inoculation of 2 African grey parrots with the isolate was fatal and reproduced lesions similar to the original condition.

Meulemans (1983) isolated reovirus alone and in combination with *Salmonella* spp. in a variety of imported psittacine species. Pathologic findings included hepatitis, splenomegaly and occasionally pneumonia. In a study of mixed viral infection in psittacine birds, in which the reovirus was the only common etiologic agent, generalised congestion, mild catarrhal enteritis, decrease in size of the Bursa of Fabricius and mild splenomegaly were observed (Sanchez-Cordon et al. 2002). Non-specific clinical signs, including incoordination, emaciation and diarrhoea, have been reported in cockatoos (as reviewed in Van den Brand et al. 2007).

Pathology

In psittacines, histological lesions have been found to be similar in naturally and experimentally infected birds, with the highest morbidity and mortality seen in birds with concurrent infections (Gaskin 1989; Graham 1987).

Histological changes include multifocal coagulation necrosis in the spleen and liver, populations of mononuclear (mostly lymphoreticular) cells in the hepatic sinusoids and remaining periportal regions, and necrotic foci in the bone marrow. The small intestinal lamina propria can contain foci of reticuloendothelial cell hyperplasia and mononuclear cellular infiltrates (Conzo et al. 2001; Graham 1987; Van den Brand et al. 2007). Gross lesions in the cells that line the respiratory and digestive tracts may mimic polyomavirus, adenovirus or Pacheco's disease (as reviewed in Ritchie 1995a; Sanchez-Cordon et al. 2002).

In some cases, mycotic pneumonia and airsacculitis have been found (Conzo et al. 2001; Meulemans et al. 1983; Van den Brand et al. 2007). Other findings have included fibrinous airsacculitis, lung congestion and ascites (Conzo et al. 2001; Meulemans et al. 1983).

Testing

Reovirus detection typically occurs post-mortem in birds submitted for necropsy, bacteriological and virological examinations. (Conzo et al. 2001; Van den Brand et al. 2007). Infection is confirmed by viral isolation from the faeces, gastrointestinal contents, liver, heart, kidney or lung (Graham 1987; Van den Brand et al. 2007).

Real-time PCR has become a widely used diagnostic method. When its use is followed by restriction enzyme fragment length polymorphism (RFLP), it can be used as a simple and rapid approach to characterising reovirus isolates. Their molecular characterisation using qPCR and nucleotide sequencing analysis, in particular by sequencing σ proteins, has been described

(Hellal Kort et al. 2013; Kant et al. 2003; Liu et al. 1999). PCR can also be used to detect the presence of concurrent infections with other pathogens (as reviewed in Jones 2013; Van den Brand et al. 2007).

The detection of reovirus by routine microscopic examination is difficult, but it can be used to demonstrate viral particles in the faeces and respiratory secretions of clinically diseased birds if sufficient virus is present. However, it is also common to find reovirus in the faeces of clinically normal birds (as reviewed in Conzo et al. 2001; Meulemans et al. 1983; Ritchie 1995a).

ELISA is the serological diagnostic method of choice, allowing for rapid, large-scale testing. A significant correlation has been found between ELISA results and virus neutralising antibody levels. An ELISA capable of differentiating between animals infected with, and those vaccinated against, reovirus has been described (Goldenberg et al. 2011; Islam & Jones 1988).

Other serological tests for detecting reovirus infections include the agar gel precipitin (AGP) test, indirect fluorescent antibody (IFA) assay and virus neutralisation by plaque reduction (PR) methods. IFA has been shown to be more sensitive than AGP, but less sensitive than the PR test in detecting reovirus infections in chickens. However, none of these are suitable as large-scale screening tests (Ide 1982; Jones 2013).

Treatment

There are no reported treatments for reovirus infections. New World psittacines are thought to be more resistant than Old World psittacines to reovirus-associated disease, and hence more likely to recover with supportive care. Notwithstanding the fact that reovirus is commonly found in healthy birds and its association with disease in psittacines is not well understood, when Old World species of psittacines suffer from disease that is thought to be caused by reovirus, the prognosis tends to be poor (Conzo et al. 2001; as reviewed in Ritchie 1995a; Van den Brand et al. 2007). Birds that recover from the disease could remain latently infected and become carriers of persistent infections (Jones & Georgiou 1984a; Van den Brand et al. 2007).

There is no evidence in currently available literature that persistently infected birds can be reliably detected.

Control

Globally, reovirus infections in chicken populations are commonly controlled with vaccination. Vaccination against reovirus is not used in Australian poultry populations as reovirus strains present here appear to be of low virulence (Hussain, Spradbrow & MacKenzie 1981; Meanger et al. 1997).

The reovirus vaccines available for chickens are of little value in protecting companion and aviary birds because virus strains commonly found in these species are generally antigenically unrelated to those found in poultry (Gaskin 1989). An experimental inactivated vaccine produced from a psittacine reovirus isolate was reported to reduce mortality levels in infected African grey parrots and cockatoos (as reviewed in Ritchie 1995a).

Given reovirus's resistance to inactivation, its stability in the environment and intermittent virus shedding from infected animals, maintaining freedom from infection in intensively housed chickens is thought to be almost impossible (as reviewed in Jones 2013). In domestic poultry flocks, disease outbreaks have been seen to recur every few months for years (Van den Brand et

al. 2007). Removing infected flocks and thorough cleaning and disinfection of housing can prevent infection of subsequent groups. Multi-component disinfectants and 0.5% organic iodine solutions are considered to be effective in inactivating reovirus. Commercially available disinfectants should be validated for efficacy against reovirus before use (as reviewed in Jones 2013).

Current biosecurity measures

Reovirus is not OIE-listed and is not notifiable nor subject to official control or eradication in Australia. Currently, no biosecurity measures exist specific to avian reovirus for importation of live animals or animal products into Australia.

Conclusion

- Avian reovirus is present worldwide and there are non-virulent poultry strains present in Australia in chickens. Information about the presence or prevalence of reovirus in Australian psittacine birds is lacking.
- In Australia, avian reovirus is not nationally notifiable and there are no control measures in place.
- Avian reovirus is not an OIE-listed disease agent and there are no recommendations in the OIE Code on measures for safe trade.
- Avian reovirus is rarely identified as the primary disease-causing agent, however, it is frequently recovered in conjunction with other pathogens. Psittacine birds are susceptible to infection with reovirus and exhibit a range of disease manifestations and associated pathology. Infections may be subclinical, or latent and persistent, making detection and control difficult.
- The wide pathogenicity range of reovirus means that forecasting consequences of establishment in Australian psittacines is difficult. However, it appears that Old World psittacine species, which are found in Australia, are more sensitive than New World psittacine species.
- There are suggestions in the literature that stress could contribute to the development of disease signs in psittacines infected with reovirus.
- There is evidence suggesting some strains of psittacine reovirus can be antigenically similar to those found in chickens, while others are antigenically distinct.

Therefore, the department concluded that further risk assessment of avian reovirus was required.

4.10.3 Risk assessment

Entry assessment

The following factors were considered relevant to the estimate of the likelihood of avian reovirus being present in imported household pet and aviary psittacine birds:

- All psittacine species are considered susceptible to infection.
- Avian reovirus has a worldwide distribution.
- Overseas, reovirus is recovered frequently in imported psittacines in quarantine, both clinically diseased and healthy. Studies report a varied detection rate from 4 to 53%.

- The incubation period varies from days to weeks. A variety of clinical signs including hepatitis, enteritis, splenomegaly and pneumonia have been reported in psittacine birds. Clinical signs may vary from nil to severe. Persistently infected birds may be carriers.

Conclusion: based on this information the likelihood of importation of avian reovirus associated with psittacine birds was estimated to be **moderate**.

Exposure assessment

The following factors were considered relevant to the estimate of the likelihood that susceptible species in exposure groups would be exposed to avian reovirus via an infected imported psittacine bird:

- The exact route of transmission remains speculative, although it is thought to be primarily via the faecal-oral route, by contact with contaminated faecal dust. Avian reovirus appears to be highly contagious.
- Avian reovirus has been identified in multiple species including poultry, raptors, pigeons and quail, with cross-species infection having been demonstrated only under experimental conditions. Old World psittacine species, which are found in Australia, appear to be highly susceptible to reovirus infection. Avian reovirus is not zoonotic.
- Avian reovirus is stable in the environment and the presence of organic matter prolongs viability.
- The main exposure groups are considered to be captive and/or wild birds.

Conclusion: based on this information the likelihood of susceptible species in exposure groups being exposed to avian reovirus associated with psittacine birds was estimated to be **moderate**.

Estimation of the likelihood of entry and exposure

Using the matrix as described in Figure 3, the overall likelihood of entry and exposure of avian reovirus in imported psittacine birds was estimated to be **low**.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible species to avian reovirus has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment as described in section 2.3.4.

The most likely outbreak scenario following exposure to avian reovirus was considered to be a **widespread outbreak**, whereby avian reovirus establishes in a directly exposed population (captive birds and/or wild birds) and spreads to other populations of captive and wild birds and becomes endemic in Australian captive and wild birds.

The following factors were considered relevant to the estimate of the likelihood of establishment and/or spread associated with the outbreak scenario:

- Latency/carrier states exist.
- Considering the epidemiology of avian reovirus, it is unlikely that this disease would be self-limiting once introduced to susceptible populations.
- Transmission of the reovirus isolates that affect psittacine birds to other species of birds outside the Psittaciformes Order is unlikely. Cross species infection has only been demonstrated under experimental conditions.

Conclusion: based on these considerations it was estimated that the likelihood of establishment and spread of avian reovirus through Australian captive and wild bird populations was **moderate**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect effects of avian reovirus were estimated. The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of avian reovirus.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Effects are likely to be limited to psittacine birds only.
- Vaccination for reovirus in poultry is practiced overseas, using poultry strains present in, and relevant to the area. The poultry vaccine is unlikely to offer protection to psittacines as poultry and psittacine strains tend to be antigenically different.
- Based on these considerations, the effect of the establishment and/or spread of avian reovirus for this criterion was estimated to be of minor significance at the national level.

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- High mortalities in wild birds are not expected.
- Based on these considerations, the effect of the establishment and/or spread of avian reovirus for this criterion was estimated to be indiscernible at the national level.

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Avian reovirus is not covered by EADRA. Individual owners would have to bear the costs of any strategies or programs they implement.
- Based on these considerations, the effect of the establishment and/or spread of avian reovirus for this criterion was estimated to be of minor significance at the national level.

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- The disease may negatively affect industries involved in breeding and selling pet/aviary psittacine birds (and related feed and equipment), and people buying birds from affected aviaries.
- Movement restrictions or other effects on domestic trade or industry are not expected.
- Based on these considerations, the effect of the establishment and/or spread of avian reovirus for this criterion was estimated to be of minor significance at the national level.

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- Avian reovirus is not OIE listed and it is not likely to cause any international trade effects.
- Based on these considerations, the effect of the establishment and/or spread of avian reovirus for this criterion was estimated to be indiscernible at the national level.

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- It is likely that any negative effects on the environment would be of minor significance at the national level. The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures.
- Effects on communities are likely to be minimal. Control measures are unlikely.
- Based on these considerations, the effect of the establishment and/or spread of avian reovirus for this criterion was estimated to be indiscernible at the national level.

Conclusion: based on the level and magnitude of effects, and using the rules outlined in Table 1, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **low**.

Estimation of the likely consequences

The estimate of the likelihood of establishment and/or spread for the scenario (**moderate**) was combined with the overall effect associated with the outbreak scenario (**low**) using Figure 4 to obtain an estimation of likely consequences of **low**.

4.10.4 Risk estimation

Using Figure 5, the likelihood of entry and exposure (**low**) was combined with the likely consequences of establishment and/or spread (**low**), which resulted in a risk estimation of **very low**.

Therefore, as the unrestricted risk estimate achieves Australia's ALOP, no specific risk management is considered necessary for this agent.

4.10.5 References

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4.11 West Nile virus

4.11.1 Background

West Nile virus (WNV) is an arthropod-borne virus ('arbovirus') belonging to the genus *Flavivirus*, family *Flaviviridae*. It is a re-emerging disease and is considered to be a significant cause of infectious encephalitis in humans worldwide.

WNV is maintained in a transmission cycle between birds and mosquitoes with occasional spill-over causing disease in humans, horses and other vertebrates (OIE 2019i). Prior to the 1990s, it was thought that WNV was non-pathogenic to birds as infections were rarely associated with clinical disease (Komar et al. 2003). However, in 1998 an outbreak in Israel resulted in high mortalities in geese (Anis et al. 2014). In the following year, the spread of WNV to New York resulted in mass avian mortalities especially in American crows and blue jays (Komar et al. 2003). Generally, infection in birds is subclinical, however, certain species are more susceptible and may develop clinical disease or even death (LaDeau, Kilpatrick & Marra 2007).

West Nile fever is an OIE listed disease and is a notifiable disease in Australia.

4.11.2 Technical information

Epidemiology

WNV has an extensive host and vector range which has enabled it to persist in most parts of the world. In nature, WNV is transmitted by mosquitoes and maintained and amplified by avian hosts (Komar et al. 2003). WNV is also capable of infecting humans, horses and other vertebrates as dead-end hosts. In birds, humans, and horses, the clinical severity of infection can vary from subclinical disease to severe neurological complications and death (OIE 2019i).

Hosts/susceptible species

Birds are considered to be the most important maintenance host because they develop sufficiently high viremia to infect mosquitoes (Komar et al. 2003). The type of bird most important for virus maintenance and transmission varies by region, however, in the majority of circumstances Passeriformes play a dominant role in the infection life cycle (Komar et al. 2003). Host susceptibility to infection has been associated with mating and breeding behaviour, body size, migratory behaviour, and co-evolution with WNV or related flaviviruses (Fang & Reisen 2006; Figuerola et al. 2008; Gancz et al. 2004). Species most susceptible to infection belong to the family Corvidae including the American crow, the blue jay, the black billed magpie and the fish crow (Marfin et al. 2001). In Europe and the United States, natural infection with WNV has been reported in 25 bird orders and more than 326 bird species (as reviewed in Gamino & Höfle 2013).

Natural infection in numerous psittacine birds has been recorded in the United States since 1999, including species such as the African grey parrot, budgerigar, cockatiel, cockatoo, conure, kea, lorikeet, lory, macaw, parakeet, parrotlet and rosella (CDC 2016; Palmieri et al. 2011). It has been suggested that there may be widespread susceptibility to WNV in the Psittaciformes order of birds (Palmieri et al. 2011), however, psittacine birds are unlikely to be competent reservoirs for transmission of virus to mosquito vectors. Experimental studies in monk parakeets and budgerigars showed that these species developed the lowest viral titres and the shortest periods of viremia compared to other bird orders such as the Passeriformes and Charadriiformes (Komar et al. 2003).

Poultry species including turkeys and chickens, generally do not show clinical signs of infection with WNV (Senne et al. 2000; Swayne, Beck & Zaki 2000). In fact, chickens are widely used as sentinel birds for WNV surveillance as they undergo seroconversion following infection, rarely transmit infection to in-contact chickens, and usually survive without developing clinical disease (Langevin et al. 2001). Domestic geese, on the other hand, are susceptible to clinical disease and may present with high rates of morbidity and mortality (Meece et al. 2006).

Humans and horses are highly susceptible to WNV infection and are considered to be dead-end hosts as they do not develop sufficient viremia for onward transmission to mosquito vectors. Most (~80%) human infections are subclinical and symptomatic infections vary from self-limiting febrile disease to neuro-invasive life threatening disease (Patnaik, Harmon & Vogt 2006). Less than 1% of human infections progress to severe disease. Severe disease generally occurs in high risk groups including the elderly, immune suppressed individuals, and those with chronic medical conditions (Patnaik, Harmon & Vogt 2006). In horses, rates of symptomatic infection appear to be similar to humans. One paper reported a high case-fatality rate of 30–40% for horses, which is arguably inflated by the propensity to euthanize horses with neurological signs for humane and/or financial reasons (Ward et al. 2006).

WNV has been associated with sporadic disease in small numbers of other species, including squirrels, chipmunks, bats, dogs, cats, white-tailed deer, reindeer, sheep, alpacas, alligators and harbour seals during intense periods of local viral activity (as reviewed in Chancey et al. 2015; OIE 2019h).

Vectors

Over 65 species of mosquitoes have been implicated in WNV transmission (Goddard et al. 2002; Turell et al. 2001). The most important vectors involved in maintaining and amplifying transmission among birds are mosquitoes of the *Culex* genus (Turell et al. 2005). Mosquitoes of the *Culex* genus occupy a worldwide distribution, spanning from tropical to cool temperate regions, enabling widespread establishment of WNV (Samy et al. 2016). Mosquitoes that feed on birds, humans and other mammals are known as bridge vectors and are responsible for outbreaks in human and other animals (Andreadis 2012). Depending on the geographic region, different *Culex* species are responsible for local transmission; *Cx. pipiens* and *Cx. modestus* in Europe, *Cx. univittatus* in Africa, and *Cx. pipiens*, *Cx. tarsalis* and *Cx. quiquefasciatus* in the United States (Andreadis 2012). In Australia, *Cx. annulirostris* is the principal vector of Kunjin virus, a strain of WNV (as reviewed in Prow 2013). Kunjin virus has also been isolated from other *Culex* and *Aedes* species of mosquito in Australia (as reviewed in Prow 2013).

Isolation of WNV has occasionally been reported from other hematophagous arthropods such as bird-feeding ticks (Kolodziejek et al. 2014), however, their importance in the virus transmission cycle is undetermined.

Modes of transmission

The main mode of transmission for birds, humans, horses and other vertebrates is through the bite of an infectious mosquito. For birds, other potential routes of transmission have been documented in laboratory studies and include the consumption of infected mosquitoes or infected dead animals, as well as contact of susceptible birds with cloacal or oral fluids from other infected birds (Komar et al. 2003). Bird-to-bird transmission is reported to occur in

communal roosting birds, however, the exact mechanism by which this occurs and the role it plays in WNV transmission is poorly understood (Janousek, Marra & Kilpatrick 2014).

Incubation period

The incubation period of WNV in birds is highly variable as many species do not develop clinical disease. In experimental infections, susceptible birds develop clinical signs from 3 to 15 days post-infection (Komar et al. 2003).

Persistence of agent

WNV is readily deactivated by commonly used disinfectants and UV light.

Distribution and prevalence

WNV was first isolated in Uganda in 1937 (Williams et al. 1964) and up until the mid-1990s was detected in Egypt, France, India, Israel and South Africa (OIE 2019i). Over the last 2 decades the frequency, severity and geographic distribution of WNV greatly expanded with human cases recorded in southern and eastern Asia, North America, Romania and Russia (as reviewed in Chancey et al. 2015; David & Abraham 2016; Ulbert 2011).

WNV was first reported in the Western Hemisphere in 1999 when it was detected in New York City. From 1999 to 2004, the virus quickly spread across the United States and into Canada, resulting in the largest epidemic of WNV neuro-invasive disease ever reported (Hayes et al. 2005). The virus is now endemic with cases being reported every year.

The epidemiological situation in Latin America has been different to that in the United States and Canada. Despite evidence of WNV activity in mosquitoes as well as in mammals, birds and humans in several Latin American countries, there have been very few reports of clinical disease in humans and animals from countries in this region (as reviewed in Chancey et al. 2015; Elizondo-Quiroga & Elizondo-Quiroga 2013).

In the United Kingdom, serological evidence of exposure to WNV in resident and migratory birds has been found, although attempts to isolate virus have failed (Buckley, Dawson & Gould 2006). Surveys in Germany and Poland also show low seroprevalence in wild birds (Hubálek et al. 2008; Linke et al. 2007).

WNV can be divided into a number of genetic lineages, with lineages 1 and 2 associated with major outbreaks in birds, humans and horses. Lineage 1 consists at least 3 clades, with clade 1a present in Africa, the Americas, Asia, Europe, and the Middle East. A strain of WNV, Kunjin virus, belongs to clade 1b and is present in Australia (May et al. 2011). Lineage 2 is historically endemic to sub-Saharan Africa and Madagascar, with recent spread into Europe and Russia (May et al. 2011). As an RNA virus, WNV undergoes rapid mutation which may result in several novel variants circulating at the same time in endemic regions or the emergence of new variants of greater epidemic potential (May et al. 2011).

The prevalence and epidemiology of WNV across the globe is highly variable. The propensity for WNV to cause outbreaks in birds, humans, horses and other animals depends on many factors including interactions between circulating virus strains, amplifying hosts, vectors, climate, vector and host density, habitat, and circulation of related flaviviruses (Guerrero-Sánchez et al. 2011; Platonov et al. 2014).

Australian status

In Australia, Kunjin virus is endemic in the northern regions of the country (Department of Health 2010) and may be detected in more southerly locations after heavy rains. The mosquito largely responsible for local transmission of Kunjin is *Cx. annulirostris*. Avian hosts responsible for the transmission of Kunjin include water birds belonging to the order Ciconiiformes, which is in contrast to WNV transmission cycles elsewhere (Marshall et al. 1982). Isolated in 1960, Kunjin virus was thought to be of negligible pathogenicity to humans and other vertebrate hosts until 2011, when a large outbreak of Kunjin viral encephalitis occurred in horses in south eastern Australia (Frost et al. 2012). Clinical signs in horses were similar to those described for WNV infection in the United States, with 982 equine cases being reported nationally (Roche et al. 2013). The increase in virulence was attributed to a newly emerged strain of the endemic Kunjin virus and an expansion of mosquito habitats following greater than average rainfall (Roche et al. 2013). Avian disease was not associated with this outbreak (Roche et al. 2013). Presently, distribution of Kunjin virus is monitored through a sentinel chicken program managed by the Department of Health (Department of Health 2019). The presence of Kunjin virus in northern Australia may provide a level of cross-immunity that could prevent the introduction and dissemination of exotic strains of WNV (Jansen, Ritchie & van den Hurk 2013). Kunjin virus is known to infect humans, although only a few reports of non-fatal encephalitis were made during the 2011 epidemic (Roche et al. 2013).

Pathogenesis

WNV isolates from clade 1a are all virulent in mice and isolates of clade 1b, 1c and lineage 2 viruses comprise both virulent and attenuated strains (Hayes et al. 2005). Differences in pathogenicity may be related to nucleotides that code for specific regions in the prM, E, or nonstructural proteins of the virus (Hayes et al. 2005).

Diagnosis

Clinical signs

Clinical signs of WNV infection in both psittacine and non-psittacine birds can range from nil to non-specific signs, progressive neurological disease and death (Palmieri et al. 2011). Non-specific signs include depression, anorexia, dehydration, and ruffled feathers. Neurological signs include seizures, ataxia, paresis, abnormal head and posture/movements (as reviewed in Gamino & Höfle 2013). Vision impairment and blindness is commonly seen in raptors and owls (Pauli et al. 2007). Long-term sequelae such as relapse of neurological signs, feather pulp abnormalities and abnormal moults have been recorded in long-lived birds (Nemeth et al. 2009). The typical duration of disease ranges from a few hours to a few days. In fatal cases, death occurs within 24 hours of the onset of clinical signs in experimental infections and from 2 to 4 days after the onset of clinical signs in naturally infected birds (Komar et al. 2003). Ultimately, the outcome of infection and the development of clinical disease is heavily dependent on viral and host factors. Both natural and experimental infections have shown that strains of virus virulent in one species may not cause disease in another, or produce minimal clinical signs only (Komar et al. 2003).

Pathology

Birds that die rapidly and birds that have low susceptibility to WNV infection may not produce observable macroscopic lesions. Birds with chronic infection are more likely to have non-specific macroscopic changes that include emaciation, dehydration, multi-organ haemorrhages,

petechiae and congestion. Splenomegaly, hepatomegaly, myocardial pallor and pale mottling in the liver, spleen or kidney may also be seen (Wünschmann et al. 2004a, 2005; Wünschmann et al. 2004b).

Microscopic lesions are mainly found in the central nervous system, heart, kidney, spleen and liver. Lymphoplasmacytic and histiocytic infiltrates, cellular degeneration and necrosis, and haemorrhages are the main microscopic findings (Wünschmann et al. 2004a, 2005; Wünschmann et al. 2004b).

Generally, WNV can be detected in the blood 1 day post-infection (Wheeler et al. 2012). In birds it has been demonstrated that the virus can persist in different organs. Viral persistence in naturally infected Passeriformes can last for 4 months and up to 6 months in experimentally infected birds (Wheeler et al. 2012).

Testing

Tests for WNV include virus isolation, molecular testing, serology, immunofluorescent staining, immunohistochemistry, in situ hybridisation, and antigen capture ELISA (AHA 2016; OIE 2019h).

In birds, tissues that are suitable for virus isolation include kidney, brain, heart and intestine. Cell cultures are most commonly used for virus isolation. Confirmation of WNV isolates is achieved by PCR, indirect fluorescent antibody staining of infected cultures or nucleic acid sequencing (AHA 2016; OIE 2019h).

Immunohistochemistry (IHC) of formalin fixed tissues is considered to be a reliable detection method of WNV for avian tissues. Tissues suitable for IHC include brain, heart, kidney, spleen, liver, intestine and lung. Detection in WNV positive birds is improved by the examination of multiple tissues. The specificity of identification (e.g. flavivirus-specific or WNV-specific) depends on the selection of detector antibody (AHA 2016).

Serological testing can be performed with IgM capture ELISA, microtitre viral neutralisation (VN) and plaque reduction neutralisation (PRN) in avian serum. In some serological assays, antibody cross-reactions with related flaviviruses, such as St Louis encephalitis virus or Japanese encephalitis virus, may be encountered. The PRN test is the most specific among WNV serological tests. An IgM capture ELISA may be used to test avian or other species provided that species-specific capture antibody is available (e.g. anti-chicken IgM). The PRN test is applicable to any species, including birds (OIE 2019h).

Treatment

There is no specific treatment for WNV (Nemeth 2012). Where indicated and in high-value birds, supportive care is given.

Control

If an outbreak of exotic WNV is detected in Australia, the national policy is to consider eradication by placing movement controls on the infected animals and vector control at the affected premises. As competent hosts and vectors are widespread in Australia, eradication is considered unlikely to succeed if WNV becomes established in an endemic cycle (AHA 2016).

If the virus is considered established, reducing the exposure of human, mammal and avian species to mosquito vectors is the primary method of disease management e.g. through the use of insect repellents and vector proofing animal enclosures. Although highly effective vaccines are available for equine species, WNV vaccines for use in avian species are not currently available (AHA 2016). In avian veterinary practice, some practitioners vaccinate birds (mainly raptors) using equine vaccines, however, the efficacy of this is unknown (Angenvoort et al. 2014).

The OIE Terrestrial Code Chapter 8.19 contains recommendations for the safe trade in birds other than poultry.

Current biosecurity measures

Clinical disease caused by WNV infection is nationally notifiable in Australia. Routine surveillance of endemic Kunjin virus is conducted through the placement of sentinel chicken flocks in multiple locations around Australia which are managed by the Department of Health.

Live imported pigeons require a declaration of exporting country freedom from WNV or negative serological test results for WNV in pre-export quarantine (AHA 2013). The OIE has recommendations for the safe trade in live birds other than poultry. These recommendations are found in [Chapter 8.19](#) of the OIE Code (OIE 2019i). Risk management includes a requirement for the absence of clinical signs of infection, pre-export quarantine, and pre-export testing of a sample of the birds to show freedom from infection.

4.11.3 Conclusion

- There are strains of WNV that are exotic to Australia and are responsible for high morbidity and mortality in birds and horses.
- In Australia, WNV infection (clinical disease) is a nationally notifiable disease.
- WNV is potentially zoonotic.
- The vectors and hosts required to establish and spread exotic WNV are present in Australia.
- Kunjin virus, which is classified as clade 1b WNV, is endemic to the northern regions of Australia.
- The presence of endemic Kunjin virus may make it difficult for exotic WNV to establish and spread. A viraemic imported parrot would most likely enter Australia in urban areas where Kunjin virus is rarely detected in mosquito populations. Thus, in urban areas there would be little protective immunity in vertebrate fauna and humans (Jansen, Ritchie & van den Hurk 2013).
- Although passerine birds, particularly of the Corvidae family, are most susceptible to WNV infection, psittacine birds are capable of being infected and may not show any clinical signs of infection. While capable of being infected, the evidence available suggests that psittacine birds are dead-end hosts and are unlikely to transmit infection to mosquito vectors.
- WNV epidemiology is highly variable across the world and the consequence of exotic WNV introduction into Australia is difficult to forecast. Consequences can vary from widespread establishment and spread causing a major public health concern to both humans and animal hosts as seen in the United States, to minimal consequence causing no significant disease similar to the situation in the Latin American countries.
- West Nile fever is an OIE listed disease and there are recommendations in the OIE Code on measures for safe trade of birds other than poultry, equidae, geese and ducks.

Therefore, the department concluded that further risk assessment of WNV was not required, however, certification will be required in accordance with the OIE Code for WNV for the importation of psittacines.

The OIE Code recommendations for live birds other than poultry (OIE 2019i) include isolation and testing. As vaccination of birds against West Nile Virus is not permitted in Australia, animals are not permitted to have been previously vaccinated.

4.11.4 References

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5 Risk management

5.1 Introduction to risk management and import requirements for psittacine birds

Risk management aims to reduce the likelihood that importation of a commodity (animal product or live animal) would lead to the entry, establishment and/or spread of a disease agent of biosecurity concern. Biosecurity risk management measures should either be consistent with the OIE Code or the result of a risk assessment.

The 2019 OIE Code states in Article 2.1.5 that:

Risk management is the process of deciding upon and implementing measures to address the risks identified in the risk assessment, whilst at the same time ensuring that negative effects on trade are minimised. The objective is to manage risk appropriately to ensure that a balance is achieved between a country's desire to minimise the likelihood or frequency of disease incursions and their consequences and its desire to import commodities and fulfil its obligations under international trade agreements (OIE 2019d).

Australia has determined that to achieve its ALOP, the unrestricted risk estimate associated with animals and animal products must be at most 'very low'. In the risk review of diseases, technical information on risk factors relevant to the biosecurity risk (encompassing entry, exposure, establishment and/or spread, and consequences) associated with the importation of psittacine birds was reviewed.

Evaluation of the risk factors relevant to each disease enabled conclusions to be drawn regarding whether the associated biosecurity risk could be managed sufficiently to achieve Australia's ALOP.

For the disease agents listed below, and following a risk assessment, this review concluded that risk management is not required to achieve Australia's ALOP:

- avian paraavulavirus 3
- avian metaavulavirus 5
- psittacid herpesvirus 2
- *Salmonella* spp
- reovirus

For some disease agents, the review concluded that risk management is required, and that risk management options are available that would achieve Australia's ALOP. This conclusion was drawn for the following disease agents:

- avian influenza viruses (OIE Code recommendations will be required)
- avian orthoavulavirus 1
- internal and external parasites (other than protozoa)
- parrot bornavirus
- psittacid alpha herpesvirus 1

- psittacine pox virus
- West Nile Virus (OIE Code recommendations will be required)

The measures outlined in this chapter provide the biosecurity policy for the importation of psittacine birds and are the basis for operational conditions for the importation of psittacine birds into Australia. This chapter also includes details of specific requirements for animal identification, transport and certification.

Diseases that have not been identified as requiring risk management to meet Australia's ALOP may still be of biosecurity concern if identified prior to release from post-entry quarantine (for example, a disease that Australia does not have). If a disease not specifically listed by this import risk assessment is detected during the import process, action may be required in order to manage that risk.

5.1.1 Overview of risk management for psittacine bird import categories

Psittacine birds will be subject to different risk management measures based on the import category (household pet or aviary; see Chapter 4). A summary of conditions for the importation of psittacine birds into Australia is in Table 4. More detailed information follows.

Table 4 Summary of conditions for the importation of psittacine birds into Australia

	Household pet psittacine birds	Aviary psittacine birds
Species	Must be on Live Import List	Must be on Live Import List
Ownership	Minimum one (1) year. Evidence required	Nil
Country of export	Birds must be exported from countries approved to export pet birds to Australia (including a residency period in that country).	Birds must be exported from countries approved to export aviary birds to Australia (including a residency period in that country).
Quantity	Maximum of two (2) birds per person	No quantity restriction that can be accommodated in quarantine facilities
Identification	Microchip or permanent leg band	Microchip or permanent leg band
Pre-export quarantine facility approval process	Approved by an Official Veterinarian or Government Approved Veterinarian of the exporting country	Approved by the competent veterinary authority of the exporting country (following a country's approval as an aviary bird approved country by the department). Thereafter, for each consignment: Approved by an Official Veterinarian of the exporting country
Pre-export quarantine duration	35 days	35 days
Pre-export inspection and testing	Avian influenza virus; avian orthoavulavirus 1; internal and external parasites; parrot bornavirus; psittacid alphaherpesvirus 1; psittacine pox virus; West Nile virus	Avian influenza virus; avian orthoavulavirus 1; internal and external parasites; parrot bornavirus; psittacid alphaherpesvirus 1; psittacine pox virus; West Nile virus
Transport to Australia	Must be accompanied by owner and meet transportation requirements	Must meet transportation requirements
Post-entry quarantine length	15 days minimum	15 days minimum
Post-entry quarantine facility	Standard room	BC3 live bird room

	Household pet psittacine birds	Aviary psittacine birds
Post-entry inspection and testing	Avian orthoavulavirus 1; internal and external parasites; parrot bornavirus; psittacid alphaherpesvirus 1; psittacine pox virus	Avian orthoavulavirus 1; internal and external parasites; parrot bornavirus; psittacid alphaherpesvirus 1; psittacine pox virus

5.1.2 Permitted species – List of Specimens Taken to be Suitable for Live Import (Live Import List) and Australian State and Territory requirements

Only psittacine species included on the List of Specimens Taken to be Suitable for Live Import (Live Import List), managed by the department (see Appendix B: Live Import List and CITES) are permitted import into Australia. It is the importer's responsibility to ensure they abide by all approval, permit and other requirements dictated by the Live Import List for the importation of psittacine birds into Australia.

Furthermore, it is the importer's responsibility to ensure they abide by all relevant Australian State or Territory legislation for the importation of psittacine birds into the relevant jurisdiction. Some jurisdictions require importers to apply in writing, and some species are not permitted entry into some jurisdictions (despite inclusion on the Live Import List). Importers must contact the relevant jurisdiction that the bird will be imported into following release from post-entry quarantine, for further information.

5.1.3 Definition of psittacine bird import categories

Psittacine birds may be imported into Australia under one of the following categories. Note that import conditions already exist for pet birds from New Zealand and this import pathway will remain unchanged at this time.

Household pet: Genuine household pet psittacine birds which are not usually housed outside, not in contact with other birds (except those intended for export to Australia), and are not kept for commercial or recreational breeding purposes or exhibition.

The household pet birds must have been in the ownership and possession of the owner for a minimum period of one year immediately preceding commencement of pre-export quarantine. The owner must be immigrating to Australia to take up residence. The birds must not be intended for sale. Importers will be required to provide a statutory declaration and evidence (Box 1) of ownership of the bird and permanent immigration to Australia at the time of import permit application.

Note:

- Provisions are in place to consider circumstances where owners of household pet birds have already immigrated to Australia and left pet birds in their country of origin, to allow for import of these birds for a period of 12 months following the import conditions first being placed on BICON.
- Owners who wish to travel overseas with their pet bird, and then import it back into Australia may do so, negating the requirement for 'The owner(s) must be immigrating to Australia to take up residence'. However, owners must meet all other requirements for importing household pet birds.

Note:

The Biosecurity Act 2015 allows a biosecurity officer to ask questions about goods (s126) and require documents to be produced (s127) as part of their assessment of the level of biosecurity risk. The level of biosecurity risk is different for household pet psittacines compared to aviary psittacines. The department will ask questions and examine documents in order to ensure that a bird is assigned to the correct category and subject to the correct import conditions. Following are examples of the sort of information the department may require. However, this is not prescriptive and the department will consider each prospective import individually, then ask relevant questions and examine relevant documents accordingly.

Box 1 Statutory declaration sample

Statutory declaration to be made:

- The bird(s) identified with microchip or leg band number(s): _____ is(are) my household pet pet(s) and has(have) been in my possession for at least the past year. I am *(delete which does not apply)*
 - Permanently immigrating to Australia
 - an Australian citizen returning to reside in Australia permanently
- and I do not own any other pet birds that will be exported to Australia.

Evidence to be provided:

- Evidence you have acquired and owned your pet for at least the past year prior to commencement of the pre-export quarantine period and that it is a genuine household pet and has not been acquired for the purpose of export (for example, breeder records, purchase receipt, photographs of the animal in the home environment, veterinary records/bills, etc.).
- Evidence that you are permanently immigrating to Australia (including residency visa documentation, or for returning Australian citizens, Australian passport, and evidence such as letter of employment, tenancy agreement, etc.).
- Evidence that you are accompanying your pet bird to Australia (for example, airline ticket).

NOTE: all required evidence may not be available at the time of permit application (for example, airline tickets and residency documentation). However, it must be provided to the department for assessment at least 10 days prior to export.

Aviary: Captive bred psittacine birds that may be kept indoors, outdoors and/or in a common aviary with other birds. This category includes birds held for hobby purposes or exhibition, in zoos, wildlife parks and conservation programs as well as birds resident in breeding centres and private collections. It also includes birds purchased from overseas and intended to be imported for the purpose of being a household pet when in Australia.

5.1.4 Quantity restrictions for psittacine birds

Household pet: Maximum number of **2** birds per adult. One permit will be issued per adult.

- Birds will be required to enter a standard room at the government quarantine facility in Mickleham, Victoria to complete post-entry quarantine.
- For family groups immigrating to Australia with pet birds, a maximum of 2 birds per adult applies, but birds may complete pre-export quarantine at the same premises. Birds may also complete post-entry quarantine in the same room, provided pre-export quarantine was completed at the same premises and depending on capacity.

Aviary: No quantity limit.

- Birds will be required to enter a BC3 live bird room at the government quarantine facility in Mickleham, Victoria. This room can accommodate approximately 500 small psittacine birds (e.g. budgerigars) or 40 large psittacine birds (e.g. macaws), depending on cage sizes.
- Joint consignments managed via a single agent will be permitted. In these cases, the department will deal exclusively with the agent (not individual syndicate members) and will manage the entire consignment as a single epidemiological unit. The health status of any single bird in the consignment may affect the outcome for the entire consignment.

5.1.5 Country of origin requirements for psittacine birds

Approved countries of export for household pet and aviary psittacine birds

The risk assessment and expert consultation processes identified a need to consider the country of export in relation to pre-export quarantine requirements, for both household pet and aviary psittacine birds.

As detailed in Animal Quarantine Policy Memorandum 1999/62 Australia takes into account the following criteria when considering the approval of countries to export animals and their products to Australia:

- the animal health status of the country
- the effectiveness of veterinary services and other relevant certifying authorities
- legislative controls over animal health, including quarantine policies and practices
- the standard of reporting to the OIE of major contagious disease outbreaks
- effectiveness of veterinary laboratory services, including compliance with relevant international standards
- effectiveness of systems for control over certification/documentation of products intended for export to Australia.

If other countries with history of trade with Australia wish to be added to the list of approved countries, a detailed assessment taking into account these criteria would be required to determine if Australia's biosecurity requirements could be met.

Household pet: Must reside in a pet bird approved country. These countries have been assessed by the department as having an effective veterinary health service, surveillance programs for avian diseases, an eradication policy for virulent Newcastle disease and highly pathogenic avian influenza, and sound trade history including compliance with Australian import conditions.

Household pet psittacine birds that do not reside in an approved country can only be exported to Australia after:

- being legally imported into an approved country,
- completing that approved country's import requirements and being free from quarantine restriction, and then
- completing all relevant Australian pre-export quarantine requirements in the approved country.

Aviary: Must reside in an aviary bird approved country and can only be exported to Australia if they complete pre-export quarantine at premises that have been approved by the competent veterinary authority as suitable for these purposes.

Aviary psittacine birds that do not reside in an approved country can only be exported to Australia after:

- 1) being legally imported into an approved country
- 2) completing that approved country's import requirements and being free from quarantine restriction,
- 3) completing all relevant Australian pre-export quarantine requirements in the approved country.

Country approval process

Countries need to apply to the department for assessment for the export of pet and/or aviary psittacine birds. The process for country assessment is for the Chief Veterinary Officer (or equivalent) of a country to prepare and forward a submission that includes details of the country's avian health status, animal health legislation, systems for control over certification of animals and products (particularly as they would relate to exports of live psittacine birds to Australia), veterinary and laboratory services, disease notification, import requirements (particularly for live birds and avian products), disease management and control programs and general veterinary services capacity. The department may request further information or conduct in-country verification activities during the assessment process. For approval, countries will be required to have well developed and resourced veterinary services, and comprehensive systems for identifying and managing avian health issues.

This approach provides a high level of confidence that each bird imported is prepared in accordance with Australia's biosecurity measures.

For further information enquiries may be directed to:

Assistant Secretary
Animal Biosecurity
Department of Agriculture, Water and the Environment
GPO Box 858
CANBERRA ACT 2601
Telephone: 1800 900 090 or (61 3) 8318 6700 (from outside Australia)
Email: animal@agriculture.gov.au

5.1.6 Pre-export and post-entry quarantine requirements

Pre-export and post-entry quarantine periods are required in order to help manage the risk associated with psittacine bird importation. Pre-export and post-entry quarantine allow isolation and separation of birds from other animal populations, reducing the possibility of exposure to disease prior to export and the exposure of Australian animals to disease if carried by imported birds. Psittacine birds are monitored during post-entry quarantine for clinical signs of disease, the presence of exotic parasites, and are tested and/or treated for disease agents of biosecurity concern, including underlying conditions and disease carrier states which may be unmasked by the stress of travel. post-entry quarantine also provides a period for verification measures to occur onshore prior to release from biosecurity control where there are concerns over a bird's health status, documentation, or provenance.

A pre-export quarantine period of 35 days for household pet birds and aviary birds is required on the basis that this is the minimum period of time that will allow the risk management measures recommended by this risk review to be performed.

A minimum post-entry quarantine period of 15 days is required for both household pet birds and aviary birds to facilitate sufficient clinical observation, disease testing and/or treatment based on the risks identified in this review. In order to be as trade facilitatory as possible whilst meeting biosecurity requirements, the post-entry quarantine facility requirements for household birds are lower (a standard room in the department's quarantine facility in the department's quarantine facility at Mickleham, Victoria) than those for aviary birds (a Biosecurity Containment Level 3 live bird room in Mickleham, Victoria).

5.1.7 Diagnostic testing

In addition to the above measures, for certain disease agents diagnostic testing during pre-export and/or post-entry quarantine is also required in order to manage the biosecurity risk associated with the importation of psittacine birds.

5.2 Detailed risk management

The following section details pre-export conditions that apply to psittacine birds under preparation for export to Australia. The export process requires various tasks to be performed by an *Official Veterinarian* of the country of export (OV) (as defined in the OIE Code [glossary](#) (2019c)). Some of these tasks may be performed by a *Government Approved Veterinarian* of the country of export (GAV). A GAV is defined as:

A veterinarian that has been approved/accredited by the government of the exporting country for the preparation of psittacine birds (household and aviary) for export (this includes but is not limited to: scanning for microchips, inspections, collection of samples, treatments, providing directions, etc.). They must prepare paperwork for presentation to the *Official Veterinarian* to give them confidence that the export preparations have been performed in accordance with the import conditions.

Different conditions exist depending on the category of bird(s) being imported.

5.2.1 All birds: General pre-export conditions

- 1) Birds must be individually identified with an implanted ISO-compatible microchip or a permanent leg band containing unique identification details.
- 2) All pre-export testing must be conducted in a laboratory recognised by the competent veterinary authority of the country of export for the purpose of testing for export.
- 3) Any treatments or medications, other than those specified in the disease-specific requirements, that birds receive during pre-export quarantine must be listed on the veterinary health certificate (active ingredient(s), dose, route of administration). The birds must not receive any antimicrobial or antiviral medication during pre-export quarantine without prior written approval from the department.

5.2.2 Household pet psittacine birds: pre-export conditions

- 1) Birds are to enter into a quarantine area suitable for the purposes of pre-export quarantine and approved by the OV or GAV prior to commencement of pre-export quarantine (see Pre-export quarantine commencement declaration). The premises must be under the *control and supervision** of an OV or GAV.

**Control and supervision* means: at minimum, provision of detailed direction to people in contact with the bird(s) regarding quarantine requirements; and oversight of all matters relating to the health, sample collection, treatments and quarantine of the bird(s).

- 2) The address of the premises at which the quarantine area is located must be listed on the veterinary health certificate.
- 3) For at least **35 days** immediately prior to export to Australia, the bird(s) must be kept continuously isolated in the quarantine area.
- 4) An OV or GAV must take samples and provide treatments to meet the disease specific requirements during the pre-export quarantine period. These tasks may also be performed by a registered veterinarian under the direct supervision of an OV or GAV. The person(s) involved in collection of samples and provision of treatments must ensure that they adhere to strict biosecurity procedures to prevent disease transmission to the bird(s).
- 5) Duties are required to be performed by OVs/GAVs, some with certain findings, in order to make the following declarations:
 - a) Ownership enquiry declaration

**This is in addition to the statutory declaration and evidence regarding ownership of the bird and permanent immigration to Australia to be provided by the owner at the time of application for an import permit (see section 5.1.3).*

An OV must perform relevant enquiries in order to make the following declaration:

Box 2 Official Veterinarian declaration sample

Following *due enquiry** I have no reason to doubt that the bird(s) identified with microchip or leg band number(s) _____ has(have) been in the ownership and possession of the owner for a minimum period of one year immediately preceding commencement of pre-export quarantine.

[**Due enquiry* means the certifying veterinarian has viewed a range of evidence such as, but not limited to:

- breeder records, purchase receipts, import permits, veterinary records, declarations, photographs, CITES documents

to satisfy themselves that the condition has been met.

If the certifying veterinarian is not satisfied based on available evidence, commencement of pre-export quarantine must be delayed until the time when sufficient evidence is available.]

- b) Pre-export quarantine commencement declaration

An OV or GAV must perform relevant duties and be satisfied to make the following declaration:

Box 3 Pre-export quarantine commencement declaration sample

I examined the bird(s) at commencement of pre-export quarantine on ...[date]... and found it (all) to be free from any clinical signs of infectious or contagious disease. This included examination of the oral cavity and cloaca.

I inspected the quarantine area at the commencement of pre-export quarantine on ...[date]... and I provided detailed instruction regarding operational requirements to all people that will be in contact with the bird(s) during the quarantine period and have ensured they understood the requirements. I am satisfied that for entirety of the quarantine period, the quarantine area is/will be:

- 1) Dedicated solely to the purpose of quarantine of the bird(s) and suitable for a 35 day quarantine period.
- 2) A mosquito-proof secure unit physically isolated from any other birds and constructed in such a way that there is no possibility of contact between the quarantined bird(s) and other birds or animals, or bird or animal products.
- 3) Run on an all-in-all-out basis i.e. no bird is to leave and no bird is to enter the quarantine area during the quarantine period.
- 4) Thoroughly cleaned and disinfected, or for quarantine areas such as a cage in a room within an owner's residence, new or thoroughly cleaned and disinfected.
- 5) Operated so that all areas containing feed and feeding equipment, bedding materials, or any other equipment which may come into contact with the bird(s) (either within or outside of the quarantine area), are adequately bird-proofed and vermin-proofed and cannot be contacted by other animals (e.g. cats, dogs, etc.) (inside an owner's residence may be suitable if access by birds, vermin and other animals is prevented).
- 6) Operated so that any fresh feed is thoroughly washed to remove any surface contamination in suitable water (as described in 9.) prior to entering the quarantine area.
- 7) Operated so that if equipment is removed from the quarantine area for cleaning (e.g. food and water dishes) only suitable water is used (as described in 9.) and the equipment must be cleaned and dried in an area as described in 5.
- 8) Operated so that absolutely no material that may have had direct or indirect contact with birds is allowed into the quarantine area (e.g. branches, flowers, fruit directly from trees, etc.).
- 9) Operated so that all water supplies for the bird or used to clean feed/equipment contacting the bird(s) are secure against contamination by wild birds (town water supply is suitable).
- 10) Operated so that all persons in contact with the bird(s) during the quarantine period have no contact with other birds; or for personnel performing quarantine inspection or sampling duties, all persons in contact with the bird(s) are required to adhere to biosecurity procedures to prevent disease transmission to the bird(s).
- 11) Operated so that all matters relating to the health, sampling, treatment and quarantine of the birds will be under my control and supervision.

c) Pre-export quarantine conclusion declaration 1

An OV or GAV must perform relevant duties and be satisfied to make the following declaration:

Box 4 Pre-export quarantine conclusion declaration sample

I examined the bird(s) within 72 hours prior to export on ...[date]... and found it (all) to be free from any clinical signs of infectious or contagious disease. This included examination of the oral cavity and cloaca, and examination for external parasites.

Following *due enquiry** I have no reason to doubt that the pre-export quarantine was operated and maintained as per the conditions outlined in the pre-export quarantine commencement declaration.

[**Due enquiry* means the certifying veterinarian has viewed a range of evidence or asked questions regarding quarantine such as, but not limited to:

- the set-up and operation of the quarantine area, visitor contact, contact with other animals e.g. pets, feeding practices, cleaning practices

to satisfy themselves that pre-export quarantine was operated and maintained as required.]

d) Pre-export quarantine conclusion declaration 2

An OV must perform relevant duties and be satisfied to make the following declaration:

**This declaration is only required if the above declaration (c) was made by a GAV.*

Box 5 Pre-export quarantine conclusion declaration 2 sample

I examined the bird(s) within 72 hours prior to export on ...[date]... and found it (all) to be free from any clinical signs of infectious or contagious disease. This included examination of the oral cavity and cloaca, and examination for external parasites.

c) Pre-export quarantine loading declaration

An OV or GAV must perform relevant duties and be satisfied to make the following declaration:

Box 6 Pre-export quarantine loading declaration sample

1) I inspected the container(s) used to transport the bird(s) and found it (them) to be:

- new or thoroughly cleaned and disinfected with an approved disinfectant (e.g. Virkon) prior to use
- mosquito-proof
- adhering to International Air Transport Association (IATA) Live Animals Regulations standards.

2) I confirmed that no feed or feed components were included within the container(s) without separate authorisation from the department.

3) I supervised the loading of the bird(s) into the container(s) at the conclusion of the pre-export quarantine period and prior to transport to the departure point, and sealed the container(s) with an official seal.

Official seal number(s): _____

[In the event of a bird arriving in Australia in an unsealed container, or in a container where the seal has been broken, the bird may not be permitted entry into Australia.]

4) I confirmed that the vehicle used for transporting the bird(s) to the point of departure was cleaned and disinfected prior to loading of the bird(s).

- 5) I confirmed that the bird(s) was(were) to be transported to the point of departure by the most direct practicable route and was not at any time to be in contact with birds not tested to an equivalent health status.

f) Pre-export quarantine departure declaration

An OV or GAV must perform relevant duties and be satisfied to make the following declaration:

Box 7 Pre-export quarantine departure declaration sample

- 1) I confirmed that the bird(s) was(were) scheduled to be consigned to Australia by air, by a route approved by the department. Any planned transshipment has received prior written approval from the department.
- 2) I confirmed that the compartments of the aircraft to be occupied by the bird(s) was cleaned and disinfected with a prescribed disinfectant to my satisfaction prior to the loading of the bird(s).
- 3) I confirmed that the bird(s) was not to be accompanied in transit by other eggs or birds (unless written approval was given by the department).

5.2.3 Aviary psittacine birds: pre-export conditions

Process of approval for eligible countries and approval of pre-export quarantine facilities

Competent veterinary authorities of countries eligible to be assessed as an aviary bird approved country must write to the department to request assessment. They must demonstrate how they will meet the following requirements for approval of pre-export quarantine premises for aviary birds, and how they will verify that pre-export quarantine requirements have been met. The department will assess this information and may request further information or conduct in-country verification activities, such as but not limited to, observation of premises approval.

Following approval as an aviary bird approved country by the department, the competent veterinary authority of the approved country may approve premises as pre-export quarantine facilities. For approval, the competent veterinary authority must ensure that premises comply with the conditions outlined in [Appendix C](#). The department may require re-assessment of a country's approval every 3–5 years or more frequently if required based on performance, compliance, animal health status and other relevant factors, and may review a country's approval at any time.

Birds must complete quarantine in premises that have been approved by the competent veterinary authority of the approved country as suitable for the purposes of pre-export quarantine. Persons interested in having premises approved for pre-export quarantine purposes are advised to contact the relevant competent veterinary authority of the country of intended export.

Once a pre-export quarantine facility has been approved, the facility may be used to consolidate groups of birds for export to Australia. Birds who complete pre-export quarantine as one epidemiological unit will be able to enter the same live bird room to complete post-entry quarantine (depending on capacity) in Australia.

Prospective importers are encouraged to speak with relevant industry/hobby associations, organisations and/or societies regarding consolidated consignments.

Conditions for each consignment

- 1) Birds are to enter approved premises (see Pre-export quarantine commencement declaration below), under the *control and supervision** of an OV or GAV.
**Control and supervision* means: at minimum, provision of detailed direction to people in contact with the bird(s) regarding quarantine requirements; and oversight of all matters relating to the health, sample collection, treatments and quarantine of the birds.
- 2) The address of the approved premises must be listed on the veterinary health certificate.
- 3) For at least **35 days** immediately prior to export to Australia, the birds must be kept continuously isolated at the approved quarantine premises.
- 4) An OV or GAV must take samples and provide treatments to meet the disease-specific requirements during the pre-export quarantine period. These tasks may also be performed by a registered veterinarian under the supervision of an OV or GAV. The person(s) involved in collection of samples and provision of treatments must ensure that they adhere to strict biosecurity procedures to prevent disease transmission to the birds.
- 5) Duties are required to be performed by OVs/GAVs, some with certain findings, in order to make the following declarations:
 - a) Pre-export quarantine commencement declaration

An OV must perform relevant duties and be satisfied to make the following declaration:

Box 8 Pre-export quarantine commencement declaration sample

I examined the birds at commencement of pre-export quarantine on ...[date]... and found all to be free from any clinical signs of infectious or contagious disease. This included examination of the oral cavity and cloaca.

I inspected the quarantine premises at the commencement of pre-export quarantine on ...[date]... The premises have documented procedures covering their operations. I have examined these documented procedures and discussed the premises operations with the personnel that will be in contact with the birds during the quarantine period and have ensured they understand the requirements. I am satisfied that for entirety of the quarantine period, the quarantine area is/will be:

- 1) Dedicated solely to the purpose of the quarantine of the birds for the 35 day period immediately prior to their export to Australia.
- 2) A mosquito-proof secure unit physically isolated from any other birds and constructed in such a way that there is no possibility of contact between the quarantined birds and other birds or animals or bird or animal products.
- 3) Run on an all-in-all-out basis i.e. no bird is to leave and no bird is to enter during the quarantine period.
- 4) Thoroughly cleaned and fumigated (using formaldehyde gas) or disinfected (using Virkon) (Alternative disinfection processes and disinfectants may be used if assessed and approved by the department as providing an equivalent level of biosecurity risk management).
- 5) Operated so that all areas containing feed and feeding equipment, flooring and or bedding materials, or any other equipment which may come into contact with the birds, are adequately bird-proofed and vermin-proofed.
- 6) Constructed so that material used for flooring and enclosures is non-porous, hostile to survival of pathogens and easy to clean and disinfect.

- 7) Operated so that any fresh feed is thoroughly washed to remove any surface contamination in suitable water (as described in 10.) prior to entering the quarantine area.
- 8) Operated so that if equipment is removed from the quarantine area for cleaning (e.g. food and water dishes) only suitable water is used (as described in 10.) and the equipment must be cleaned and dried in an area as described in 5.
- 9) Operated so that absolutely no material that may have had direct or indirect contact with birds is allowed into the quarantine area (e.g. branches, flowers, fruit directly from trees, etc.).
- 10) Constructed and operated so that all water supplies for the birds or used to clean feed/equipment contacting the birds are secure against contamination by wild birds (town water supply is suitable).
- 11) Operated so that all persons in contact with the birds during the quarantine period have no contact with other birds; or for personnel performing quarantine inspection or sampling duties, all persons in contact with the birds are required to adhere to biosecurity procedures to prevent disease transmission to the birds.
- 12) Operated so that all matters relating to the health, sampling, treatment and quarantine of the birds will be under my control and supervision.

b) Pre-export quarantine conclusion declaration

An OV must perform relevant duties and be satisfied to make the following declaration:

Box 9 Pre-export quarantine conclusion declaration sample

I examined the birds within 72 hours prior to export on ...[date]... and found all to be free from any clinical signs of infectious or contagious disease. This included examination of the oral cavity and cloaca, and examination for external parasites.

After *due enquiry** I have no reason to doubt that pre-export quarantine was maintained as per the conditions in the Pre-export quarantine commencement declaration.

[**Due enquiry* means the certifying veterinarian has viewed a range of evidence or asked questions regarding quarantine such as, but not limited to:

- the set-up and operation of the quarantine area, visitor contact, contact with other animals e.g. pets, feeding practices, cleaning practices

to satisfy themselves that pre-export quarantine was operated and maintained as required.]

c) Pre-export quarantine loading declaration

An OV or GAV must perform relevant duties and be satisfied to make the following declaration:

Box 10 Pre-export quarantine loading declaration sample

- 1) I inspected the container(s) used to transport the birds and found it (them) to be:
 - new or thoroughly cleaned and disinfected with an approved disinfectant (e.g. Virkon) prior to use
 - mosquito-proof
 - adhering to International Air Transport Association (IATA) Live Animals Regulations standards.
- 2) I confirmed that no feed or feed components were included within the container(s) without separate authorisation from the department.

- 3) I supervised the loading of the birds into the container(s) at the conclusion of the pre-export quarantine period and prior to transport to the departure point, and sealed the container(s) with an official seal.

Official seal number(s): _____

[In the event of a consignment arriving in Australia in an unsealed container, or in a container where the seal of which has been broken, the consignment may not be permitted entry into Australia.]

- 4) I confirmed that the vehicle used for transporting the birds to the point of departure was cleaned and disinfected prior to loading of the birds.
- 5) I confirmed that the birds were to be transported to the point of departure by the most direct practical route and were not at any time to be in contact with birds not tested to an equivalent health status.

b) Pre-export quarantine departure declaration

An OV or GAV must perform relevant duties and be satisfied to make the following declaration:

Box 11 Pre-export quarantine departure declaration

- 1) I confirmed that the birds were scheduled to be consigned to Australia by air, by a route approved by the department. Any planned transshipment has received prior written approval from the department.
- 2) I confirmed that the compartments of the aircraft to be occupied by the birds were cleaned and disinfected with a prescribed disinfectant to my satisfaction prior to the loading of the birds.
- 3) I confirmed that the birds were not to be accompanied in transit by other eggs or birds (unless written approval was given by the department).

5.2.4 All birds: pre-export disease-specific requirements

Note: All laboratory results must be provided to the department prior to export.

Avian influenza virus

- The bird has not been vaccinated for avian influenza in the past. *[Owner declaration]*
- Agent Identification: Within 14 days prior to export to Australia, oropharyngeal and cloacal swabs are to be collected from each bird. Virus isolation or PCR is to be performed for avian influenza virus with a negative result. **Cloacal swabs should be visibly coated with faecal material and swabs should be transported in appropriate transport media.*
- Declaration: A declaration by the OV or GAV collecting and submitting samples must be provided, attesting to the above.
- The bird has been held in pre-export quarantine for at least 21 days immediately before export and has been isolated from other birds not of equivalent health status.

Avian orthoavulavirus 1

- Agent Identification: At least 28 days after the commencement of pre-export quarantine, oropharyngeal and cloacal swabs are to be collected from each bird. Virus isolation or PCR is to be performed to confirm freedom from avian orthoavulavirus 1. **Cloacal swabs should be visibly coated with faecal material and swabs should be transported in appropriate transport media.*

Declaration: A declaration by the OV or GAV collecting and submitting samples must be provided, attesting to the above.

The bird has been held in pre-export quarantine for at least 35 days immediately before export and has been isolated from other birds not of equivalent health status.

Internal and external parasites (other than protozoa)

- Treatment for internal parasites: Within 14 days prior to export to Australia, each bird was treated with a registered broad spectrum anthelmintic(s) effective against nematodes and cestodes at the manufacturer's recommended dose.
- Treatment for external parasites: Within 14 days prior to export to Australia, each bird was treated with a registered broad spectrum parasiticide(s) effective against mites, ticks and fleas at the manufacturer's recommended dose.
- Declaration: A declaration by the OV or GAV administering the treatments must be provided, attesting to the above and including details of the active ingredients and dosages administered.

Parrot bornavirus

- Agent identification: Within 35 days prior to export to Australia, cloacal swabs are to be collected from each bird and tested for freedom from parrot bornavirus by PCR with negative results. **Cloacal swabs should be visibly coated with faecal material and transported in appropriate transport media.*
- Declaration: A declaration by the OV or GAV collecting and submitting samples must be provided, attesting to the above.

Psittacid alphaherpesvirus 1

- Agent identification: Within 35 days prior to export to Australia, combined oral mucosal and cloacal swabs are to be collected from each bird and tested for freedom from psittacid alphaherpesvirus 1 using PCR with negative results.
- Declaration: A declaration by the OV or GAV collecting and submitting samples must be provided, attesting to the above.

Psittacine pox virus

- The bird has been held in pre-export quarantine for at least 35 days immediately before export and has been isolated from other birds not of equivalent health status.
- Declaration: A declaration by the OV performing the examination of the bird(s) at the commencement of pre-export quarantine and within 3 days of export that either:
 - no lesions suggestive of avian pox were found, or
 - lesions suggestive of avian pox in psittacine birds were present but after due investigation (including testing of the lesion(s)), were shown not to be caused by psittacine pox virus. Evidence of this investigation (including a description of lesion location(s) and test results) must be attached to the certificate.

West Nile virus

- The bird has not been vaccinated for West Nile virus in the past. *[Owner declaration]*
- Agent Identification: Within 27 days prior to export to Australia, serum was drawn from a statistically valid sample of birds and subjected, with negative results, to an IgM capture ELISA or plaque reduction neutralisation (PRN) test.

- Declaration: A declaration by the OV or GAV collecting and submitting samples must be provided, attesting to the above.
- The bird has been held in pre-export quarantine for at least 30 days immediately before export and has been isolated from other birds not of equivalent health status.

5.2.5 On arrival in Australia

- 1) The first point of entry into Australian territory must be Melbourne International Airport.
- 2) The bird(s) will be transported directly from the first point of entry to the department's quarantine facility in Mickleham, Victoria, by the most direct route.
- 3) At least one week prior to arrival, the owner (or agent) must provide the department's quarantine facility with a suitably sized cage(s) with perches of appropriate size, all feed and a feeding plan, and any items for environmental enrichment.

5.2.6 Household pet psittacine birds: post-entry conditions

Birds must enter a standard room at the department's quarantine facility in Mickleham, Victoria, for a **minimum period of 15 days AND until satisfactory laboratory results are received** for all pathogens of concern.

A standard room is an indoor room where birds will be individually housed and isolated from each other. Pet birds from the same household who have completed pre-export quarantine together may be held in a live bird room together, provided the room has capacity.

5.2.7 Aviary psittacine birds: post-entry conditions

Birds must enter a Biosecurity Containment Level 3 (BC3) live bird room at the department's quarantine facility in Mickleham, Victoria, for a minimum period of **15 days AND until satisfactory laboratory results are received** for all pathogens of concern.

The room must be run on an all-in all-out basis. Birds that have completed pre-export quarantine together will be permitted to be housed together. The room can accommodate approximately 500 small psittacine birds (e.g. budgerigars) or 40 large psittacine birds (e.g. macaws), depending on cage sizes.

5.2.8 All birds: post-entry disease-specific requirements

Avian orthoavulavirus 1

Agent Identification: Between 4 and 7 days after the commencement of the post-entry quarantine period, oropharyngeal and cloacal swabs will be collected from each bird and tested for freedom from avian orthoavulavirus 1 by PCR. Results must be negative.

Internal and external parasites (other than protozoa)

If there is evidence of internal or external parasites on arrival or during post-entry quarantine, the department will require that the bird(s) be treated with a registered broad spectrum anthelmintic(s) and/or parasiticide(s).

Owner consent must be provided acknowledging that they understand that birds may develop medical complications, or even die, following parasite treatment, and that they choose to treat rather than re-export the bird.

Parrot bornavirus

Agent identification: Between 4 and 7 days after the commencement of the post-entry quarantine period, cloacal swabs will be collected from each imported bird and tested for parrot bornavirus by PCR. Results must be negative.

Psittacid alphaherpesvirus 1

Agent identification: Between 4 and 7 days after the commencement of the post-entry quarantine period, cloacal swabs will be collected from each imported bird and tested for psittacid alphaherpesvirus 1 by PCR. Results must be negative.

Psittacine pox virus

Examination: On arrival at the quarantine facility, each imported bird will be examined for lesions suggestive of avian pox and lesions must not be present. If lesions are present, the bird(s) must be accompanied by evidence from the pre-export examining Government Approved Veterinarian that the lesions were investigated and found to not be caused by psittacine pox virus.

If lesions are present but not accompanied by the above evidence, the bird will be subject to investigation (including testing of the lesion(s)). If psittacine pox virus is confirmed, the bird will not be released from quarantine and must be re-exported or euthanised.

5.2.9 All birds: additional conditions**Detection of a disease in post-entry quarantine**

- Birds will be monitored during post-entry quarantine for clinical signs of disease and tested and/or treated for disease agents of biosecurity concern.
- Any abnormalities in any bird will be subject to a full veterinary investigation.
- If any investigation or specified test indicates the presence of a pathogen of biosecurity concern in the quarantined bird(s), the bird(s) shall remain in isolation.
- At the discretion of the department and in consultation with the laboratory carrying out the investigations or tests, further investigations and additional testing may be carried out to clarify the situation.
- If a diagnosis cannot be established on the basis of clinical examination and testing, in consultation with the owner, the bird may be held in isolation for further testing or euthanised and submitted for laboratory examination with specific investigations for pathogens of biosecurity concern.
- If the department has biosecurity concerns about any bird in quarantine, this can have implications for the management of other birds in the same consignment. Actions will be taken based on the department's assessment of the individual situation. Actions could include (but are not limited to) extending the quarantine period, testing or treating other birds in the consignment, or in extreme cases, exporting or destroying other birds in the consignment.

Death in post-entry quarantine

- A departmental Veterinary Officer shall require all birds that die during post-entry quarantine before routine testing is complete, to be tested for freedom from the pathogens of biosecurity concern at the owner's expense.
- At the discretion of the department, an entire consignment of imported birds may be destroyed and disposed of as biosecurity waste.

Eggs laid in post-entry quarantine

- Any eggs that are laid during post-entry quarantine will be disposed of as biosecurity waste.

Release

- Each bird will be examined within 24 hours prior to release to ensure it is healthy. It will only be released subject to satisfactory results of the program of testing and treatment required by the department.

5.3 Review of processes**5.3.1 Review of policy**

The Department of Agriculture, Water and the Environment reserves the right to review the import policy when there is reason to believe that a biosecurity risk is present, when there has been a change to scientific understanding of any pathogens of biosecurity concern or at any other time when the department considers that changes may be necessary to ensure biosecurity continues to be managed in accordance with Australia's ALOP.

5.3.2 Equivalence

In accordance with Australia's international obligations under the Application of Sanitary and Phytosanitary Measures Agreement, the principle of equivalence applies to these biosecurity measures. Where the Competent Authority of an exporting country can objectively demonstrate that alternative biosecurity measures to those required by the Department of Agriculture, Water and the Environment would provide an equivalent level of sanitary protection, the Department of Agriculture, Water and the Environment will consider relevant submissions.

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Appendix A: Explanatory comments for identified hazards

This appendix contains explanatory comments for selected hazards identified in the hazard identification list.

Avian adenoviruses

Avian adenoviruses exhibit high genetic diversity and have been described in numerous species of birds worldwide. In psittacine birds, 10 adenoviral isolates have been reported to date, 2 of which have been formally characterised, Psittacine atadenovirus A (named psittacine adenovirus 3) (To et al. 2014) and Psittacine aviadenovirus B (named poicephalus adenovirus 4) (Das et al. 2017). The other reported viruses have been provisionally named psittacine adenovirus 1, psittacine adenovirus 2 and psittacine adenovirus 5 (Ballmann & Vidovszky 2013; Cassmann et al. 2019; Milani et al. 2018). These viruses are able to cross the species barrier and have been reported to infect multiple species of psittacine birds (Ballmann & Vidovszky 2013; Wellehan et al. 2009).

The epidemiology of adenoviruses in parrots is poorly understood, however, based on adenoviruses in poultry and survey studies in parrot populations, it is hypothesised that adenoviral infections in parrots are predominantly subclinical infections and disease may occur under stress or immunosuppression (as review in Phalen et al. 2019; Yang et al. 2019). In Australia, psittacine adenovirus 2 and poicephalus adenovirus 4 have been detected, and psittacine adenovirus 2 is deemed to be endemic in the Greater Sydney area (Das et al. 2017; Phalen et al. 2019). In a survey study in 2015, psittacine adenoviruses were found in aviary birds in Victoria, however, the virus type was not identified (Hulbert et al. 2015). Furthermore, preliminary findings from unpublished research on the prevalence of psittacine adenoviruses in Australia suggest that all of the adenoviruses (or very closely related viruses) isolated overseas, have been found in Australian birds (David Phalen [The University of Sydney] 2019, pers. comm., 16 July).

Considering the epidemiology of adenoviruses, it is highly probable that adenoviruses affecting psittacine birds found overseas are also present in Australia, and the discovery of more novel adenoviruses is likely to continue (as reviewed in Phalen et al. 2019). In light of the information, it was decided that avian adenoviruses would not be retained for further risk review.

Avian polyomavirus

In a survey study in New Zealand, a novel polyomavirus was detected in exhibition budgerigars from 3 breeding aviaries (Baron et al. 2013). Infected birds in the survey study are described to have 2 clinical presentations: birds with feather abnormalities and clinically normal birds without any feather abnormalities. This is no different from infection with the known avian polyomavirus present in Australia and as such the novel polyomavirus would not be retained for risk review.

Avihepadnavirus

Very little is known about avihepadnavirus infection in psittacine birds. A novel avihepadnavirus was identified in Poland from one diseased bird and subsequently from archived livers of psittacine birds (Piasecki et al. 2013). The authors of the study found livers infected with avihepadnavirus were co-infected with either avian polyomavirus or beak and feather disease

virus. The authors concluded that all 3 viruses may be part of an unrecognised disease complex. Moreover, through personal communication the department understands that at the time of writing, research into lorikeet paralysis syndrome found partial sequences of avian hepadnaviruses in lorikeet tissues (David Phalen [The University of Sydney] 2019, pers. comm., 1 March). The implications for this are unknown. In light of the information, it was decided avihepadnaviruses would not be retained for risk review because of the association of the virus with disease syndromes that are present in Australia.

Budgerigar herpesviruses

Detailed information on budgerigar herpesvirus (BHV) is unavailable, however, a review by Gaskin (1989) noted that it may cause decreased egg hatchability. BHV has never been reported in Australia, however, no routine surveillance is undertaken for this virus. A review of exotic diseases of parrots by Snowden (Snowdon 1995) concluded that infection with BHV results in minimal effects. Therefore, on the basis of the above information it was decided that BHV would not be retained for risk review.

Haemosporidia

Haemosporidia consists of an unknown number of protozoan blood parasites including *Haemoproteus* spp., *Leucocytozoon* spp., and *Plasmodium* spp. Representatives of these genera occur in many native Australian species of birds, but rarely occur in native Australian psittacine species (Peacock, Gonçalves da Silva & Clarke 2016). These parasites are highly host adapted and are thought to only cause disease when a new species is introduced into a naïve species (Beadell et al. 2004). Historically, haemosporidia have been reported to sporadically infect wild caught psittacine species imported into North America and Europe, but infection is rare in domestically raised birds (Olias et al. 2011). Considering the worldwide distribution and that many haemosporidia are already in Australian birds, it is highly unlikely that live bird imports would introduce a new species of haemosporidia that would become established and cause adverse effects in Australia's bird populations.

Mycobacterium avium and *M. genavense*

Mycobacteriosis is a well-recognised but uncommon disease of birds and has been diagnosed in nearly every avian order (Lennox 2007). It is a slow progressing, eventually fatal, disease (WHA 2013b). *Mycobacterium avium* consists of 4 subspecies: *M. avium* subsp. *avium*, *M. avium* subsp. *hominissuis*, *M. avium* subsp. *silvaticum*, and *M. avium* subsp. *paratuberculosis*. (OIE 2019b) and is present in Australia (AHA 2018). Infection with *M. avium* is a nationally notifiable disease and in 2018, investigations diagnosed 2 cases in chickens and 2 in other birds (AHA 2018). *M. genavense* has also been recognised as a primary cause disease in psittacine and passerine birds (Schmitz et al. 2018). Several investigations in psittacine birds have found a higher prevalence of infection with *M. genavense* compared to *M. avium* (Palmieri et al. 2013; Schmitz et al. 2018). *M. genavense* is also present in Australia but not nationally notifiable. Control of mycobacteriosis in wild birds is not possible, and low level spontaneous infections are expected to continue to occur in wild and captive birds (WHA 2013b). It may be more of an issue in captive collections where a build-up of the organisms in the environment (due to factors including high density and poor hygiene) may lead to increased prevalence (WHA 2013b). Due to the epidemiology of these organisms and their presence in Australia, it was decided that *M. avium* and *M. genavense* would not be retained for further risk review.

***Pasteurella* spp.**

There are limited reports in the literature of isolating *Pasteurella multocida*, other *Pasteurella* spp. and unclassified or unnamed Pasteurellaceae-like bacteria from both healthy and diseased psittacine birds (Bisgaard et al. 2017). The relevance of these bacteria in causing disease in psittacine birds is poorly understood. However, the most common reason for birds to be infected with *P. multocida* is because of a cat bite, and usually these birds die (David Phalen [The University of Sydney] 2019, pers. comm., 1 March).

Respiratory herpesviruses including Amazon tracheitis virus and psittacine herpesvirus 3 (PsHV-3)

Amazon tracheitis (AT) is a scantily documented upper respiratory and tracheal disease of psittacine birds, of which the causative herpesvirus is poorly characterised but considered to be a possible mutation of Gallid alphaherpesvirus 1 (genus *Iltovirus*, subfamily *Alphaherpesvirinae*), the etiological agent of infectious laryngotracheitis (ILT) in poultry (Gerlach 1994; Guedes et al. 2001; Krautwald-Junghanns, Orosz & Tully 2007). The first reports of the virus appear to be from Germany, in imported birds from multiple quarantine stations in 1978 (Winteroll & Gylstorff 1979).

A herpesvirus antigenically similar to ILT virus was isolated in affected parrots, mostly severe macaws (*Ara severus*), blue-fronted Amazons (*Amazona aestiva*) and yellow-crowned Amazons (*Amazona ochrocephala*). Lazic et al. (2008) reported on respiratory herpesvirus infection in 2 Indian ringneck parakeets (*Psittacula krameri*), imported into the United States from Australia. Lower respiratory tract disease was present and the virus was speculated to be a variant of ILT or AT viruses. The lesions suggested the causative virus was similar, if not the same, as a respiratory herpesviruses previously reported in the United States (Helfer et al. 1980) and Japan (Tsai et al. 1993). Helfer et al. identified a virus in a dead Bourke's parakeet (*Neophema bourkii*) that appeared to have a tropism for the lung and was thought to possibly be an aberrant strain of laryngotracheitis virus. Tsai et al. reported on 14 parakeets (*Psittacula krameri manillensis*) that were found to have respiratory herpesvirus infection with lower respiratory tract tropism.

In recent years psittacine herpesvirus-3 (PsHV-3) (also subfamily *Alphaherpesvirinae*) has been identified as a cause of similar respiratory disease in psittacine birds. PsHV-3 was isolated from captive Bourke's parrots (*Neopsephotus bourkii*) in the United States, and more recently, 2 captive eclectus parrots (*Eclectus roratus*) in Australia (Gabor et al. 2013; Shivaprasad & Phalen 2012). PsHV-3 is currently the only genetically characterised herpesvirus responsible for respiratory disease in psittacine birds (Gabor et al. 2013).

As further research is invested in psittacine diseases, a clearer picture may emerge of the relationship between AT virus, PsHV-3 and other herpesviruses implicated in respiratory disease. However, it was decided that these viruses would not be retained for risk review because the scant amount of literature suggests prevalence of clinical disease is very low, the causative viruses may be related or possibly the same, and PsHV-3 is present in Australia.

Sarcocystis falcatula

The most common cause of sarcocystosis in avian species is infection with *Sarcocystis falcatula* (Godoy et al. 2009). The definitive hosts of *S. falcatula* are the Virginian opossum (*Didelphis virginiana*), the white-eared opossum (*D. albiventris*) and the common opossum (*D. marsupials*)

(Dubey et al. 2000a; Dubey et al. 2001; Dubey et al. 2000b). Psittacine birds can be intermediate hosts for *S. falcatula* following ingestion of sporocysts shed in infected opossum faeces (Clubb & Frenkel 1992; Hillyer et al. 1991a). The geographical range of the parasite is limited by the range of the definitive host, the opossum, thus *S. falcatula* has only been identified in North and South America. The definitive host species are not present in Australia and so a psittacine bird infected with *S. falcatula* would be a dead-end host in this country.

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Appendix B: Live Import List and CITES

The import of live animals into Australia is also regulated by the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act), as administered by the Department of Agriculture, Water and the Environment (DAWE).

Animal specimens considered suitable for live import into Australia are listed on the Live Import List. The list is divided into 2 parts:

- Part 1 is a list of live specimens that do not require an import permit under the EPBC Act.
- Part 2 is a list of live specimens that require an import permit under the EPBC Act. Conditions and restrictions may be imposed on any imports of these specimens.

If a live specimen is not included on the Live Import List then the specimen cannot be imported.

In addition, Australia implements the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) through the EPBC Act. CITES is an international agreement between governments that aims to ensure that the international trade in wildlife does not threaten wild populations of plants and animals.

Australia registers a List of CITES Species under the EPBC Act (List of CITES Species for the Purposes of the Act). This list includes:

- details of the CITES Appendix in which a species is listed
- the date on which the CITES provisions first applied to the species
- any conditions or restrictions that may apply to the specimen.

Both commercial and non-commercial trade of CITES listed animals is regulated. This includes the transfer of live animals between zoos.

Animals are listed under CITES in 1 of the 3 Appendices, depending on the threat of international trade to the survival of the species:

- Appendix I: lists species currently threatened with extinction from international trade
- Appendix II: lists species not currently threatened with extinction but could become so if trade is not regulated
- Appendix III: lists specific populations of species or species threatened only in a specific country.

Further information can be found on the following websites, or by contacting DAWE:

[DAWE website](#) – International wildlife trade

[List of CITES Species for the Purposes of the Act](#)

[Live Import List](#)

Appendix C: Requirements for pre-export quarantine facilities

Aviary bird approved countries

Premises suitable for approval as a pre-export quarantine facility must at minimum comply with the following conditions:

- 1) The quarantine facility must be a separate building(s) which is (are) at least 100 metres away from other bird holdings (i.e. bird holdings that are not under the direct observation and control of the quarantine facility management).
- 2) Each quarantine unit of the quarantine facility must occupy a separate airspace and if multiple units exist, be operationally and physically separated from other units.
- 3) Each unit must contain only birds of the same consignment, with the same health status, and therefore treated as a single epidemiological unit.
- 4) The quarantine facility must be bird, mosquito and vermin proof and sealable so as to allow for fumigation or otherwise designed to allow full disinfection. Additionally, handwashing facilities and hygiene barriers must be installed at all entrances/exits to the quarantine facility and quarantine unit(s).
- 5) Feed, water and bedding must be free from pathogens of biosecurity concern (e.g. treated or from pathogen free sources).
- 6) Storage of litter must be bird and rodent proof and protected against insects. Litter and waste material must be collected regularly, and treated in such a way to avoid spread of disease-causing agents.
- 7) The quarantine period must only start when the last bird is introduced into a quarantine unit.
- 8) Precautions must be taken to prevent cross-contamination between incoming and outgoing consignments.
- 9) Following release of each consignment of birds quarantined, the quarantine unit must be thoroughly cleaned and either fumigated using formaldehyde gas, or disinfected using Virkon. (Alternative disinfection processes and disinfectants may be used if assessed and approved by the department as providing an equivalent level of biosecurity risk management)
- 10) Unauthorised persons must not enter the quarantine facility.
- 11) No outside contacts between personnel entering the facility and potential contaminants (live bird or fomite) must take place which may cause contamination of the quarantine facility.
- 12) The necessary supervision and treatments of birds must be carried out in consultation with and under the control of the Official Veterinarian overseeing the consignment.
- 13) The person in charge of the approved quarantine facility must keep up-to-date records of bird movements including identifying information, significant observations, disease investigations, diagnostic testing, types and dates of treatments, staff movements, and visitor movements.

If the premises are not solely dedicated to pre-export quarantine of birds (e.g. other birds reside permanently on-site such as in zoos or breeding centres), the following conditions must also be met:

- The building or buildings dedicated to the quarantine of birds must be operationally and physically separated from the remainder of the premises.
- Personnel operating in the quarantine area must not have contact with birds outside the quarantine area whilst birds are held under quarantine.
- The premises must keep up-to-date records of bird movements including identifying information, significant observations, disease investigations, diagnostic testing, types and dates of treatments, staff movements, and visitor movements.
- The premises must have a disease surveillance program in place, including quarantine and screening of incoming birds for freedom from pathogens of biosecurity concern (including at a minimum, all pathogens identified in this review as requiring risk management measures). If pathogens of biosecurity control are present in birds held on the premises, a control program for prevention of their spread to birds held under quarantine must be in place.

Glossary

Term or abbreviation	Definition
AA	Approved arrangement. An arrangement for which an approval is in force under paragraph 406(1)a of the Biosecurity Act 2015.
ACT	Australian Capital Territory
ALOP	Appropriate level of protection
AUSVETPLAN	Australian Veterinary Emergency Plan
Australian territory	Australian territory as referenced in the <i>Biosecurity Act 2015</i> refers to Australia, Christmas Island and Cocos (Keeling) Islands.
BA	Biosecurity advice
BICON	Australia's Biosecurity Import Condition System
biosecurity	The prevention of the entry, establishment or spread of unwanted pests and infectious disease agents to protect human, animal or plant health or life, and the environment.
biosecurity import risk analysis (BIRA)	The <i>Biosecurity Act 2015</i> defines a BIRA as an evaluation of the level of biosecurity risk associated with particular goods, or a particular class of goods, that may be imported, or proposed to be imported, into Australian territory, including, if necessary, the identification of conditions that must be met to manage the level of biosecurity risk associated with the goods, or the class of goods, to a level that achieves the ALOP for Australia. The risk analysis process is regulated under legislation.
biosecurity measures	The <i>Biosecurity Act 2015</i> defines biosecurity measures as measures to manage any of the following: biosecurity risk, the risk of contagion of a listed human disease, the risk of listed human diseases entering, emerging, establishing themselves or spreading in Australian territory, and biosecurity emergencies and human biosecurity emergencies.
biosecurity risk	The <i>Biosecurity Act 2015</i> refers to biosecurity risk as the likelihood of a disease or pest entering, establishing or spreading in Australian territory, and the potential for the disease or pest causing harm to human, animal or plant health, the environment, economic or community activities.
EADRA	Emergency animal disease response agreement
ELISA	enzyme-linked immunosorbent assay
endemic	Belonging to, native to, or prevalent in a particular geography, area or environment.
FAO	Food and Agriculture Organization of the United Nations
GAV	Government Approved Veterinarian
goods	The <i>Biosecurity Act 2015</i> defines goods as an animal, a plant (whether moveable or not), a sample or specimen of a disease agent, a pest, mail or any other article, substance or thing (including, but not limited to, any kind of moveable property).
host	An organism that harbours a parasite, mutual partner, or commensal partner, typically providing nourishment and shelter.
import permit	Official document authorising a person to bring or import particular goods into Australian territory in accordance with specified import requirements.
IRA	Import risk analysis
non-regulated risk analysis	Refers to the process for conducting a risk analysis that is not regulated under legislation (<i>Biosecurity import risk analysis guidelines 2016</i>).
NSW	New South Wales

NT	Northern Territory
OIE	World Organisation for Animal Health
OIE Code	OIE Terrestrial Animal Health Code
OIE Manual	OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
OV	Official Veterinarian
pathogen	A biological agent that can cause disease to its host.
PCR	Polymerase chain reaction. For simplicity, in this report PCR is used to refer to both polymerase chain reaction and reverse transcriptase polymerase chain reaction, recognising that the former aims to detect DNA and the latter RNA.
Psittacine	Psittacine birds include all bird species within the order Psittaciformes. Examples include lorries, cockatoos, cockatiels, rosellas, lovebirds and parrots.
Qld	Queensland
qPCR	Real-time PCR. As with PCR, in this report qPCR is used to refer to both real-time polymerase chain reaction and real-time reverse transcriptase polymerase chain reaction, recognising that the former aims to detect DNA and the latter RNA.
quarantine	Official confinement of regulated articles for observation and research or for further inspection, testing or treatment.
restricted risk	Risk estimate with phytosanitary measure(s) applied.
risk analysis	Refers to the technical or scientific process for assessing the level of biosecurity risk associated with the goods, or the class of goods, and if necessary, the identification of conditions that must be met to manage the level of biosecurity risk associated with the goods, or class of goods to a level that achieves the ALOP for Australia.
SA	South Australia
SPS Agreement	WTO Agreement on the Application of Sanitary and Phytosanitary Measures.
stakeholders	Government agencies, individuals, community or industry groups or organisations, in Australia or overseas, including the proponent/applicant for a specific proposal, that have an interest in the policy issues.
surveillance	An official process that collects and analyses information related to animal health.
unrestricted risk	The risk estimate in the absence of risk mitigation measures.
vector	An organism that does not cause disease itself, but which causes infection by conveying pathogens from one host to another.
Vic.	Victoria
WA	Western Australia
WTO	World Trade Organization
