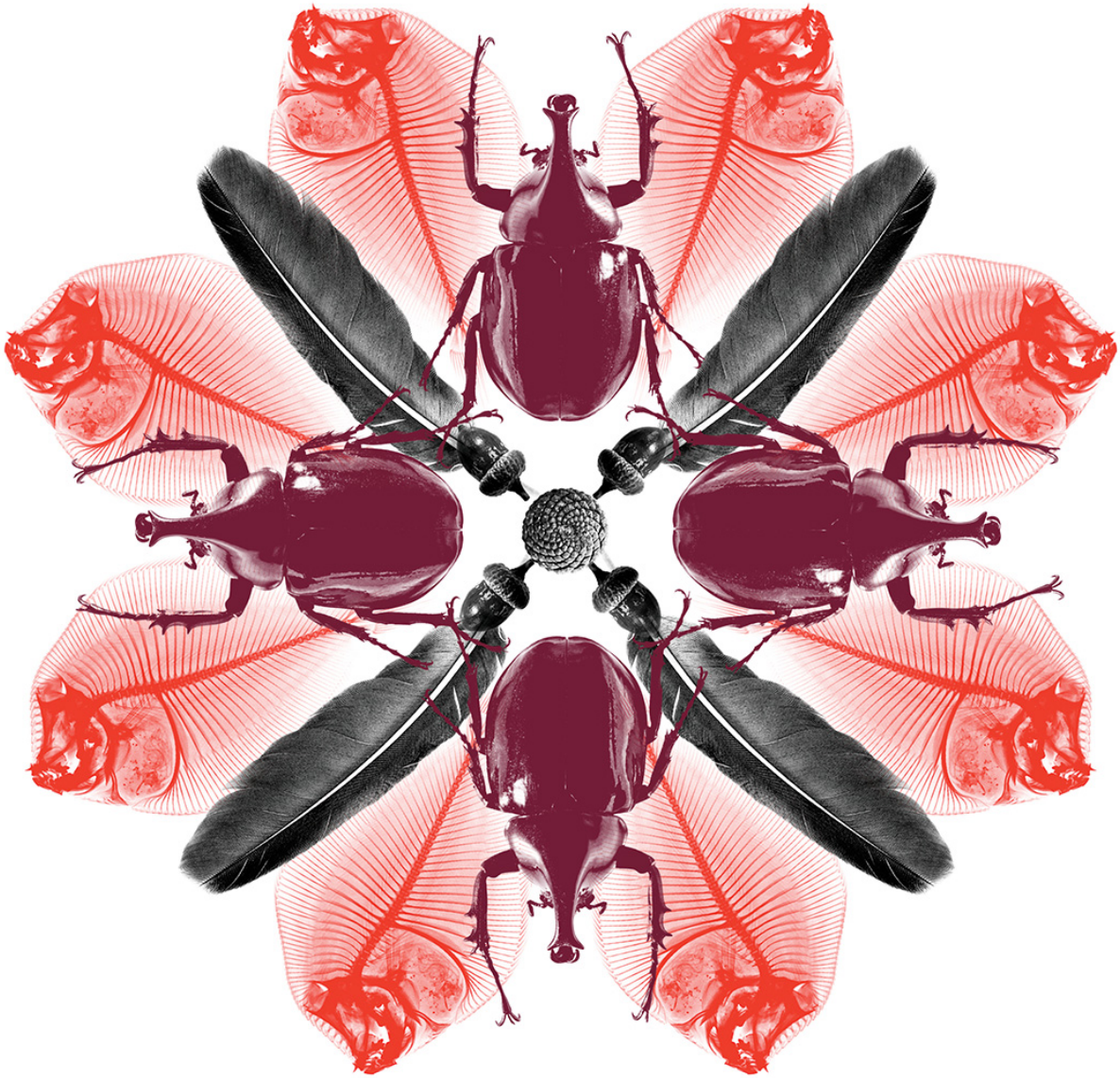
Importation of cooked turkey meat from the United States

Draft review

August 2016



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Submissions

This draft report has been issued to give all interested parties an opportunity to comment and draw attention to any scientific, technical, or other gaps in the data, misinterpretations and errors. Any comments should be submitted to the Australian Department of Agriculture and Water Resources within the comment period stated in the related Biosecurity Advice on the website. The draft report will then be revised as necessary to take account of the comments received and a final report prepared.

Comments on the draft report should be submitted to:

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Glossary

aMPV	Avian metapneumovirus
Appropriate level of protection (ALOP)	The level of protection deemed appropriate by the Member establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within its territory
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
CD	Clostridial dermatitis
CM IRA/chicken meat IRA	Generic Import Risk Analysis Report for Chicken Meat 2008
Code	OIE Terrestrial Animal Health Code 2015
The department	The Australian Government Department of Agriculture and Water Resources
DNA	Deoxyribonucleic acid
DPI	Days post inoculation
ELISA	Enzyme-linked immunosorbent assay
EM	Electron microscopy
Endemic	Belonging to, native to, or prevalent in a particular geography, area or environment
FSANZ	Food Standards Australia New Zealand
Host	An organism that harbours a parasite, mutual partner, or commensal partner, typically providing nourishment and shelter
IBD	Infectious bursal disease
IBDV	Infectious bursal disease virus
Import permit	Official document authorising importation of a commodity in accordance with specified phytosanitary import requirements

ND	Newcastle disease
OIE	World Organisation for Animal Health
Pathogen	A biological agent that can cause disease to its host
Pathway	Any means that allows the entry or spread of a pest
PCR	Polymerase chain reaction
Pest	Any species, strain or biotype of plant, animal, or pathogenic agent injurious to plants or animals
Quarantine	Official confinement of regulated articles for observation and research or for further inspection, testing or treatment
Quarantine pest	A pest of potential economic importance to the area, not yet present there, or present but not widely distributed and being officially controlled
Restricted risk	Risk estimate with sanitary measure(s) applied
RNA	Ribonucleic acid
RT-PCR	Reverse transcription polymerase chain reaction
spp	Species
Stakeholders	Government agencies, individuals, community or industry groups or organizations, whether in Australia or overseas, including the proponent/applicant for a specific proposal, who have an interest in the issues and import conditions
Surveillance	An official process which collects and records data on pest occurrence or absence by surveying, monitoring or other procedures
TCV	Turkey coronavirus
TVH	Turkey viral hepatitis
Unrestricted risk	Unrestricted risk estimates apply in the absence of risk mitigation measures

US	United States
vvIBDV	Very virulent infectious bursal disease virus
WTO	World Trade Organisation

Summary

The Australian Government Department of Agriculture and Water Resources has prepared this draft review of import conditions in response to a request by the United States (US) for market access to Australia for cooked turkey meat.

This draft review takes into account new and relevant peer-reviewed scientific information and advice from international scientific experts.

Australia currently only permits the importation of canned or retorted turkey meat products that meet specific temperature and time requirements during the manufacturing process.

This draft review proposes that the importation of cooked turkey meat to Australia from the US be permitted, subject to biosecurity measures.

This draft review identifies hazards that could be present in turkey meat from the US and therefore requires biosecurity measures to manage these risks to a very low level in order to achieve Australia's appropriate level of protection (ALOP).

This draft review proposes the following: sourcing turkey meat from abattoirs and processing facilities approved by the United States Department of Agriculture, importation of muscle meat only (no whole birds), and cooking the meat to a minimum temperature of 76.6 °C (170 °F) for at least 30 minutes. These measures will reduce any biosecurity risks to a level that is consistent with Australia's ALOP.

This draft review contains details of the risk assessments and the biosecurity measures for the identified hazards. Interested stakeholders are invited to provide comments and submissions to the Department of Agriculture and Water Resources within the 60 day consultation period.

1 Introduction

1.1 Australia's biosecurity framework

Australia's biosecurity framework aims to protect Australia from the entry, establishment and spread of exotic pests and diseases that would threaten Australia's agricultural industries and unique flora and fauna that are relatively free from serious pests and diseases.

The risk analysis process is an important part of developing and reviewing Australia's biosecurity system. It enables the Australian Government to formally assess the risks associated with proposals to import new products into Australia. If the risks are found to exceed Australia's ALOP, risk management measures are recommended to reduce the risks to an acceptable level. If it is not possible to reduce the risks to an acceptable level, no trade will be allowed.

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

The Department of Agriculture and Water Resources science-based risk assessment process is consistent with Australian Government policy as well as Australia's rights and obligations under the World Trade Organization (WTO)'s Agreement on the Application of Sanitary and Phytosanitary Measures. This assessment may take the form of an import risk analysis, a non-regulated review of existing conditions or technical advice.

Further information about Australia's biosecurity framework is provided in the *Import Risk Analysis Handbook 2011* located on the department's website (www.agriculture.gov.au).

The department recognises that there might be new scientific information and technologies, or other measures that may provide an equivalent level of biosecurity protection form the disease agents identified as requiring risk management. Submissions supporting equivalence measures will be considered on a case-by-case basis.

1.2 This draft review

1.2.1 Background

Currently, only canned or retorted turkey meat products that meet specific temperature and time requirements during the manufacturing process are permitted into Australia.

This draft review of the biosecurity risks associated with the importation of cooked turkey meat from the US has been undertaken in response to requests from the United States Department of Agriculture for access to Australian markets for cooked turkey meat.

Many diseases of turkeys also occur in chickens; therefore, this draft review was developed as an extension of the *Generic Import Risk Analysis Report for Chicken Meat* (Biosecurity Australia 2008) (chicken meat IRA). The disease information that was relevant to turkey meat was reviewed and updated to include any advances in scientific knowledge that have occurred since the release of the chicken meat IRA in 2008. Specific diseases that occur in turkeys but not chickens were also reviewed.

1.2.2 Scope

The scope of this draft review is limited to an assessment of the biosecurity risks posed by the importation of cooked turkey meat from the US. The definition of cooking is taken from the US Code of Federal Regulations *Title 9: food and drugs. Part 315 - rendering or other disposal of carcasses and parts passed for cooking* (FDA 2014), which requires cooking of poultry parts 'to a temperature not lower than 170 °F (76.6 °C) for a period of not less than 30 minutes.

In this draft review the definition of turkey meat is limited to muscle tissue from any domestic turkey (*Meleagris gallopava*), blood contained within muscle vasculature and tissues such as nerves, skin and fat that may be considered inseparable from muscle. This draft review does not include an assessment of the risks associated with turkey offal. The turkey must have been slaughtered in an abattoir that meets standards at least equivalent to those contained in the *Australia New Zealand Food Standards Code – Standard 4.2.2 – Primary Production and Processing Standard for Poultry Meat (Australia Only)* (FSANZ 2010).

This draft review is limited to diseases covered in the chicken meat IRA that affect turkeys, as well as diseases that were not considered in the IRA because they infect turkeys but not chickens. It also takes into account relevant changes in scientific knowledge since the release of the chicken meat IRA. Disease agents in the chicken meat IRA that were clearly identified as susceptible to the cooking parameters described above were removed from further assessment at the hazard identification stage, as described in Chapter 3.

1.2.3 Existing conditions

International arrangements

Import conditions exist for canned meat (including turkey meat) and meat based flavours (including turkey meat) from all countries, for human consumption.

The import requirements for these commodities can be found at the department's website (www.agriculture.gov.au).

Australia takes into account the following when considering the approval of and conditions for the export of animals and their products to Australia from any country:

- the animal health status of the country
- the veterinary services and other relevant certifying authorities
- legislative controls over animal health, including biosecurity
- the standard of reporting of major contagious disease outbreaks to the World Organisation for Animal Health (OIE)
- the veterinary laboratory services in the country, including compliance with relevant international standards
- systems in place to maintain the integrity of the certification/documentation of products intended for export to Australia.

Domestic arrangements

The Commonwealth Government is responsible for regulating the movement of animals and animal products into and out of Australia. However, the state and territory governments are responsible for animal health and environmental controls within their individual jurisdiction. 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 biosecurity officers, they may be subject to interstate movement conditions. It is the importer's responsibility to identify and ensure compliance with all requirements.

Trade movements of all types of turkey meat can occur freely between all states and territories in Australia. Restrictions have existed from time to time due to outbreaks of exotic disease such as virulent Newcastle disease (ND) or avian influenza. These outbreaks were managed by stamping out or, in the case of ND, stamping out and vaccination.

1.2.4 Turkey meat industry

There are two types of commercial turkey farms in Australia. The first is the large, commercial contract grower similar to the commercial contract broiler chicken grower. Contract growers produce more than 85% of turkey grown in Australia. The second is the small, low input, integrated farm accounting for the remainder of production (Scott et al. 2009). Some of these producers have small numbers of commercial free range chickens and commercial free range turkeys. Contract growers grow broiler birds for two vertically integrated turkey companies that are based around Bargo and Beresfield in New South Wales.

Available information indicates there is only a very small number of backyard turkeys kept in Australia. Most backyard poultry flocks are chickens that are maintained for table egg production and turkeys are not usually farmed for this purpose. The large size of mature turkeys makes them less suitable for backyard production. The number of breeders offering turkeys to the backyard market is relatively small when compared to other poultry species [www.backyardpoultry.com/directory].

1.2.5 Next Steps

This draft review is released for 60 days public consultation to give stakeholders the opportunity to provide technical comment. Stakeholder submissions will be considered when finalising the draft review.

The final review will be published on the department's website along with a notice advising stakeholders of the release. The department will also notify the proposer, the registered stakeholders and the WTO Secretariat about the release of the final report. Publication of the final report represents the end of the process. The conditions recommended in the final report will be the basis of any imports permitted.

References

Biosecurity Australia 2008, *Generic import risk analysis report for chicken meat: final report. Part C - detailed assessments*, Biosecurity Australia, Canberra, available at http://agriculture.gov.au/biosecurity/risk-analysis/animal/chicken-meat.

FDA 2014, *Title 9: food and drugs. Part 315 - rendering or other disposal of carcasses and parts passed for cooking*, Code of Federal Regulations, Food and Drug Administration, available at

https://www.gpo.gov/fdsys/pkg/CFR-2016-title9-vol2/pdf/CFR-2016-title9-vol2-part315.pdf (pdf 88kb).

FSANZ 2010, 'Standard 4.2.2: primary production and processing standard for poultry meat (Australia only)', *Australia New Zealand Food Standards Code*, Food Standards Australia New Zealand, Canberra, available at https://www.legislation.gov.au/Details/F2012L00292.

Scott, P, Turner, A, Bibby, S & Chamings, A 2009, *Structure and Dynamics of Australia's Commercial Poultry and Ratite Industries*, DAFF, Victoria, Australia, available at http://www.agriculture.gov.au/animal-plant-health/animal/livestock_movement_in_australia_and_emergency_disease_preparedness/structure_and_dynamics_of_australias_commercial_poultry_and_ratite_industries.

2 Method

The World Organisation for Animal Health (OIE), in its *Terrestrial Animal Health Code* (OIE 2014), describes the components of risk analysis in chapter 2.1. as:

- 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 conducted as an ongoing process, and includes both formal and informal consultation with stakeholders.

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

- the Generic Import Risk Analysis Report for Chicken Meat (Biosecurity Australia 2008)
- the *Terrestrial Animal Health Code* 2014 (OIE 2014)
- a review of relevant scientific literature
- expert opinion.

The department considered that the comprehensive chicken meat IRA was still relevant and applicable, and provided a strong framework for a review of the biosecurity risks for the importation of cooked turkey meat from the US. Assessments made in the chicken meat IRA which was completed as a semi quantitative (as a numerical estimate) assessment, were accepted in the draft review. Where an agent was not covered or new information that may affect the final outcome of an assessment was available, the department applied the qualitative (in words) assessment method described in this review.

The chicken meat IRA was completed to cover raw product from any country. The draft review for the importation of cooked turkey meat from the US considered only cooked turkey product from the US.

2.1 Review of hazard identification

Hazard identification is described in the Code (Article 2.1.2) as a classification step that is undertaken to identify potential hazards that may be associated with the importation of a commodity (OIE 2014).

In accordance with the Code, a disease agent was considered to be a potential hazard relevant to the importation of cooked turkey meat if it was assessed to be:

- a disease of turkeys
- OIE-listed, emerging or capable of producing adverse consequences.

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

- it was not present in Australia, or present in Australia and a notifiable disease or subject to official control or eradication

- it was present in the country of export (the US).

However, as this draft review is for the hazards associated with cooked turkey meat, disease agents that were identified in the chicken meat IRA as being susceptible to inactivation by cooking (definition in Section 1.2.2 of this document) were not retained for risk assessment.

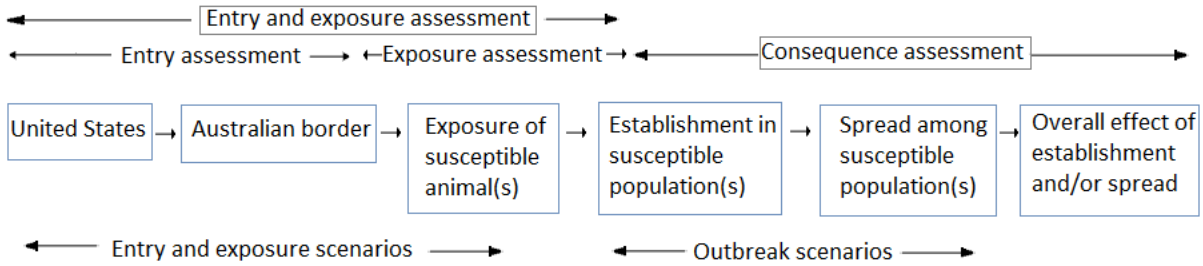
2.2 Risk assessment

Details of the risk assessment process relevant to animals and animal products are provided in Chapter 2.1 of the Code (OIE 2014).

In accordance with the Code, the entry assessment describes the probability of the entry of each of the potential hazards in an importing country and exposure assessment consists of describing the biological pathways necessary for exposure of animals and estimating 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 unrestricted risk estimate is the combination of the likelihood of entry, exposure, establishment and/or spread, and the overall effect of establishment and/or spread.

Steps in determining the unrestricted risk estimate are illustrated diagrammatically in Figure 1.

Figure 1. Components of the unrestricted risk estimate



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

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.

Evaluating and reporting likelihood

For those hazards retained for risk assessment, the assessment was conducted using a qualitative approach based on the same qualitative descriptive definitions as described in the chicken meat IRA.

Entry assessment

The entry assessment considered a single entry scenario defined as the period from slaughter, cooking and export up to arrival in Australia. A number of factors were taken into account in determining the likelihood of a disease agent entering Australia in cooked turkey meat such as:

- prevalence of the hazard in US turkeys
- ante-mortem inspection
- post-mortem inspection

- tissue distribution of disease agent
- cross contamination at slaughter
- the effect of cooking including
    - inactivation temperature (and duration required) for the disease agent
    - the effect of storage and transport.

For each disease agent, a qualitative likelihood was then assigned to describe the likelihood of the disease agent being present in the imported cooked turkey meat products cleared at the Australian border. The final outcome of the entry assessment was the likelihood of entry of a potential hazard into Australia.

Exposure assessment

The exposure assessment describes the process that was used to estimate the likelihood that a susceptible bird in Australia will be exposed to the cooked turkey meat contaminated with a disease agent. 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.

Exposure groups

The term exposure group categorises a group of animals that may be susceptible to one or more of the potential hazards/pathogens considered in risk assessments. The chicken meat IRA identified the four most likely exposure groups to imported poultry meat:

- wild birds
- low biosecurity poultry—backyard poultry and free-range commercial poultry including ratites
- medium biosecurity poultry—non-genetic stock commercial poultry
- non-avian species, where appropriate.

The chicken meat IRA analysed the sequence of steps for imported infected chicken meat to cause infection in susceptible animals (the exposure pathways). The exposure pathways for imported turkey meat are similar. As only cooked products would be imported, it is likely that there would be less on-shore processing than what was assumed in the chicken meat IRA. Therefore most imported product would move from retailer/distributor direct to household consumers or to food service. Product not consumed would be either dumped, where it may be safe or exposed to wild birds or to non-avian species or, more likely in product sold directly to households, it could be exposed to backyard (low biosecurity) poultry. There are restrictions on feeding poultry meat or the by-products of poultry processing to ruminants, however, this material may be fed to birds or poultry.

Given there will probably be minimal on-shore processing, the likelihood of waste product from processing being made into rendered product, and hence into poultry feed, will be lower than assumed in the chicken meat IRA.

In addition to the distribution variables, summarised above and described in detail in the chicken meat IRA, there are a number of exposure group dependent variables

- the likelihood that birds/animals in each exposure group will ingest turkey meat material should they be exposed to it
- the likelihood that feed containing rendered turkey meat may be fed to birds/animals in each exposure group

and pathogen dependent variables, including:

- the hardiness of the pathogen and the likelihood it will remain viable after exposure in the environment over the period before it is exposed to the susceptible animals
- the likelihood that an infective dose is consumed.

These variables are discussed in detail in the chicken meat IRA.

For each agent, the final outcome of the exposure assessment was an estimate of the likelihood that susceptible birds in each exposure group would be exposed to the disease agent via the contaminated imported product (i.e. the likelihood of exposure).

Estimation of likelihood of entry and exposure

The likelihood of entry and exposure for each exposure group was estimated by combining the likelihood of entry and the corresponding likelihood of exposure using the matrix as described in Table 1.

Table 1. Matrix for combining qualitative likelihoods

Likelihood of entry	Likelihood of exposure: *Negligible*	*Extremely low*	*Very Low*	*Low*	*Moderate*	*High*
High	Negligible	Extremely low	Very low	Low	Moderate	High
Moderate	Negligible	Extremely low	Very low	Low	Low	Moderate
Low	Negligible	Extremely low	Very low	Very low	Low	Low
Very low	Negligible	Extremely low	Extremely low	Very low	Very low	Very low
Extremely low	Negligible	Negligible	Extremely low	Extremely low	Extremely low	Extremely low
Negligible	Negligible	Negligible	Negligible	Negligible	Negligible	Negligible

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

Once exposure of a susceptible population has occurred, a number of possible outbreak scenarios could follow, representing a continuum ranging from no spread to widespread establishment. The chicken meat IRA grouped all likely outbreak scenarios into four categories:

- the disease agent does not establish or is not recognised within the directly exposed population
- the disease agent establishes within the directly exposed population only, is identified and is eradicated
- the disease agent establishes within the directly exposed population, spreads to other populations, including other exposure groups if applicable, but is eradicated
- the disease agent establishes within the directly exposed population and spreads to other populations.

For each exposure group, all categories of outbreak scenarios were evaluated for plausibility, based on the epidemiology of each disease agent. 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 disease chapter).

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

When estimating the effects associated with the outbreak scenario, qualitative descriptors were used as described in the chicken meat IRA.

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

The overall effects of establishment and/or spread were addressed in terms of direct and indirect effects on the life and health of susceptible animals on a national scale, including adverse human health, environmental and socioeconomic effects. Impacts on human life and health are the responsibility of the Australian Government Department of Health with Food Standards Australia New Zealand (FSANZ). The department consults with these agencies on assessments for zoonotic agents.

Direct effects:

- life or health (including production effects) of susceptible animals, including public health consequences
- the living environment, including life and health of wildlife, and any effects on the non-living environment.

Indirect effects*:*

- new or modified eradication, control, monitoring or surveillance and compensation strategies or programs
- domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries
- international trade, including loss of markets, meeting new technical requirements to enter or maintain markets and changes in international consumer demand
- the environment, including biodiversity, endangered species and the integrity of ecosystems
- communities, including reduced tourism, reduced rural and regional economic viability and loss of social amenity, and any ‘side effects’ of control measures.

An effect was not assessed more than once and direct effects were considered separately from indirect effects.

The overall effect of establishment and/or spread associated with the outbreak scenario took into account the geographic 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 geographic level and magnitude of effects, the overall effect of establishment and/or spread was determined using the rules described in Table 2.

Table 2. Rules for determining the overall effect of establishment and/or spread

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.
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.

Consequence assessment

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 Table 3.

Table 3. Likely consequences: a combination of the likelihood and overall effect of establishment and/or spread

Likelihood of establishment and/or spread	*Negligible*	*Very low*	*Low*	*Moderate*	*High*	*Extreme*
High	Negligible	Very low	Low	Moderate	High	Extreme
Moderate	Negligible	Very low	Low	Moderate	High	Extreme
Low	Negligible	Negligible	Very low	Low	Moderate	High
Very low	Negligible	Negligible	Negligible	Very low	Low	Moderate
Extremely low	Negligible	Negligible	Negligible	Negligible	Very low	Low
Negligible	Negligible	Negligible	Negligible	Negligible	Negligible	Very low

Overall effect of establishment and spread

2.3 Risk estimation and evaluation

Risk estimation is the integration of likelihood of entry and exposure, and likely consequences of a hazard introduced by the importation of cooked turkey meat from the US into Australia.

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.

Combining the likelihood of entry and exposure and likely consequences was undertaken using the rules shown in the risk estimation matrix in Table 4.

Table 4. Risk estimation matrix

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

Risk evaluation is described in the OIE Code as the process of comparing the estimated risk with a country's ALOP.

A risk estimation that was either 'very low' or 'negligible' was considered sufficient to achieve Australia's ALOP. This provided a benchmark for evaluating risk and determining whether risk management was required.

The use of a benchmark for evaluating risks for each disease agent is illustrated in the process outlined below:

- 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.

This was considered the final output of the risk assessment.

2.4 Risk management

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

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 being to meet Australia's ALOP by reducing the restricted risk to 'very low' or 'negligible'.

Risk management options considered in this report aim to reduce the likelihood that cooked turkey meat from the US would lead to the entry, exposure, and establishment and/or spread of disease agents of quarantine concern in Australia. These may be imposed pre-border and aim to reduce the likelihood of hazards entering Australia in cooked turkey from the US, or post-arrival aiming to prevent the exposure and/or establishment and spread of the hazard in susceptible local populations.

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

2.5 Risk communication

Risk communication is defined in the Code 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 2014).

In conducting import risk analyses and draft reviews, the department consults with the Australian Government Department of Health to ensure that public health considerations are included 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 draft reviews to enable stakeholder assessment and feedback on draft conclusions and recommendations about Australia's animal biosecurity measures.

References

Biosecurity Australia 2008, *Generic import risk analysis report for chicken meat: final report. Part C - detailed assessments*, Biosecurity Australia, Canberra, available at http://agriculture.gov.au/biosecurity/risk-analysis/animal/chicken-meat.

OIE 2014, *Terrestrial Animal Health Code 2014*, World Organisation for Animal Health (OIE), available at http://www.oie.int/international-standard-setting/terrestrial-code/.

3 Hazard identification

The list of diseases (hazards) of potential biosecurity concern was compiled from:

- diseases identified in the chicken meat IRA and *Conditions for the importation from approved countries of fertile eggs (domestic turkey)(Biosecurity Australia 2005, 2008)*
- other diseases identified in the literature as occurring in turkeys.

The method of hazard identification and refinement is described in Section 2.1. The hazard identification decision making process is shown in Figure 2. The preliminary list of diseases/disease agents is shown in Table 5. This table summarises the results of the hazard refinement process, including the reason for removal or retention of each identified hazard.

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 included in the hazard refinement process.

The initial hazard list was taken from the chicken meat IRA. It was updated to include agents that are specific to turkeys and considered relevant to this draft review following a search for any hazards that have emerged since the release of the chicken meat IRA.

Hazards that were assessed in the chicken meat IRA as having an unrestricted risk below Australia's ALOP were not further assessed in this draft review.

Similarly, hazards that were assessed in the chicken meat IRA as being susceptible to the cooking parameters as described in the scope (Section 1.2.2), were not further assessed in this draft review.

An exception to the two statements above was when the department determined there was evidence of significantly different epidemiology or adverse effects of an agent between chickens and turkeys. These hazards were reassessed as primary pathogens of turkeys.

The diseases retained after hazard identification and refinement in Table 5 are listed at the end of this chapter.

Figure 2. Hazard identification and refinement

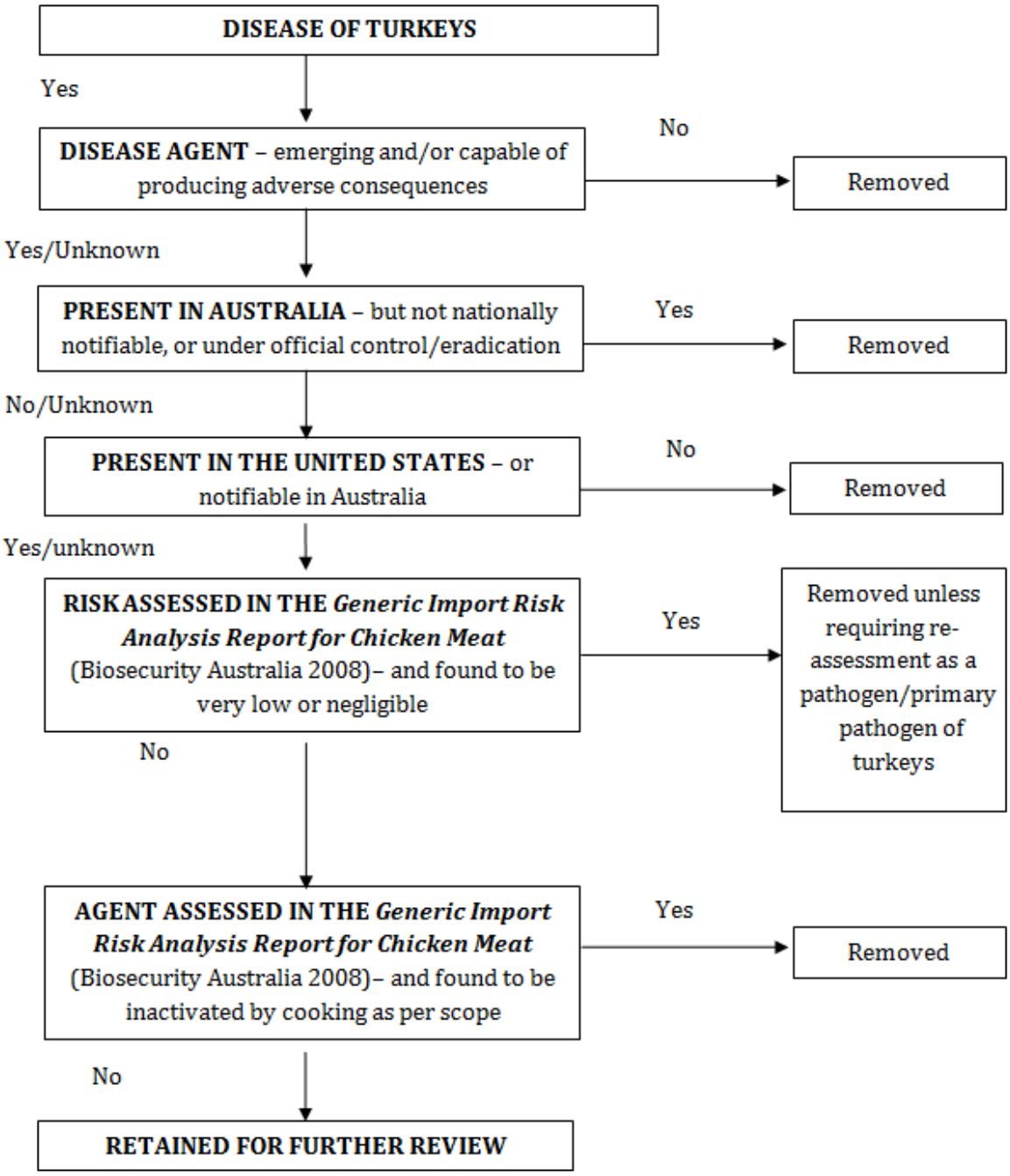


Table 5. Hazard identification

Disease (disease agent)	Emerging and/or capable of producing adverse consequences	Present in Australia	Present in the US	Assessed in CM IRA as either not requiring risk assessment or not requiring risk management	Agent assessed in CM IRA and found to be inactivated by cooking as defined in scope	Retained for risk review
Adenovirus Group 1	Yes	Yes	No	Yes	No	No
Adenovirus Group 2 (Haemorrhagic enteritis virus)	Yes	Yes	Yes	Yes	No	No
Astrovirus	Unknown	Unknown	Yes	No	No	Yes – is present as an agent in multicausal enteric syndromes
Avian encephalomyelitis virus	Yes	Yes	Yes	Yes	No	No
Avian leucosis virus	Yes	Yes	Yes	Yes	No	No
Avian metapneumovirus	Yes	No	Yes	Yes	No	Yes – requires reassessment as a primary pathogen of turkeys
Avian nephritis virus	Yes	Yes	Yes	Yes	No	No
Avian Paramyxovirus 2	Yes	No	Yes	Yes	No	No
Avian Paramyxovirus 3	Yes	No	Yes	Yes	No	No

Disease (disease agent)	Emerging and/or capable of producing adverse consequences	Present in Australia	Present in the US	Assessed in CM IRA as either not requiring risk assessment or not requiring risk management	Agent assessed in CM IRA and found to be inactivated by cooking as defined in scope	Retained for risk review
Avian reovirus (emerging strain in US turkeys)	Yes	No	Yes	No	No	Yes – emerging strains in the US appear to be more pathogenic than those in Australia
Bordatellosis (Turkey coryza)	Yes	Yes	Yes	No	No	No
Brachyspira spp	Yes	Yes	Yes	No	No	No
Campylobacter jejuni	Yes	Yes	Yes	Yes	No	No
Chlamydophila psittaci	Yes	Yes	Yes	Yes	No	No
Clostridium colinum (Ulcerative enteritis)	Yes	Yes	Yes	No	No	No
Clostridial gangrenous dermatitis	Yes	Some strains	Yes	No	No	Yes – identified as an emerging problem in the US
Eastern equine encephalitis/Western equine encephalitis viruses	Yes	No	Yes	Yes	No	No

Disease (disease agent)	Emerging and/or capable of producing adverse consequences	Present in Australia	Present in the US	Assessed in CM IRA as either not requiring risk assessment or not requiring risk management	Agent assessed in CM IRA and found to be inactivated by cooking as defined in scope	Retained for risk review
Enterohaemorrhagic Escherichia coli (EHEC)	No	Yes	Yes	Yes	No	No
Enteroviruses	Yes	Yes	Yes	No	No	No
Erysipelas rhusiopathae	No	Yes	Yes	No	No	No
External parasites	Yes	Yes	Yes	Yes	No	No
Fowl pox	Yes	Yes	Yes	Yes	No	No
Highly pathogenic avian influenza virus	Yes	No	Occasional outbreaks in some states	No	Yes	No
Infectious bursal disease virus serotype 1 (very virulent and variant strains)	Yes	No	Yes	No	No	Yes – assessed as a primary pathogen of chickens, requires reassessment as a pathogen of turkeys
Infectious bursal disease virus serotype 2	No	Yes	Yes	Yes	No	No
Internal parasites (including protozoa)	Yes	Yes	Yes	Yes	No	No

Disease (disease agent)	Emerging and/or capable of producing adverse consequences	Present in Australia	Present in the US	Assessed in CM IRA as either not requiring risk assessment or not requiring risk management	Agent assessed in CM IRA and found to be inactivated by cooking as defined in scope	Retained for risk review
Israeli turkey meningitis virus	Yes	No	No	No	No	No
Japanese Encephalitis virus	Yes	Yes	Yes	Yes	Yes	No
Low pathogenicity avian influenza virus	Yes	No	Occasional outbreaks in some states	No	Yes	No
Lymphoproliferative disease virus	No	No	Yes	No	No	No – limited data on agent and infection is not recognised in commercial flocks
Mycobacterium avium	Yes	Yes	Yes	Yes	No	No
Mycoplasma gallisepticum	Yes	Yes	Yes	Yes	No	No
Mycoplasma iowae	Yes	No	Yes	Yes	No	Yes – requires reassessment as a primary pathogen of turkeys
Mycoplasma meleagridis	Yes	Yes	Yes	Yes	No	No
Mycoplasma synoviae	Yes	Yes	Yes	Yes	No	No

Disease (disease agent)	Emerging and/or capable of producing adverse consequences	Present in Australia	Present in the US	Assessed in CM IRA as either not requiring risk assessment or not requiring risk management	Agent assessed in CM IRA and found to be inactivated by cooking as defined in scope	Retained for risk review
Newcastle disease virus	Yes	Yes – ongoing vaccination program in place	No	No	Yes	No
Ornithobacterium rhinotracheale	Yes	Yes	Yes	Yes	No	No
Pasteurella multocida	Yes	Yes	Yes	Yes	No	No
Reticuloendotheliosis virus	Yes	Yes	Yes	Yes	No	No
Riemerella anatipestifer	Yes	Yes	Yes	Yes	No	No
Rotavirus	Yes	Unknown	Yes	No	No	Yes – is present as an agent in multicausal enteric syndromes
Salmonella arizonae	Yes	Yes	Yes	No	Yes	No – although a primary pathogen of turkeys the chicken meat IRA found it to be inactivated by heat within the cooking parameters under the scope

Disease (disease agent)	Emerging and/or capable of producing adverse consequences	Present in Australia	Present in the US	Assessed in CM IRA as either not requiring risk assessment or not requiring risk management	Agent assessed in CM IRA and found to be inactivated by cooking as defined in scope	Retained for risk review
Salmonella enteritidis	Yes	No	Yes	No	Yes	No
Salmonella gallinarum	Yes	No	No	No	Yes	No
Salmonella pullorum	Yes	No	No	No	Yes	No
Salmonella typhimurium, antibiotic resistant strains	Yes	No	Unknown	No	Yes	No
Turkey coronavirus	No	No	Yes	Yes	No	Yes – requires reassessment as a primary pathogen of turkeys
Turkey torovirus	No	No	Yes	No	No	No – no recent evidence (<10 years) to support this agent being a pathological agent of interest for this draft review
Turkey viral hepatitis	Yes	No	Yes	No	No	Yes
West Nile virus	Yes	Yes	Yes	Yes	No	No

The following diseases were retained for risk assessment on the basis of the information provided in Table 5:

- astrovirus (assessed in multicausal enteric syndromes)
- avian metapneumovirus
- avian reovirus
- clostridial gangrenous dermatitis
- infectious bursal disease virus (very virulent and variant strains)
- *Mycoplasma iowae*
- rotavirus (assessed in multicausal enteric syndromes)
- turkey coronavirus
- turkey viral hepatitis.

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4 Risk reviews

4.1 Avian metapneumovirus

4.1.1 Background

Avian metapneumovirus (aMPV) is a significant respiratory pathogen in turkey and chicken flocks, causing serious economic losses in birds of all ages. Pheasants, guinea fowl and ducks may be infected with aMPV but don't develop disease (Gough et al. 1988; Shin et al. 2002b). In turkeys, initial infection of the upper respiratory tract with aMPV is frequently complicated by secondary bacterial infections and in hens it can cause substantial reductions in egg production. In turkeys aMPV causes a disease known as turkey rhinotracheitis (TRT) while in chickens it produces mild respiratory symptoms and sometimes a swollen head syndrome in meat birds and breeders (Cook 2000b; Jones 1996).

The disease caused by aMPV was first identified in the late 1970s in South Africa (Cook, Kinloch & Ellis 1993), followed by the UK in the mid 1980s where the causal agent was first characterised (McDougall & Cook 1986; Wyeth et al. 1986). Subsequently, aPMV has been reported in the US, Brazil, Central America, France, Israel, Japan, Morocco and Zimbabwe (Cook 2000a; Jones 1996).

TRT is an OIE-listed disease (OIE 2015) and nationally notifiable in Australia. It has not been isolated in Australia.

4.1.2 Technical information

Agent properties

The virus is an enveloped single-stranded RNA virus and is classified as the type strain of the genus *Metapneumovirus* in the family Paramyxoviridae. Molecular analysis has led to division of the various aMPV isolates into four subtypes A, B, C and D. These subtypes are distributed worldwide and can be differentiated based on virus neutralisation tests (Cook & Cavanagh 2002). Subtype aMPV-C is present in the US and is serologically distinct from the European subtypes A and B (Seal 1998, 2000).

aMPV can remain viable in turkey litter for some time, particularly in cold climates. When inoculated into turkey litter the virus remained infective for 3 days at room temperature, up to a month at 8 °C and up to 60 days at minus 12 °C (Velayudhan et al. 2003).

In the laboratory setting aMPV has been inactivated at 56 °C for 30 minutes, 50 °C for 6 hours, 37 °C for 72 hours, and by extremes of pH (<3 and >10) (Jones & Rautenschlein 2013; Townsend et al. 2000).

A survival study on aMPV isolated from Minnesota turkeys indicated that it is very hardy when exposed to different environmental conditions. A cell culture-grown preparation of the virus was not inactivated by freezing at minus 20 °C, there was no loss of infectivity after multiple cycles of freezing and thawing and the virus remained viable after seven days of drying at room temperature. A range of regular disinfectants reduced the log titre of the virus however these results did not consider substances such as organic matter, detergents and surfactants and hard water that could modify the activity of disinfectants in field situations (Townsend et al. 2000).

Epidemiology

Turkeys are the primary hosts. Disease also occurs in chickens however it usually only causes a mild respiratory disease unless complicated by the presence of other pathogens, and the role of aMPV as a primary pathogen in chickens is not well established (Al-Ankari et al. 2001; Cook 2000b). The only other birds known to support the replication of aMPV are guinea fowl, Muscovy ducks and pheasants. Available evidence suggests that no carrier state exists and that aMPV does not exist as a latent infection (Cook 2000b).

The virus is highly infectious and spreads rapidly in susceptible flocks where birds are housed in close proximity. Transmission is likely to be airborne as replication of the virus occurs primarily in the turbinates and lower respiratory tract (Cook 2000b; Seal 2000). Transmission by contaminated water and movement of infected birds and fomites (personnel, vehicles, egg trays) is considered possible although only in-contact spread has been confirmed (Gough & Jones 2008). A recent outbreak in turkeys was caused by exposure to an aMPV vaccine-derived virus that had been present in the environment for at least six months (Lupini et al. 2011).

In the US, aMPV was first isolated in 1997 after a respiratory disease outbreak in turkeys in Colorado (Pedersen, Reynolds & Ali 2000). This outbreak was controlled by slaughter and biosecurity measures and, with no new outbreaks, the disease has been declared eradicated in Colorado. In Minnesota however, the virus is widespread with year round exposure. This may be due to the many large commercial turkey operations with high stocking densities as well as the large number of migratory water birds (Shin et al. 2002b). Wild birds have been implicated as a possible cause of spread of the virus because viral RNA as well as antibodies have been detected in samples from wild birds in Minnesota and other states of the US. However the specific role of wild birds remains unclear (Bennett et al. 2004; Shin et al. 2000; Turpin et al. 2008). Even though Minnesota lies directly under a major wildfowl flyway from Canada to Central and South America, there has been no evidence of southern spread of type C aMPVs from Minnesota or type A and B viruses from Central and South America (Gough & Jones 2008).

Antibodies to the virus have been detected in turkey flocks in states neighbouring Minnesota—North and South Dakota, Wisconsin and Iowa—however it is present at a much lower incidence than in Minnesota where 42% of flocks tested were seropositive (Bennett et al. 2004).

aMPV can be isolated for only a short time (5–7 days) in the infected, non-vaccinated bird although detection of viral RNA in cloacal and pharyngeal swabs of experimentally infected chickens can be achieved for several weeks (Hess et al. 2004). Field evidence suggests that transmission of aMPV through the egg (either transovarially or by egg contamination) is unlikely to occur (Cook 2000b). However, simultaneous aMPV infections in neonatal turkey flocks were reported in three separate states of the US where a common parent breeder source flock was identified for some of the affected flocks, raising the possibility that the infections were egg-transmitted (Shin et al. 2002a).

Pathogenesis

Replication of aMPV in growing turkeys appears to be limited to the respiratory tract. This is generally of short duration (up to ten days post-inoculation) with failure to isolate the virus from turkey cloacal swabs four and ten days after intranasal inoculation (Gough et al. 1988).

In 2005, a more virulent aMPV isolate was detected in turkeys in Minnesota (Velayudhan et al. 2005). Investigation of the tissue distribution of this isolate identified viral RNA up to 11 days post-inoculation from nasal turbinates and up to 9 days from the trachea. Viral RNA was not detected in

liver, lungs or spleen and viral antigen was detected in lung tissue in only two birds out of five. In another study, viral antigen was detected in epithelial cells of turbinates, trachea and lung, with the most severe lesions seen in the turbinates (Majó et al. 1995).

aMPV has been isolated from the cloaca and magnum of turkeys and viral antigen has been detected in uterine epithelium and the oviduct (Jones et al. 1988). However, replication of aMPV in the oviduct cannot be demonstrated (Kherna & Jones 1999).

Diagnosis

Clinical signs

aMPV causes an acute, highly contagious, upper respiratory tract infection which is often exacerbated by secondary bacterial infection. The disease is characterised by sneezing, tracheal rales, nasal and ocular discharge and swollen infraorbital sinuses. Coughing and head shaking are also observed. In laying turkeys the respiratory infection is usually much less severe however a substantial drop in egg production may occur (Cook 2000a). Severity of clinical disease is influenced by management practices such as ventilation, hygiene and stocking densities (Naylor & Jones 1993).

In experimentally inoculated turkey poults the most commonly observed signs were nasal discharge, swelling of the infraorbital sinuses and frothy ocular discharge (Jirjis et al. 2002).

Post-mortem examination

In naturally occurring infections in turkeys that are complicated by secondary infections, post-mortem findings include airsacculitis, pericarditis, pneumonia and perihepatitis (Gough & Jones 2008). In poults experimentally inoculated with aMPV, post-mortem findings were limited to swelling of the infraorbital sinuses with accumulation of clear frothy fluid and frothy mucoid fluid within the nasal cavity (Jirjis et al. 2002).

In a histopathologic and immunocytochemical study of chickens and turkeys experimentally infected with aMPV, the most consistent and severe lesion was seen in the turbinates (Majo et al 1995).

Testing

aMPV may be isolated from infective mucous, nasal secretions or sinus scrapings in chicken or turkey embryo tracheal organ culture (McDougall & Cook 1986; Wyeth et al. 1986), or in embryonated eggs inoculated via the yolk sac (Cook et al. 1999). Due to the short duration of virus shedding, isolation should be attempted at the first sign of clinical disease. The virus may be grown in chick embryo fibroblasts, chick embryo liver cells or Vero cells (Gough & Pedersen 2008).

4.1.3 Current biosecurity measures

There are current biosecurity measures in place for this disease in the *Conditions for the importation from approved countries of fertile eggs (domestic turkey)*. The requirement is either certification of country freedom from aMPV or certification of disease freedom of the source flock. The source flock must be certified as free from signs of disease for the 90 days prior to egg collection and tested serologically for freedom from disease within 21 days before the start of egg collection.

4.1.4 Conclusion

- aMPV is present in the US but has not been identified in Australia.

- Viral replication occurs principally in tissues of the respiratory tract which are generally removed from turkey carcasses at slaughter. It may be present if remnants of these organs remain after processing or if the carcass is contaminated during processing.
- aMPV is inactivated by heat (Jones & Rautenschlein 2013) and cooking at the levels described in Section 1.2.2 are sufficient to address biosecurity concerns.

Therefore the department concluded that further risk assessment of avian metapneumovirus was not required.

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4.2 Avian reovirus, emerging strains in turkeys

4.2.1 Background

Avian reoviruses are associated with a number of poultry disease conditions including malabsorption syndrome, runting/stunting syndrome, diarrhoea, myocarditis, respiratory disease and sudden death, although in many cases a causal association is not proven. However, avian reoviruses are most often identified as the cause of infectious viral arthritis/tenosynovitis in chickens and occasionally in turkeys (Jones 2000).

While reovirus infections are widespread, 85–90% of avian reoviruses are considered to be non-pathogenic (Jones 2000). Production losses due to viral arthritis/tenosynovitis have prompted the development of vaccines in some countries to prevent infections in commercial poultry.

Reovirus is not OIE-listed and is not notifiable or subject to official control or eradication in Australia. Vaccination is not practised in Australia as Australian reovirus strains appear to be of low virulence and are rarely found as the sole causative organism in arthritis/tenosynovitis (Hussain, Spradbrow & MacKenzie 1981; Meanger et al. 1997).

A problem has been identified over the last few years in turkeys farmed in the Midwestern US where strains of reovirus appear to be causing synovitis and rupture of the flexor digital tendons leading to lameness, poor weight gains and increased mortality (Stockam et al. 2012; Wojcinski 2012). This problem appears to be re-emerging after it was first reported in the late 1980s (AL Afaleq & Jones 1989; Mor et al. 2013).

This risk assessment only considered tenosynovitis in turkeys caused by the emerging, exotic strains of reovirus that have been identified in the US.

Avian reovirus (emerging strains in turkeys) is not an OIE-listed disease (OIE 2015) and is not nationally notifiable in Australia. It has not been isolated in Australia.

4.2.2 Technical information

Agent properties

Avian reoviruses are non-enveloped, double-stranded RNA viruses, belonging to the genus *Orthoreovirus*, family Reoviridae. Eleven serotypes and many subtypes of avian reoviruses exist, of which only some are pathogenic for poultry (Saifuddin et al. 1989). Most reovirus isolates in the US and most of their vaccine strains belong to the standard S1133 serotype (Van der Heide 1996). Australian strains appear to have evolved separately from US strains, with variation in the nucleotide sequences between the two strains. The serological relationship between US and Australian strains is unknown (Liu, Giambrone & Nielsen 1997).

Avian reoviruses are stable for more than two months at room temperature, more than three months at 4 °C, and over four years at minus 20 °C (Dutta & Pomeroy 1967; Robertson & Wilcox 1986; Rosenberger 2003). Avian reoviruses have 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).

Reoviruses are quite heat resistant and have been reported as stable at 50 °C for up to two hours (Carboni et al. 1975; Deshmukh & Pomeroy 1969; Glass et al. 1973; Robertson & Wilcox 1986) while another study reported 50% of the reovirus infectivity remaining after 30 minutes at 50 °C (Estes et al. 1979). At 56 °C reoviruses remained stable for 60 minutes (Glass et al. 1973) or were only partially inactivated within 10 to 30 minutes (Dutta & Pomeroy 1967; Mustaffa-Babjee, Spradbrow & Omar 1973). In a more recent study reoviruses were destroyed within three minutes when effluent was heated at 82.2 °C (Chmielewski et al. 2011).

Reoviruses are relatively resistant to disinfectants such as 2% formaldehyde at 4 °C and 2% phenol at room temperature, but sensitive to 100% ethyl alcohol and to chlorine disinfectants (Meulemans & Halen 1982; Robertson & Wilcox 1986). Reoviruses are stable over a wide pH range with studies reporting stability at pH 3.0 and pH 9.0 for four hours at 4 °C, at pH 3.0 for at least one hour, and at pH 3.0 and pH 7.0 for three to five hours at room temperature (Gershowitz & Wooley 1973; Glass et al. 1973; Robertson & Wilcox 1986).

Epidemiology

Reoviruses have been identified in a number of species other than chickens and turkeys, including Muscovy ducks, pigeons and parrots (Jones 2000). Although there is potential for cross-species infection, wild birds have not been demonstrated to act as a reservoir for infection of poultry (Jones 2003).

The disease being observed in turkeys in the US has mainly affected males over 14 weeks of age. Females can be affected but they are usually processed by this age. Multiple producers across a number of Midwestern states have reported affected flocks with a sudden onset of symptoms and rapid spread (Mor et al. 2013; Wojcinski 2012).

Vertical transmission from breeder flocks appeared to be the most likely mode of transmission in early field outbreaks as successive flocks placed in affected farms have not always shown symptoms (J. Stockham pers. comm., Western Poultry Disease Conference 2 April 2012; J. Trites pers. comm., Western Poultry Disease Conference 2 April 2012). Vertical transmission has been identified in other strains of reovirus (Al-Muffarej, Savage & Jones 1996; Menendez, Calnek & Cowen 1975; Van der Heide & Kalbac 1975). Studies on vertical transmission of these US strains have not been done however, there is experimental evidence that horizontal transmission can occur (Sharafeldin et al. 2014b).

The incidence in the US is uncertain—106 farms were said to be affected in 2011 but one operator reported they had processed over 300 flocks showing some evidence of viral arthritis/tenosynovitis in that year (Clark, Corsiglia & Bailey 2011). To date, only flocks in the Midwestern US have been affected and the incidence appears to be decreasing following the introduction of control measures (Clark & Bailey 2014).

There are reports of clinical signs similar to those in turkey broilers now being seen in chicken broilers. This has occurred in the progeny of donor flocks vaccinated with conventional S1133 strain vaccines and these novel chicken reoviruses are serologically different to the strains causing disease in turkeys (Putnam et al. 2014; Rosenberger et al. 2014).

Pathogenesis

Reovirus has been isolated from digital flexor tendons, synovial fluids and gastrocnemius tendons in turkeys showing the clinical signs described above. Reoviruses isolated from the intestinal tracts of these birds appear to be genetically different from the reoviruses isolated from the affected joints

(Mor et al. 2014; Rosenberger et al. 2012). Early infectivity studies using strains isolated from turkey hocks showed that turkeys appear to be considerably more resistant than chickens to avian reovirus, and chickens inoculated with the turkey strains developed significant arthritic changes (AL Afaleq & Jones 1989; Al Afaleq & Jones 1991).

Inoculation of young poults with viruses isolated from the gastrocnemius and digital flexor of affected turkeys via the oral, intra-tracheal and footpad routes led to virus detection in tendons, intestines and internal organs one and four weeks post-inoculation (Sharafeldin et al. 2014a). Changes in the gastrocnemius tendons of inoculated birds can be detected by histopathology at one week post-inoculation, without overt lameness, suggesting that poults are affected early in life and only show clinical signs as they reach older ages and heavier weights (Sharafeldin et al. 2014b).

Diagnosis

Clinical signs

Abnormal gait, lameness, swollen hocks and extended lateral toes on one or both feet are the first clinical signs observed along with sudden mortality (Rosenberger et al. 2012; Stockam et al. 2012). Within a flock, morbidity varies from 2 to 70% and mortality from 2 to 35% (Mor et al. 2013; Trites et al. 2012).

Testing

The most common lesions observed in turkeys on post-mortem are rupture of the digital flexor tendons and synovitis (Rosenberger et al. 2012). Also a significant correlation between aortic rupture and digital flexor tendon rupture has been identified post mortem (Stockam et al. 2012).

Because of the high prevalence of subclinical and clinical infections in poultry, serology is not generally useful for the diagnosis of reovirus (McNulty 1993). Serology may be used to monitor the status of specific pathogen free (SPF) flocks or antibody levels in vaccinated breeder flocks and commercial ELISA tests are now available for this purpose. Diagnosis is best achieved using virus isolation, but virus can also be demonstrated in tissues with the use of PCR, direct immunofluorescence staining and other techniques (Jones 2000).

Treatment

Vaccines based on the standard S1133 serotype appear not to be protective (Rosenberger et al. 2012). An autogenous killed vaccine has been developed and along with changes to flock management, this appears to be reducing incidence in the field (Clark & Bailey 2014).

4.2.3 Current biosecurity measures

Currently, no biosecurity measures exist for avian reovirus in turkeys. Only canned or retorted turkey meat products that meet specific temperature and time requirements during the manufacturing process are permitted for import into Australia at this time.

4.2.4 Conclusion

- Although some strains of avian reovirus are endemic in Australian poultry, and 85–90% of reovirus strains are considered to be non-pathogenic, the strains responsible for the synovitis seen in the Midwestern US over the past few years are considered to be exotic to Australia.

- Reoviruses associated with joint lesions in chickens can persist in joints for long periods (Jones 2000). New strains causing joint disease in grower turkeys are likely to be similar. Therefore, reovirus may be present in joints or tendon sheaths of grower turkeys at slaughter.
- Reoviruses are heat resistant, and are able to withstand 60 °C for eight to ten hours (Rosenberger 2003). Reoviruses were destroyed within three minutes when effluent was heated at 82.2 °C (Chmielewski et al. 2011) however it is possible that viable virus would persist in tissues after cooking at the level described in Section 1.2.2.
- Reovirus 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 emerging reoviruses in turkeys was required.

4.2.5 Risk assessment

Entry assessment

- Reovirus infections are widespread in both Australia and the US, and 85-90% of avian reoviruses are considered to be non-pathogenic (Jones 2000). However, the emerging strains responsible for the synovitis seen in Midwestern US turkeys over the past few years are considered exotic to Australia.
- The emerging strains of reovirus are confined to a specific region within the US (Clark, Corsiglia & Bailey 2011).
- Within a flock, morbidity varies from 2-70 % and, mortality from 2-35 % (Mor et al. 2013; Trites et al. 2012).
- The incidence appears to be decreasing in the face of control measures (Clark & Bailey 2014).
- The disease is associated with a sudden onset of symptoms (abnormal gait, lameness, swollen hocks and the extension of lateral toes on one or both feet) (Wojcinski 2012), but it can be subclinical. Therefore, it may only be recognised at ante-mortem and, to a lesser extent, post-mortem examination.
- Reovirus may be present in joints or tendon sheaths of grower turkeys at slaughter and contamination may occur at processing.
- While it is possible that viable virus would persist in tissues after cooking at the levels described in Section 1.2.2, it is likely that cooking would reduce the infectious viral load.

Conclusion: based on this information, the likelihood of entry of emerging reovirus strains associated with the importation of cooked turkey meat from the US was estimated to be **low**.

Exposure Assessment

- The emerging reovirus strains being assessed are pathogenic only for turkeys. Although there are reports of similar symptoms in chickens that have been vaccinated with standard S1133 reovirus vaccines, these novel chicken reoviruses are serologically different to the strains causing symptoms in turkeys (Putnam et al. 2014).

- Current data indicates that turkeys are infected early in life and develop symptoms as they reach heavier weights (Sharafeldin et al. 2014b).
- Although there is experimental evidence that emerging reoviruses can be transmitted laterally, the major route of transmission in the field appears to be vertical (J. Stockham pers. comm., Western Poultry Disease Conference 2 April 2012; J. Trites pers. comm., Western Poultry Disease Conference 2 April 2012).
- The only exposure pathway for commercial turkeys is via feed containing meat meal made from waste from imported turkey meat. Rendering will inactivate any reovirus present.
- Backyard turkeys may be exposed to the waste from domestic consumption of imported turkey meat. However, given the very limited population of backyard turkeys in Australia, and the age at which they would have to be exposed to become infected, this exposure pathway has a low likelihood.
- Backyard chickens and wild birds may also be exposed to the waste from domestic consumption of imported turkey meat. The ability of these emerging strains to infect and reproduce in species other than turkeys is not known but there are differences in the virus strains that cause similar symptoms in chickens and turkeys (Mor et al. 2014; Putnam et al. 2014). Wild birds can be infected by reovirus strains however the importance of wild birds as a reservoir of reovirus infection for poultry has not been demonstrated (Jones 2000).

Conclusion: Based on this information, the likelihood of exposure of domestic poultry to emerging, exotic, reovirus strains associated with the importation of cooked turkey meat from the US was estimated to be **very low**.

Estimation of the likelihood of entry and exposure

The likelihood of entry of this agent to Australia and the corresponding likelihood of its exposure to the Australian turkey population was estimated by using the matrix of rules described in Table 1. As the estimate of the likelihood of entry is low and the likelihood of exposure is very low the estimation of the likelihood of entry and exposure of reovirus was estimated to be **very low**.

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

The most likely outbreak scenario following exposure to emerging reoviruses is considered to be establishment in populations of susceptible turkeys with vertical spread to more than one state.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible turkeys to emerging strains of reovirus:

- Turkeys are the only species identified as being affected by these emerging strains.
- These emerging strains are primarily transmitted vertically.
- Reoviruses can persist in the environment. All-in, all-out farming and cleanout and disinfection of housing between batches in commercial turkey operations will limit the establishment of the infection on these sites.

Conclusion: Based on these considerations, it was estimated that the likelihood of establishment and spread of emerging strains of reovirus through the Australian turkey population was **low**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect impacts of emerging reoviruses were estimated at the national, state or territory, district/region and local levels. Adverse effects were evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were assessed as relevant to decide on the effects of the establishment and/or spread of emerging strains of reovirus.

Direct effects

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

- Emerging strains of reovirus appear to cause symptoms only in turkeys around market age. Emerging reoviruses can cause widespread morbidity and mortality in affected turkey flocks (Mor et al. 2013; Stockam et al. 2012; Trites et al. 2012).

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

- **There are no known effects on the living environment—wild birds can be infected with reoviruses but it is not known if emerging strains in turkeys can either infect wild birds, or cause adverse effects.**

Indirect effects

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

- Avian reovirus is not notifiable in any Australian jurisdiction and there are no control, monitoring or surveillance programs in place.
- Infection in medium biosecurity poultry would likely provoke similar control and eradication measures that have been introduced in the affected areas of the US—surveillance and the development and administration of autogenous vaccines. There would be little or no effects on the non-commercial turkey population.

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

- Infection in medium biosecurity poultry would likely require inputs from the biotechnology industry in the development and deployment of new vaccines. Commercial customers may be affected by shortages of turkey meat due to direct losses of stock.
- There would be no effects on consumer demand.

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

- There would be no impact on international trade.

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

- There would be no discernible effects on the environment.

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

- There would be no discernible effects on communities.

Conclusion for overall direct and indirect effects: Based on the geographic level and magnitude of effects, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **low** from Table 2. The effect is likely to be significant for directly affected parties but indiscernible at any other level.

Consequence assessment

The estimate of the overall effect associated with the outbreak scenario (low) was combined with the likelihood of establishment and/or spread for the scenario (low) using Table 3 to obtain an estimation of likely consequences (**very low**).

4.2.6 Risk estimation and evaluation

Risk estimation is the integration of likelihood of entry and exposure, and likely consequences of establishment and/or spread to derive the risk associated with entry, exposure, establishment and/or spread of emerging strains of reovirus being introduced by cooked turkey meat imported into Australia.

Using Table 4, the likelihood of entry and exposure (very low) was combined with the likely consequences of establishment and/or spread (very low), which resulting in a risk estimation of **negligible**.

Therefore, as the unrestricted risk estimate achieves Australia's ALOP, no specific risk management was considered necessary for this agent.

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4.3 Clostridial dermatitis

4.3.1 Background

Clostridial dermatitis (CD) is the term agreed to in 2008 to describe an emerging and increasingly severe disease syndrome with specific pathology identified in growing turkeys in the US. It has also been referred to as clostridial cellulitis and gangrenous dermatitis (Clark et al. 2010; USDA 2012). Although individual birds showing pathology consistent with CD have been recognised for some time, CD as a major flock problem in turkeys emerged in the mid 1990s and increased in incidence and severity in the 2000s (Carr et al. 1996; Opengart 2008; USDA 2012).

Current evidence identifies *Clostridium septicum* as the main causative agent. *Clostridium perfringens* and occasionally *Clostridium sordelli* have been implicated in clostridial dermatitis cases in some studies (Clark et al. 2010; Thachil et al. 2010; USDA 2012).

CD is not OIE-listed and is not notifiable or subject to official control or eradication in Australia. Although the putative causative agents are present, CD has not been reported as a disease in turkeys in Australia.

4.3.2 Technical information

Agent properties

Clostridia are gram positive, anaerobic, spore-forming bacteria commonly present in the environment. *Clostridium perfringens* and *C. septicum* produce numerous toxins including a lethal and necrotising α-toxin that directly damages soft tissues (Huff, Huff & Rath 2013; Songer 1996; Thachil 2011).

In meat, *C. perfringens* spores isolated from cases of food poisoning exhibit high resistance to various physical factors including moist heat, osmotic, nitrite, and pH-induced stress, prolonged frozen storage and high hydrostatic pressure (Akhtar et al. 2009). In one study, *C. perfringens* spores survived after heating at 85 °C to 135 °C (Adams 1973).

Yolk cultures of *C. colinum*, another clostridia found in avian species, survive heating at 70 °C for three hours, 80 °C for one hour and 100 °C for three minutes (Songer & Uzal 2013).

Epidemiology

CD describes a condition only seen in turkeys despite its similarities to gangrenous dermatitis syndromes seen in chicken broiler flocks. In chickens, infection with clostridia, staphylococcus or other agents usually occurs secondary to immune suppression that is often due to infectious bursal disease virus and chicken anaemia virus (Rosenberger et al. 1975; Vielitz & Landgraf 1988).

CD has only been recognised in the US and is most often seen in male flocks approaching processing age, that is, those over 13 weeks. By this age most female flocks have been processed. However, the incidence in hens and in younger birds (it has been seen in flocks as young as seven weeks) has also been increasing (Clark et al. 2010; Huff, Huff & Rath 2013).

The transmission route of CD is poorly characterised but is likely to be transmitted bird to bird either via a break in the skin or by ingestion from a contaminated environment (Clark et al. 2010). Management and production practices in the US, including the common practice of the reuse of litter

that provides a significant source of *C. septicum*, may explain why CD only occurs in the US and not in other areas where heavy turkey production occurs, such as Europe (Clark et al. 2010; Huff, Huff & Rath 2013).

A USDA study reported that 42% of farms surveyed had seen some evidence of CD in the previous 12 months. This varied by region with CD not being recognised in the western states of the US (west coast and Rocky Mountain states) but identified as prevalent in the east and central areas of the country (USDA 2012). The prevalence and severity of CD has continued to increase and the disease has been identified as one of the most important disease problems associated with turkey production (Huff, Huff & Rath 2013; Thachil et al. 2012).

Pathogenesis

The pathogenesis of CD is not known due to the lack of experimental data however, there are two theories (i) disease is the result of penetration of clostridia through the skin (the 'outside-in' approach) or (ii) disease is the result of the proliferation and spread of clostridia present naturally in the gastrointestinal tract (the 'inside-out' approach) (Clark et al. 2010). *C. septicum* has been isolated from liver and spleen in birds suffering from CD, supporting the theory that there is systemic spread via the circulation to the affected areas (USDA 2012).

The disease has been reproduced by subcutaneous and intravenous challenge with *C. septicum* as well as exposure to used litter containing *C. septicum* suggesting that it is the primary cause of CD (Davis 2011; Tellez et al. 2009; Thachil et al. 2010).

Dexamethasone-immune suppressed turkeys exposed to *C. septicum* orally and subcutaneously were more likely to develop CD than non-immune suppressed turkeys (Nagaraja et al. 2011). CD was also seen in dexamethasone-immune suppressed turkeys that were used as a model to study turkey osteomyelitis complex (Huff, Huff & Rath 2013). In these turkeys, the immune suppression appeared to be the major factor in the development of CD with clinical signs and mortality occurring without additional bacterial challenge. This indicates that immune suppression, caused by stress, is a major factor in the occurrence of CD and the potential influence of infectious immune suppressive agents should be considered (Clark et al. 2010).

Diagnosis

Clinical signs

Lateral recumbency, anorexia, ataxia and cyanosis of the head have been reported, often with a rapid onset of high mortality (Clark et al. 2010). Inflammation of the subcutaneous tissue, particularly on the tail and vent (described as a bubbly tail) and accumulation of gelatinous fluid over the breast area is often seen (Huff, Huff & Rath 2013).

Post-mortem examination

Gross pathology is distinctive—discolouration of the skin, serosanguinous gelatinous exudates and/or crepitus from gas are present in the subcutaneous tissue. There may be blistering or bubbling around the feather follicles of the tail and darkening and petechial haemorrhaging of underlying musculature (Carr et al. 1996; Clark et al. 2010; USDA 2012).

Testing

Impression smears of skin underlying lesions may show characteristic Gram positive rods. A definitive diagnosis can be made by fluorescent antibody test or by culturing skin (dermis) on a suitable

anaerobic media, confirmed by biochemical reactions. A PCR for the α toxin of *C. septicum* has also been developed (Clark et al. 2010).

Treatment

Antibiotics have been used in outbreaks as well as prophylactically in attempts to reduce the impact of the disease (Carr et al. 1996; Clark et al. 2010). Development of vaccines using *C. septicum* is underway at a number of centres (Tellez et al. 2009; Thachil et al. 2013).

Clark et al (2010) list 31 suggestions to either prevent or control CD. These include the importance of early diagnosis, therapeutics and the management of flocks such as stocking densities, sanitation, biosecurity, in-shed environment, gut health and feed additive use (Clark et al. 2010).

4.3.3 Current biosecurity measures

Currently, no biosecurity measures exist for the specific agents associated with clostridial dermatitis in turkeys. Only canned or retorted turkey meat products that meet specific temperature and time requirements during the manufacturing process are permitted for import into Australia at this time.

4.3.4 Conclusion

- CD is present in the US and not in Australia. However, the causative agents are present in Australia.
- Clostridial spores are highly resistant to heat and are not destroyed by normal cooking temperatures.
- CD appears to be a disease of production where the interaction of environmental and other stresses lead to immune suppression and clinical disease.
- Clostridial organisms could be present in the carcasses of diseased birds as well as in unaffected birds but given the rapid course of the disease and the extensive gross pathology, birds with CD are very unlikely to be processed.

Therefore, the department concluded that further risk assessment of clostridial dermatitis was not required.

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4.4 Exotic antigenic variant and very virulent strains of infectious bursal disease virus

4.4.1 Background

Infectious bursal disease (IBD) is an acute and highly contagious viral infection causing varying degrees of mortality and immunosuppression in chickens (Lukert & Hitchner 1984; OIE 2008). Clinical signs and severity depend on the genetic lineage and immune status of the chickens and the dose and type of infectious bursal disease virus (IBDV) (Eterradossi & Saif 2013). IBDV only produces clinical disease in chickens however natural infection can occur in turkeys, with evidence of subclinical disease reported (Giambrone et al. 1978; OIE 2008). IBD was first recognised as a disease in chickens in 1957 and was named Gumboro disease after the area in the US where IBDV was first identified (Cosgrove 1962).

There are two serotypes of IBDV, serotype 1 and serotype 2 (Jackwood, Saif & Hughes 1982; McFerran et al. 1980; McNulty & Saif 1988). Serotype 1 is an important pathogen of chickens while serotype 2 can often be present in both chickens and turkeys however it does not cause clinical disease in either species (Jackwood, Saif & Hughes 1982; Jackwood, Saif & Moorhead 1985; Weisman & Hitchner 1978). IBDV serotype 1 is commonly differentiated into two major groups antigenically—classic and variant, and into three groups pathogenically—attenuated (vaccine), classic virulent and very virulent. Pathogenic strains are confined to serotype 1 and reassortants of serotypes 1 and 2 (Ismail et al. 1990; Jackwood et al. 2011; van den Berg et al. 2004).

For the purposes of this risk assessment, exotic antigenic variant strains are defined as variant strains that are antigenically and genetically different from those that exist in Australia. This risk assessment is concerned with IBD viruses that are exotic to Australia, including the very virulent IBDV strains and IBDV strains that are antigenically and genetically different from Australian strains.

IBDV is an OIE listed disease, and exotic antigenic variant forms of IBDV and vvIBDV are nationally notifiable in Australia.

4.4.2 Technical information

Agent properties

IBDV is a member of the Birnaviridae family, *Avibirnavirus* genus (Dobos et al. 1979; ICTV 2014; Ignjatovic & Prowse 1997). It is a single shelled, non-enveloped virion with a genome consisting of two segments of double-stranded RNA— segment A and segment B (Jackwood et al. 2011; Macdonald 1980).

IBDV can persist for extended periods in the environment. The virus remained viable for more than 12 months in unused, dry chicken sheds; at least 6 months in dry litter; up to 122 days in the shed environment, and up to 52 days in feed, water and faeces (Benton, Cover & Rosenberger 1967; Edgar & Cho 1976).

IBDV is very resistant to heat and certain temperature and time combinations may reduce the viral load, while others will give complete inactivation. In one study, reduction of the infectivity by 1 $\log_{10}$ took 18.8 minutes at 70 °C, 11.4 minutes at 75 °C and three minutes at 80 °C (Alexander & Chettle 1998). Unpublished work conducted in 1997 at the Quality Control Unit, Central Veterinary Laboratory, Alderstone, United Kingdom, showed that a mixture of bursal homogenate (23%), skin

and fat (4%), muscle tissue (23%) and peptone broth (50%) contained no viable IBDV only after cooking at 80 °C for at least 120 minutes (Quality Control Unit).

Gamma irradiation has a limited effect on IBDV. At 3 kilograys there was no reduction in the titre of pathogenic strains while some strains remained viable after application of 10 kilograys (Jackwood, Sommer-Wagner & Pharo 2007).

IBDV has been shown to be resistant to ether, chloroform and pH 2, but inhibited by pH 12 and iodine complex disinfectants (Benton et al. 1967). In another study only disinfectants which contained aldehyde (at 20–22 °C) or chlorine (at 4 °C and 20–22 °C) were effective against IBDV (Meulemans & Halen 1982). Subsequent research demonstrated that invert soaps containing 0.05% sodium hydroxide with a pH of at least 12, at or above room temperature, inactivated or strongly inhibited the virus (Shirai et al. 1994).

A 5 $\log_{10}$ reduction in virus was achieved at minus 20 °C when Virkon and Surface Decontamination Foam (SDF) was applied to a dried suspension of IBDV and organic matter for 2 and 24 hours respectively. In comparison, bleach produced no measurable reduction in infectivity at minus 20 °C; however, there was a reduction of 5 $\log_{10}$ within two hours at 23 °C and 4 °C, while SDF and Virkon applied at 23 °C and 4 °C reduced IBDV by 5 $\log_{10}$ within 15 minutes (Guan et al. 2014).

Epidemiology

IBDV occurs in all major poultry producing areas worldwide except New Zealand. IBDV was first identified in the US and both serotypes 1 and 2 are present in chicken and turkey flocks (Candelora, Spalding & Sellers 2010; Cosgrove 1962). Serotype 2 is widespread in turkeys in the US and produces much higher antibody titres in turkeys than serotype 1 (Chin et al. 1984; Jackwood, Saif & Hughes 1982).

IBDV is extremely hardy and highly contagious. It can be transmitted horizontally via faeces, with spread mainly by the faecal-oral route, on fomites, or through airborne dissemination of feathers and poultry shed dust (Benton, Cover & Rosenberger 1967; Candelora, Spalding & Sellers 2010; Giambrone et al. 1978). There is no evidence of a carrier state in recovered birds or of vertical transmission (Eterradossi & Saif 2013).

IBDV can infect wild birds but is not known to cause disease. Serological evidence of infection of wild birds with both IBDV serotypes 1 and 2 has been identified (Candelora, Spalding & Sellers 2010; Ogawa et al. 1998; Oladele 2010; Wilcox et al. 1983). Antibodies to IBDV serotype 1 have been detected in Australian flesh-footed shearwaters, silver gulls and black ducks (Wilcox et al. 1983). Lesser mealworms (litter beetles) have been identified as a reservoir host for IBDV (Eterradossi & Saif 2013; McAllister et al. 1995; Okoye & Uche 1986). IBDV has also been shown to infect rats and dogs though they have no known role in the spread of the virus (Okoye & Uche 1986; Pagès-Manté et al. 2004).

Both variant and very virulent strains of serotype 1 are present in the US but variant strains are more common (Jackwood & Sommer-Wagner 2010). Virulent reassortant strains that are pathogenic for chickens but not turkeys are also present (Jackwood et al. 2012; Jackwood et al. 2011).

Variant strains of serotype 1 were identified in the US in 1985 and they now make up the majority of strains present in the US (Ismail et al. 1990; Jackwood & Sommer-Wagner 2010). These differ from the classical IBDV strains and therefore vaccines used at the time they appeared were not effective as they were based on the classical strains (Jackwood & Saif 1987). Although antigenic variants have

been identified in Australia, they are genetically distinct from those in the US (Jackwood et al. 2006; Sapats & Ignjatovic 2000) and the vaccines used in Australia are not protective against variants present in the US (Ignjatovic, Sapats & Gould 2001).

Serotype 1 IBDV is pathogenic for chickens while serotype 2 IBDV infects chickens and turkeys but is avirulent for both (Mahgoub 2012). In a study of six to eight week old turkeys that were inoculated with IBDV, the birds developed no clinical signs and attempts at virus isolation failed. However, the turkeys did respond serologically by producing virus neutralising antibodies which indicates they may have been sub-clinically infected (Weisman & Hitchner 1978).

Another study inoculated three to six week old turkeys with IBDV isolated from clinically infected chickens (presumed to be IBDV serotype 1). The virus was passaged six times in turkey poults to increase the pathogenicity for turkeys however, no clinical signs developed. Subclinical infection was identified on post-mortem and infected poults developed virus neutralising antibodies but at much lower levels than typically observed in chickens (Giambrone, Dormitorio & Brown 2001). The virus isolated from the poults was then inoculated back into chickens, producing clinical IBDV in the chickens. The authors concluded that it was possible for IBDV of chickens to naturally adapt to turkeys and that turkeys may serve as a reservoir for IBDV (Giambrone et al. 1978).

A recent study investigated the persistence and tissue distribution of IBDV serotype 1 in turkeys following inoculation. The authors concluded that turkeys can be infected with IBDV serotype 1 but do not show signs of disease (Abdul, Murgia & Saif 2015).

The very virulent form of IBDV (vvIBDV) was first identified in Belgium and the Netherlands in the 1980s. It causes an immunosuppressive disease characterised by high mortality (van den Berg, Gonze & Meulemans 1991). vvIBDV has since been reported in Africa, Asia, Europe, Japan, Latin America and the US (Eterradossi et al. 1999; Hernandez et al. 2006; Lin et al. 1993; van den Berg et al. 2004). The first outbreak of disease caused by vvIBDV in the US occurred in California in 2008 and was identified in Washington in 2014 (Stoute et al. 2009; Washington Animal Disease Diagnostic Lab 2015). Very virulent IBDV has not been identified in Australia (Ingrao et al. 2013; Sapats & Ignjatovic 2000).

Turkeys can be naturally infected with vvIBDV. Phylogenetic analysis of samples taken from turkey flocks in Nigeria experiencing unusually high mortalities identified that all of the IBDV sequences were clustered within the very virulent genotype and two turkey strains were indistinguishable from a cluster of nine chicken viruses. Genomic sequencing performed on one turkey isolate identified a high degree of similarity between the turkey isolate and serotype 1 very virulent strains from chickens (Owoade et al. 2004). In Iran, IBD virus was isolated from bursas of 10 week old turkeys in a flock experiencing a 7% mortality rate. PCR and sequencing identified a vvIBDV similar to isolates from chickens (Razmyar & Peighambari 2009). The higher virulence of the vvIBDV strains may allow them to infect a wider variety of avian species, thereby increasing the chance that they will encounter serotype 2 viruses, for example in turkeys, and produce reassortants. These reassortants have the potential to infect chickens (Jackwood et al. 2011).

Recent studies have identified a number of reassorted vvIBD viruses in California (Jackwood et al. 2012; Jackwood et al. 2011). Research on the genome of these reassorted vvIBD viruses using RT-PCR and sequencing has shown that turkeys can be infected with vvIBDV comprising segment A of viral RNA from serotype 1 and segment B from serotype 2. This reassorted vvIBDV was not pathogenic in turkeys; therefore, it is possible for turkeys to act as asymptomatic reservoirs for IBDV (Jackwood et al. 2011).

Prevalence of variant IBDV in chickens in the US

Antigenic variants which are not present in Australia are endemic in US chicken flocks (Hamoud & Villegas 2006; Jackwood & Nielsen 1997; Jackwood & Sommer-Wagner 2005; Rosales et al. 1989; Snyder 1990). Since the release of the chicken meat IRA, more studies on the prevalence of IBDV in chickens in the US have been completed. A review of samples taken from 114 broiler farms across 12 states between 2009 and 2011 showed that nearly all of the 117 sequences identified were variants (Cookson, Jackwood & Turpin 2012). In a study of 26 US poultry processing plants, pooled bursal tissue samples from 47 farms were examined using RT-PCR and SPF chick challenge. Twenty five per cent of pooled samples were positive for IBDV, representing 42% of the processing plants. Phylogenic analysis of the positive samples showed that none were on branches containing classic or vvIBDV; therefore, these samples were all identified as variants (Jackwood & Sommer-Wagner 2010).

Prevalence of variant IBDV in turkeys in the US

Early studies on IBDV in turkeys concluded that only IBDV serotype 2 could infect turkeys and that positive antibody titres to IBDV serotype 1 were due to cross reactivity or vaccination with commercial IBDV serotype 1 vaccines (Barnes, Wheeler & Reed 1982; Chin et al. 1984; Jackwood, Saif & Hughes 1982; Sivanandan et al. 1984). These prevalence studies are now over 30 years old and the strains of IBDV circulating in the US today are very different to those present in the 1980s so their prevalence in turkeys cannot be predicted based on those studies (Dr D. Jackwood, 2015, pers. comm., 6 May).

In addition, these studies may not have accurately indicated prevalence at the time they were completed due to the lack of standardised diagnostic assays for serotype 1 and 2. For example some of the ELISA assays used were later found to be poor at distinguishing between serotype 1 and 2 and an antigenic virus standard was not used in the virus neutralisation assays. It is also possible that the reassorted genomes and recombination events may have influenced the data as the presence of reassortants was unknown when the studies were conducted in the 1980s (Dr D. Jackwood, 2015, pers. comm., 16 April).

The prevalence of IBDV serotype 1 in turkeys is unknown, however, based on the data available the incidence in the US is considered to be very low (Dr. J. Clifford, pers. comm., 29 July 2015).

Prevalence of vvIBDV in turkeys in the USA

Studies in the US have identified multiple reassorted vvIBDV in turkeys in California however whether reassortants are present in other states is not known. Naturally occurring reassorted vvIBD viruses have reduced pathogenicity in both chickens and turkeys so they may occur without being identified. Therefore molecular identification of both genome segments during diagnosis is required to determine the presence of reassortants (Jackwood et al. 2012; Jackwood et al. 2011; Stoute 2012; Wei et al. 2008). Studies to date have only identified reassortant vvIBDV in turkeys in California however strains of vvIBDV continue to emerge from various natural reassortments and the emergence of strains with new antigenic and pathotypic properties is expected to continue (Jackwood et al. 2012).

Transmission in turkey meat

The chicken meat IRA considered that transmission of virus in chicken muscle tissue could then lead to IBD in naive chickens. Infection from carcasses cross-contaminated by gastrointestinal content and direct contact with bursae during processing was also considered a risk. Additionally, a study of eastern US broiler processing plants concluded that contamination of equipment and products posed

a risk of disseminating infectious IBDV to chicken carcasses (Biosecurity Australia 2008; Jackwood & Sommer-Wagner 2010). However, there is no data for turkey processing plants where the disease prevalence and viral load of any IBDV are expected to be lower. Bursae will still be present in turkeys at the time of processing as it starts to regress at 22 weeks and is completely regressed at approximately 32 weeks of age (Cecil & Bakst 1991). Therefore if flocks are infected, it is expected that some birds may be positive for the virus at the time of slaughter (between 9 and 24 weeks). Good hygiene practices at abattoirs will limit contamination from faeces and bursal material that may be infected.

A recent study examines the experimental persistence and tissue distribution of IBDV serotype 1 in turkeys. The study included two age groups (two and four week old poults) and two inoculum doses of IBDV. In two week old poults inoculated with the higher dose (10^4 EID_{50}/0.2 ml/poult), IBD virus was isolated from the bursa up to 14 days post infection (DPI) and was detected by RT-PCR in bursal tissue up to 21 DPI and in splenic tissue up to 7 DPI. All other tissues were IBDV negative. In four week old poults inoculated with the lower dose (10^2 EID_{50}/0.2 ml/poult), splenic and hepatic tissues were RT-PCR positive at 14 DPI, breast muscle and kidney tissue positive at 7 DPI and lung and pancreatic tissue positive at 3 DPI. Only bursal tissues were tested for virus isolation and no virus was isolated. The authors concluded that turkeys can be infected with IBDV serotype 1 but show no signs of disease due to IBDV, and that differences in tissue distribution may be due to age and the infectious dose received (Abdul, Murgia & Saif 2015).

Pathogenesis

In turkeys, no gross pathology has been identified as the result of infection with IBDV (Giambrone et al. 1978; Owoade et al. 2004). Bursal atrophy associated with respiratory disease in turkeys seropositive for IBDV has been described but, as yet, there is no definitive cause (Barnes, Wheeler & Reed 1982). Oladele identified muscular haemorrhage between 12 and 24 hours post inoculation in turkeys experimentally infected with IBDV (Oladele 2010).

Microscopic changes in lymphoid tissue at three, four and five days post inoculation were observed in one study where poults were experimentally infected with serotype 1 virus that had been passaged six times in poults in an attempt to increase pathogenicity. Lesions found in the bursa of Fabricius were variable and included different sized degenerating follicles with cysts and scattered lymphoid cells in the medulla, as well as follicles with large necrotic areas in the medulla and occasional heterophils. Changes to other tissues were minimal and included small numbers of degenerating lymphoid cells in the spleen (bursa-dependent follicles), thymus (cortical area) and caecal tonsils (bursa-dependent follicles) (Giambrone et al. 1978).

Diagnosis

IBDV infection in turkeys usually produces no clinical signs, no gross pathologic lesions and, in many cases, no microscopic lesions. Therefore, presence of the virus is confirmed by demonstration of specific antibodies to IBDV or detection of the virus in tissues using immunological or molecular methods.

Exposure to IBDV can be confirmed by antibody identification in unvaccinated birds or by detecting viral antigen or genomic RNA in tissues (OIE 2014). The most accurate and accepted method of identification of vvIBDV is genomic analysis by nucleotide sequencing in conjunction with pathogenicity testing in chickens (Ignjatovic et al. 2004; Jackwood et al. 2012). However, results of pathogenicity testing can vary depending on experimental design, viral dose used and the genetics of the tested chickens (Jackwood et al. 2012; van den Berg et al. 2004).

Virus isolation and identification is done using homogenates of the bursa of Fabricius (OIE 2008). Virus strains differ in their ability to be cultured in embryonated eggs or cell culture, with very few field strains of IBDV being able to replicate in the latter (Dr D. Jackwood, pers. comm., 16 April 2015). In addition to pathogenicity testing in specific antibody free chickens, strain identification can be performed using the virus neutralisation test (VNT), monoclonal antibodies or determination of the nucleotide sequence from RT-PCR amplification products (OIE 2014).

Viral antigens in the bursa can be demonstrated by direct and indirect immunofluorescence or by immunoperoxidase staining of thin sections of bursal tissue. Other tests such as agar gel immunodiffusion (AGID) and agglutination tests can also be used to demonstrate the presence of viral antigens but are relatively insensitive. PCR, DNA probes and nucleotide sequencing have also been used to demonstrate and characterise the presence of IBDV (Brown, Green & Skinner 1994; OIE 2014). Antibodies to IBDV can be detected using AGID, counterimmunoelectroosmophoresis (CIEOP), ELISA or VNT (Oladele 2010). VNT is the only serological test that can differentiate between the IBDV serotypes (Eterradossi & Saif 2013; Ismail & Saif 1990).

The classic and variant strains present in Australia are a distinct group of strains that are different from other classical and variant strains overseas. This enables differentiation of most if not all exotic IBDV strains from Australian strains by nucleotide sequencing (Ignjatovic & Prowse 1997; Ignjatovic & Sapats 2002; Sapats & Ignjatovic 2000).

Clinical signs

Clinical signs in chickens are described in the *Generic Import Risk Analysis Report for Chicken Meat 2008* (Biosecurity Australia 2008). Clinical disease due to infection with IBDV has not been described in turkeys. Turkeys experimentally infected with IBDV show no clinical signs of disease. Chickens may be severely affected by serotype 1 as it causes clinical infection and immune suppression in chickens younger than 10 weeks of age (Jackwood et al. 2011; OIE 2008). It is important to identify the serotype and strain of IBDV present to assess the potential implications for chicken producers (Giambrone et al. 1978; Owoade et al. 2004).

4.4.3 Current biosecurity measures

There are current biosecurity measures in place for this disease in the *Conditions for the importation from approved countries of fertile eggs (domestic turkey)*. These require that cloacal swabs be collected and tested from a sample of the quarantine flock at six weeks of age sufficient to give 99% confidence of detecting 5% prevalence. In addition the sentinel chickens placed with the quarantine flock are tested serologically for IBDV.

Current biosecurity measures are also in place for this disease in the *Generic Import Risk Analysis Report for Chicken Meat 2008.* Requirements are either a country or zone that has been recognised as free of variant IBDV and vvIBDV by the Australia Government or heat treatment to inactivate any virus present at a minimum core temperature 80 °C for at least 125 minutes (or time/temperature equivalent).

Conclusion

- Exotic antigenic variant strains of IBDV and vvIBDV are present in the US and have not been identified in Australia.

- Natural reassortments of vvIBDV are known to occur and infect turkeys (Jackwood et al. 2011).
- The prevalence of IBDV serotype 1 in turkeys is unknown, however in US turkeys it is likely to be very low (Dr. J. Clifford, pers. comm., 29 July 2015).
- Based on the prevalence of IBDV serotypes in chickens, exotic antigenic variant strains of IBDV would be more likely to be circulating than classical strains (Jackwood & Sommer-Wagner 2010).
- IBDV serotype 1 is capable of infecting turkeys, usually subclinically, so they could act as a reservoir of infection for chickens (Giambrone et al. 1978; Owoade et al. 2004; Razmyar & Peighambari 2009).
- In two week old poults experimentally infected with a high dose (10^4 EID_{50}/0.2 ml/poult) of IBDV serotype 1, viral RNA has been detected in turkey breast tissue up to 7 days and in the bursa up to 21 DPI and virus has been isolated from bursal tissue up to 14 DPI (Abdul, Murgia & Saif 2015).

Therefore, the department concluded that further risk assessment of IBDV exotic antigenic variant strains in turkeys was required. IBDV serotype 1 viruses in the US are likely to be exotic antigenic variant strains. Further review of vvIBDV and their reassortments was not considered necessary as these strains are known to be rare in the US.

4.4.4 Risk assessment

The department has previously conducted a risk assessment in relation to the importation of IBDV exotic and antigenic variant strains into Australia in chicken meat. This risk analysis concluded that risk management measures were justified in order to prevent the inadvertent introduction of IBDV.

The pathways by which IBDV in imported poultry meat may be exposed to Australian poultry, identified in the chicken meat IRA, are the same as those which apply to importation of cooked turkey meat from the US. Therefore, the conclusions reached in the chicken meat IRA for the likelihoods of exposure, establishment and spread, and the impacts of the introduction of exotic antigenic variant strains of IBDV into Australia have been carried across to this risk assessment, and the only difference between the two risk assessment outcomes is due to differences in the entry assessment.

Entry assessment

- IBDV serotype 1 is capable of infecting turkeys but its prevalence in turkeys in the US is considered to be very low.
- Based on the prevalence of IBDV serotype 1 in chickens, exotic antigenic variant strains of IBDV would be more likely to be circulating than classical and very virulent strains (Jackwood & Sommer-Wagner 2010).
- Available evidence indicates that if turkeys are infected, IBDV causes subclinical infection in turkeys. Therefore, it would not be recognised at ante-mortem or post-mortem examination (Giambrone et al. 1978), and infected turkeys would therefore not be removed from the export chain.

- IBDV serotype 1 viral RNA has been detected in experimentally infected 2 week old poults (10^4 EID_{50}/0.2 ml/poult) in bursal tissue up to 21 DPI. Viral RNA was also detected in splenic and hepatic tissue up to 14 DPI, in breast muscle and kidney tissue at 7 DPI and in lung and pancreatic tissue at 3 DPI. Virus can be isolated from bursal tissue up to 14 DPI (Abdul, Murgia & Saif 2015).
- It is likely that a viraemia occurs subsequent to infection of turkeys, possibly extending up to 21 DPI (Abdul, Murgia & Saif 2015; Jackwood et al. 2012; Stoute 2012).
- There is no evidence of a carrier state in recovered birds or of vertical transmission (Eterradossi & Saif 2013).
- Despite the lack of a carrier state in recovered birds, the existence of viraemia for up to 21 DPI, combined with the usual production cycle of turkeys, indicates that if flocks are infected with IBDV serotype 1, some birds may be viraemic at the time of slaughter.
- During experimental attempts to produce clinical infection with serotype 1 in turkeys it has been consistently noted that while seroconversion occurs it is difficult and often impossible to isolate the virus or detect the virus using PCR without passing it through various materials (including intestinal and bursal tissue) through embryonated eggs (Giambrone et al. 1978). This indicates a low viral load.
- Infection due to cross-contamination of carcasses at abattoirs was considered a possibility, however, limiting the scope to muscle meat (no whole birds) and good hygiene practices limit contamination from faeces and bursal material that may be infected.
- IBDV is very heat resistant so viable virus would persist in tissues after cooking at the levels described in Section 1.2.2. However, there would be a reduction in viral load as a result of the cooking temperature that would be applied to the turkey meat.

Conclusion:

Based on these considerations, it was estimated that the likelihood of entry of IBDV serotype 1 associated with the importation of cooked turkey meat from the US would be **low.**

Exposure Assessment

- IBDV serotype 1 only causes subclinical infection in turkeys, it causes serious disease in chickens (Giambrone et al. 1978; Owoade et al. 2004).
- IBDV serotype 1 antibodies have been detected in wild birds. However, establishment of IBDV infection has not been reported. Wild birds are considered to have an extremely low likelihood of transmitting IBDV (Ogawa et al. 1998).
- IBDV is highly contagious; it can be transmitted horizontally via faeces, with spread mainly by the faecal-oral route, on fomites or through airborne dissemination of feathers and poultry shed dust (Benton, Cover & Rosenberger 1967; Candelora, Spalding & Sellers 2010; Giambrone et al. 1978).
- Backyard chickens have a high likelihood of being exposed to the waste from domestic consumption of imported turkey meat.

- Available information indicates there are only very small numbers of backyard turkeys kept in Australia, thus limiting potential exposure (Section 1.2.4).
- As IBDV is an extremely hardy virus, any virus present in domestic scraps would likely remain viable up to the time of consumption by backyard poultry (Benton, Cover & Rosenberger 1967; Edgar & Cho 1976).
- The most likely exposure pathway for commercial turkeys and commercial chickens is via fomites, as feed containing meat meal made from waste from imported turkey meat will undergo rendering which will inactivate any IBDV present (Quality Control Unit).

Conclusion: It is extremely unlikely that backyard or commercial turkeys would be exposed to IBDV from imported cooked turkey meat. However, given that backyard chickens have a high likelihood of being exposed to domestic waste, and any virus present in the waste product is likely to be viable, the likelihood of exposure of backyard chickens to IBDV serotype 1 was estimated to be **moderate**.

Estimation of the likelihood of entry and exposure

The likelihood of entry of this agent to Australia and the corresponding likelihood of its exposure to the Australian chicken population was estimated by using the matrix of rules described in Table 1. As the estimate of the likelihood of entry is low and the likelihood of exposure is moderate, the estimation of the likelihood of entry and exposure of IBDV was estimated to be **low**.

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

The most likely outbreak scenario following exposure to IBDV serotype 1 was considered to be establishment in populations of susceptible chickens with horizontal spread likely in more than one state.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible chickens to IBDV serotype 1:

- Chickens are the only species identified as being clinically affected by IBDV serotype 1 (Jackwood et al. 2011; OIE 2008).
- IBD vaccines used in Australia are not protective against variants identified in the US (Ignjatovic, Sapats & Gould 2001).
- IBDV is extremely hardy and highly contagious; it can be transmitted horizontally via faeces, with spread mainly by the faecal-oral route on fomites or through airborne dissemination of feathers and poultry shed dust (Benton, Cover & Rosenberger 1967; Candelora, Spalding & Sellers 2010; Giambrone et al. 1978).
- There is no evidence of a carrier state in recovered birds or of vertical transmission (Eterradossi & Saif 2013).
- Backyard chickens tend to be older birds, which are refractory to infection (Eterradossi & Saif 2013).
- Spread through a commercial poultry flock is likely to be rapid and while presentation may vary due to strain, maternal antibody to Australian strains may delay detection (Biosecurity Australia 2008).

- IBDV is an extremely hardy virus and will persist in the environment. Attempts to eradicate the disease would be difficult (Biosecurity Australia 2008).

Conclusion: Based on these considerations, the likelihood of establishment and spread of IBDV serotype 1 through the Australian chicken population was estimated to be **low**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect impacts of IBDV serotype 1 were estimated at the national, state or territory, district/region and local levels. Adverse effects were evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of IBDV serotype 1:

Direct effects

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

- Chickens are the only species identified as being clinically affected by IBDV serotype 1 (Jackwood et al. 2011; OIE 2008).
- IBD is an acute and highly contagious viral disease causing varying degrees of mortality and immunosuppression in chickens (Lukert & Hitchner 1984; OIE 2008).

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

- There are no known effects on the living environment—although IBDV serotype 1 antibodies have been detected in wild birds, establishment of IBDV infection and clinical disease shown to be due to IBDV infection has not been reported (Ogawa et al. 1998).

Indirect effects

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

- Exotic antigenic variant forms of IBDV and vvIBDV are notifiable in Australia.
- A detection would result in destruction of the flock and increased surveillance and monitoring. Vaccination may be considered.

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

- Infection in the commercial poultry industry would likely require inputs from the biotechnology industry in the development and deployment of new vaccines. Commercial customers may be affected by shortages of chicken meat and eggs due to direct losses of stock and quarantine movement restrictions.
- There would be no effect on consumer demand.

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

- There would be no impact on international trade.

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

- There would be no discernible effect on the environment.

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

- There would be no discernible effect on communities outside affected areas. Affected areas may experience minor issues while movement restrictions are in place.

Conclusion for overall direct and indirect effects: Based on this information the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **moderate**.

Consequence assessment

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3 to obtain an estimation of likely consequences.

Therefore, the likelihood of establishment and/or spread (low) when combined with the estimate of the overall effect of establishment and/or spread (moderate) resulted in **low** likely consequences.

4.4.5 Risk estimation and evaluation

Risk estimation is the integration of likelihood of entry and exposure, and likely consequences of establishment and/or spread to derive the risk associated with entry, exposure, establishment and/or spread of IBDV introduced by imported cooked turkey meat into Australia.

Using Table 4, the likelihood of entry and exposure (low) was combined with the likely consequences of establishment and/or spread (low), to give a risk estimation of **very low**.

Therefore, as the unrestricted risk estimate achieves Australia's ALOP, no specific risk management was considered necessary for this agent.

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4.5 Multicausal enteric syndromes

4.5.1 Background

A number of enteric syndromes occur in turkey poults that are referred to by various names and involve infectious intestinal diseases of young turkeys. These include poult enteritis mortality syndrome (PEMS), poult enteritis syndrome (PES), and poult enteritis complex (PEC) (Barnes, Guy & Vaillancourt 2000; Jindal et al. 2010). Rarely has a single agent been identified as the sole causative factor of these enteric syndromes. Although a potential pathogen has been identified, other pathogens were usually present, suggesting involvement in the disease process (Barnes & Guy 2003).

Despite the isolation of a number of agents from cases of enteric disease, many of these agents have also been detected in otherwise healthy turkey flocks (Day et al. 2010).

These syndromes are not OIE listed and are not notifiable in Australia or subject to official control or eradication.

4.5.2 Technical information

Agent properties

No single agent has been identified as the cause of enteric disease. Agents isolated from birds described as suffering from PEMS include coronavirus, astrovirus, reovirus and enteropathogenic *E. coli* (Heggen-Peay et al. 2002; Koci, Seal & Schultz-Cherry 2000; Pakpinyo et al. 2002; Schultz-Cherry, King & Koci 2001; Yu et al. 2000). Agents isolated from turkeys described as suffering from PES include turkey astrovirus, rotavirus, reovirus, adenovirus, Salmonella, *E. coli* and Enterococcus (Jindal et al. 2010; Jindal et al. 2009). PEC is a term used for a group of multifactorial diseases that includes coronaviral enteritis, malabsorption syndrome, maldigestion syndrome, runting and stunting syndrome of turkeys and turkey viral enteritis (Barnes, Guy & Vaillancourt 2000; Jindal et al. 2010).

Two viruses (coronavirus and reovirus) identified as hazards in this draft review are covered in detail in separate sections.

Epidemiology

Enteric syndromes generally occur as transmissible, infectious diseases of young turkeys less than six weeks of age. The increased susceptibility of young turkeys to enteric disease has been described as due to an immature intestinal epithelium in the first weeks of development which has a reduced absorptive capacity, making it vulnerable to various infectious agents (Ismail, Tang & Saif 2003; Moura-Alvarez et al. 2013).

Interactions between the intestinal tract and other body systems can affect the severity and progress of the disease, making it difficult to determine the role of specific pathogens in enteric disease (Pantin-Jackwood 2013).

Transmission of agents is primarily faecal-oral and prevention is based on eliminating the infectious agents from contaminated premises thereby preventing introduction into flocks (Barnes, Guy & Vaillancourt 2000).

Numerous viruses associated with enteric disease are known to be circulating in turkey flocks in the US, in both healthy and sick turkeys (Day et al. 2010; Pantin-Jackwood et al. 2008; Pantin-Jackwood

et al. 2007). A survey of enteric viruses in healthy turkey flocks across the US identified astrovirus, reovirus and rotavirus as often being present as concomitant infections. There was no clear pattern of virus distribution but it would appear that enteric viruses are widespread in poultry throughout the US (Pantin-Jackwood et al. 2008).

A study of Brazilian turkey flocks identified the presence of astrovirus, coronavirus, rotavirus and adenovirus. They occurred more commonly in turkeys in the growing phase (1-4 weeks) compared to the finishing phase (5-18 weeks), and flocks exhibiting clinical signs of intestinal disease had a higher rate of positive samples than healthy flocks (Moura-Alvarez et al. 2013).

Pathogenesis

The basic pathogenesis involves damage to the intestinal mucosa, usually by one or more viruses infecting enterocytes, followed by inflammation and subsequent proliferation of and colonisation by intestinal bacteria and protozoa (Barnes, Guy & Vaillancourt 2000).

The mechanisms by which infectious agents produce enteric disease include increased or decreased motility, alterations in intestinal permeability or osmotic gradients, malabsorption, and changes in the number of mature intestinal epithelial cells present. Bacterial toxins and some enteric viruses are also known to stimulate secretion of intestinal crypt cells beyond the absorptive capacity of the intestinal epithelial cells. Agents that produce diarrhoea usually induce a combination of these mechanisms (Barnes & Guy 2003).

The only reliable method to reproduce the clinical signs of enteric syndromes in experimental situations is oral inoculation with crude preparations of intestinal contents from naturally infected birds (Day et al. 2010).

Diagnosis

Clinical signs

Enteric syndromes are characterised by signs such as diarrhoea, growth depression, retarded development, impaired feed utilisation and nutritional deficiencies. Mortality is generally low however immune dysfunction is common and increases susceptibility of the flock to other infectious diseases (Barnes, Guy & Vaillancourt 2000).

Testing

Traditionally, diagnosis of viral enteric infections in turkeys has been made by electron microscopy (EM), immunofluorescent assay and genome electropherotyping to detect and identify the viruses, and ELISA to detect antibodies (Pantin-Jackwood et al. 2007).

Recent developments in molecular diagnostics (RT-PCR) for detecting enteric viruses provide many advantages over the traditional diagnostic methods. These include greater sensitivity and specificity, detection of multiple viruses in one sample, no need for virus propagation and the ability to test a large number of samples quickly (Pantin-Jackwood et al. 2008). Molecular-based diagnostic tests have been commercialised for turkey coronavirus, turkey astrovirus-2, reovirus and adenoviruses (Pantin-Jackwood et al. 2007). More recently molecular diagnosis of coronavirus and rotavirus was reported and metagenomic studies have been used for the establishment of the complete intestinal DNA profile of enteric pathogens (Day et al. 2010; Moura-Alvarez et al. 2013).

However, identification of agents that are present in birds with enteric disease does not ensure that the agent isolated is the cause of the disease or is just an opportunistic pathogen or a normal commensal agent (Moura-Alvarez et al. 2013; Pantin-Jackwood et al. 2008).

Control

Control of enteric diseases requires an integrated approach, incorporating drug treatment, supportive therapy and management components (Barnes, Guy & Vaillancourt 2000). Good management practices can help reduce or eliminate exposure of young birds to enteric viruses, but the ubiquity and genetic variation of the many enteric viruses make it difficult or impractical to keep commercial flocks free of infection (Pantin-Jackwood 2013).

4.5.3 Current biosecurity measures

Currently, there are no specific biosecurity measures for this group of agents in turkeys. Only canned or retorted turkey meat products that meet specific temperature and time requirements during the manufacturing process are permitted for import into Australia at this time.

4.5.4 Conclusion

- Due to the multi-agent nature of enteric syndromes it is not feasible to propose risk management measures.
- Turkey coronavirus and emerging strains of reovirus have been considered in separate chapters as potential agents of concern.

Therefore, the department concluded that further risk assessment of multicausal enteric syndromes was not required.

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4.6 Mycoplasma iowae

4.6.1 Background

There are many species of Mycoplasmas and *Mycoplasma iowae* (*M. iowae*) is one of the more important Mycoplasma species involved in poultry disease. *M. iowae* is primarily a pathogen of turkeys, causing embryo mortality and reduced hatchability; however, infections and pathology can also occur in chickens and other avian species (Bradbury & Kleven 2003; Catania et al. 2012).

M. iowae is widely distributed, with evidence of its occurrence in Asia, Europe, India, Japan, Pakistan and North America (Al Ankari & Bradbury 1996).

M. iowae is not OIE-listed, and is not notifiable in Australia or subject to official control or eradication. It has not been isolated in Australia.

4.6.2 Technical information

Agent properties

M. iowae belongs to the family Mycoplasmataceae, is coccobacillary in form with some pleomorphism and no cell wall (Bradbury & Kleven 2003). There are many different strains of *M. iowae* as well as marked within-species antigenic variation (Rhoades 1984).

Like other avian mycoplasmas, *M. iowae* is presumed to be susceptible to common disinfectants (Bradbury & Kleven 2003). However a study of *M. iowae* in a poultry housing environment showed that the organism can survive for varying lengths of time depending on the material tested, but for at least six days on feathers. *M. iowae* proved to be hardier in the environment than other avian mycoplasmas in this study (Christensen et al. 1994).

In a study to determine the temperature sensitivity of various avian mycoplasmas, they were heated in a bouillon culture with full mycoplasmacidal effect after 6 hours at 45 °C, 150 minutes at 50 °C, 90 minutes at 52 °C and 30 minutes at 55 °C (Goren 1978). Although *M. iowae* was not included in this study, its sensitivity to heat can be presumed to be similar. In another study *M. iowae* remained viable in turkey semen after 48 hours at 40 °C (Shah-Majid & Rosendal 1986b).

Epidemiology

Turkeys and chickens are the natural hosts of *M. iowae*, although it occurs more often in turkeys (Bradbury et al. 1990). The organism has also been isolated from geese, grey partridges, Amazon parrots, and other wild birds including starling, cormorants, heron, wood pigeons and an eider duck in a zoo (Al Ankari & Bradbury 1996; Bradbury & Kleven 2003; Catania et al. 2014).

Vertical transmission following infection of eggs in the oviduct is an important mode of spread. Infected embryos that survive through to hatch remain in the population to infect the following generation (Wood & Wilson 2013). The rate of vertical transmission appears to decline with age, decreasing when the hens are in their second laying season, and varies with individuals in a flock. Some birds lay no or few infected eggs, while others lay many infected eggs (Al Ankari & Bradbury 1996; Bradbury et al. 1990).

Venereal transmission with infected tom semen and artificial insemination is an important mode of lateral transmission and initiates oviduct infection within a breeder hen (Shah-Majid & Rosendal

1986a). The rate of infection is probably increased by semen pooling, a process commonly used commercially. Lateral spread via infected faeces within the hatchery and during brooding is possible but considered a less significant route of infection (Al Ankari & Bradbury 1996; Wood & Wilson 2013). Aerosol transmission does not appear to occur in contrast to other important avian mycoplasmas (Wood & Wilson 2013).

Documentation of the prevalence of *M. iowae* is complicated by the poor serological response to infection, even in persistently infected birds, and the difficulty in isolating the organism, especially from live adult birds (Bradbury et al. 1990). In pooled serum samples taken from 122 commercial turkey flocks in the US, only 18% had antibodies against *M. iowae* (Cummins & Reynolds 1990). Also, both false positive and false negative results are possible in serological testing for mycoplasma (Bradbury 2001; Wood & Wilson 2013).

Transmission in turkey meat

The organism is capable of persistence in the alimentary and reproductive tracts and has been isolated from the joints of infected birds. Therefore, there is potential for carcass contamination with *M. iowae*.

Pathogenesis

Unlike most avian mycoplasmas which are generally localised in the respiratory tract, *M. iowae* exhibits a predilection for the digestive tract. Day-old turkey poults orally inoculated with *M. iowae* developed intestinal infection, and became persistent faecal shedders of the organism (Mirsalimi, Rosendal & Julian 1989). *M. iowae* was also recovered from the kidney, spleen, trachea, lung and thoracic air sac suggesting *M. iowae* is invasive and/or can cause infection via inhalation (Shah-Majid & Rosendal 1987).

Diagnosis

Clinical signs

Clinical signs of disease due to *M. iowae* are not commonly observed in natural infections of adult turkeys (Al Ankari & Bradbury 1996; Bradbury et al. 1990; Trampel & Goll 1994). The most common indication of infection is a reduction in hatchability of 2 to 5% of eggs in turkey flocks due to embryonic deaths (Bradbury & Kleven 2003; Wood & Wilson 2013). However, flocks of young turkeys can develop *M. iowae* leg abnormalities. These include hock swelling, lameness, valgus deformities, splay legs, curling of the toes and vertebral chondrodystrophy with signs first seen between one and four weeks of age (Ley et al. 2010; Trampel & Goll 1994).

M. iowae has been cultivated from the cloaca and small intestine of turkeys in flocks affected with mild respiratory disease, followed by abnormal leg development and bone weakness between four and six weeks of age (Catania et al. 2012).

Clinical signs following experimental infection in turkey poults varied with the age of the bird, route of inoculation and the strain of *M. iowae* used. Turkeys infected in ovo failed to hatch or were stunted and died within three weeks. Those infected at one day of age were stunted, and developed poor feathering and leg abnormalities such as ruptured tendons, swollen hocks and splayed legs (Bradbury, Ideris & OO 1988).

Pathology

There is no gross pathology evident when mature birds are infected. In young turkeys showing clinical signs there are gross signs of chondrodystrophy—legs bowed and shortened, and enlarged hock joints. Vertebral columns may be shortened with deformities both of the spine and ribs (Ley et al. 2010).

Testing

Culture remains the most common diagnostic test for *M. iowae* however, it is time-consuming and complicated, requiring two to three weeks, and sensitivity is poor (Cai et al. 2008; Wood & Wilson 2013).

PCR tests with high sensitivity and specificity have been developed and these have proven more efficient in detecting positive birds in the field (Cai et al. 2008; Wood & Wilson 2013).

There is no reliable serological test available due to antigenic variability and a weak serological response to infection (García et al. 1997). Flock eradication programs rely on culture and PCR testing.

Treatment

As for other avian mycoplasmas, *M. iowae* is susceptible to antibiotics such as tetracyclines and tylosin that act on sites other than the cell wall. However there is evidence that *M. iowae* is less susceptible to other antibiotics, such as macrolides, than the other avian mycoplasmas (Gautier-Bouchardon et al. 2002). Antibiotics given to adult birds will reduce the spread of infection and the losses due to embryonic mortality but will not entirely eliminate an infection from a flock (Wood & Wilson 2013).

4.6.3 Current biosecurity measures

There are current biosecurity measures in place for this disease in the *Conditions for the importation from approved countries of fertile eggs (domestic turkey)*. The requirement is to demonstrate flock freedom from infection within 21 days before export by culture of semen of all toms used for the artificial insemination of the source flock. In addition, information from regular monitoring of the source flock must demonstrate freedom from infection at all times or all females in the source flock must be tested by culture and found to be free of *M. iowae*.

4.6.4 Conclusion

- *M. iowae* is present in the US but not in Australia. *M. iowae* has caused clinical disease in turkeys in the US.
- The organism is capable of persistence in the alimentary and reproductive tracts and has been isolated from the joints of infected birds. It may be present if remnants of these organs remain after processing or if the carcass is contaminated during processing.
- *Mycoplasma* spp are inactivated by heat (Goren 1978). Cooking at the temperature described in Section 1.2.2 is sufficient to address any biosecurity concerns.

Therefore, the department concluded that further risk assessment of *Mycoplasma iowae* was not required.

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4.7 Turkey coronavirus

4.7.1 Background

Turkey coronavirus (TCV) causes acute enteritis in turkeys and is characterised by watery diarrhoea, inappetence, weight loss and failure to thrive (Guy 2013; Ismail, Tang & Saif 2003). In older turkeys, TCV also causes a drop in egg production (Awe et al. 2013). TCV enteritis was first described by Peterson and Hymes (1951) as mud fever in poults, similar to bluecomb disease in chickens (although these are separate diseases), and later became known as bluecomb disease of turkeys (Guy 2003). The causative agent of TCV enteritis was not identified until 1973 (Panigrahy, Naqi & Hall 1973; Ritchie et al. 1973). Other names for TCV enteritis include transmissible enteritis and coronaviral enteritis (Guy 2013).

TCV is often associated with poult enteritis and mortality syndrome (PEMS), a multifactorial infectious syndrome which typically affects poults between one and three weeks of age (Barnes, Guy & Vaillancourt 2000).

Turkey coronavirus is not OIE-listed, and is not notifiable in Australia or subject to official control or eradication. It has not been isolated in Australia.

4.7.2 Technical information

Agent properties

TCV is a linear, non-segmented, positive sense, single-stranded RNA enveloped virus (Guy 2013). TCV belongs to the family Coronaviridae, species avian coronavirus (ICTV 2014). TCV is a type III coronavirus and it shares a high degree of sequencing identity with avian infectious bronchitis virus (IBV) (Gomaa et al. 2008; ICTV 2014).

Coronaviruses are quite sensitive to heat and are inactivated at 56 °C for 15 minutes, and 60 °C for 30 minutes for samples containing protein (Deshmukh & Pomeroy 1974; Jackwood & de Wit 2013). TCV has been shown to be stable at pH 3.0 at 22 °C for 30 minutes and resistant to 50 °C for one hour with 1 M magnesium sulphate (Guy 2013). TCV can remain viable in intestinal tissues stored at minus 20 °C or lower for more than five years (Guy 2013). No TCV was detected in media supplemented with 5% foetal calf serum after ten days stored at 21.6 °C ± 1.4 °C or after 40 days stored at 4.1 °C ± 1.6 °C indicating a longer survival time at lower temperatures (Guionie et al. 2013).

Treatment with chloroform at 4 °C for ten minutes readily inactivates the virus (Guy 2013). Saponified cresol and formaldehyde are effective disinfectants for elimination of TCV from contaminated buildings (Patel, Gonder & Pomeroy 1977).

Epidemiology

In the US, TCV was identified as causing enteritis in turkeys 30 years ago and since then has been isolated from turkeys in Brazil, Canada, France, Italy, Poland and the United Kingdom (Cavanagh 2005; Cavanagh et al. 2001; Dea & Tijssen 1988; Domanska-Blicharz et al. 2010; Lin et al. 2002; Maurel et al. 2011; Teixeira et al. 2007). The prevalence of TCV in the US was assessed as 30% in 2005 by virus isolation and antibody levels in sera (Cavanagh 2005). A survey by the United States Animal Health Association of US turkey production professionals identified an increase in the prevalence of disease caused by TCV between 2008 and 2012 (Clark, Kromm & Bailey 2012).

Turkeys are believed to be the only natural host for TCV. Pheasants, seagulls, coturnix quail and hamsters cannot be infected with TCV (Guy 2013). Experimentally infected chickens show seroconversion and virus and viral antigens can be isolated from intestinal contents and cloacal bursa, however, these chickens show no clinical signs of disease (Guy 2013). All ages of turkeys can be affected however, disease is more common in poults during the first few weeks of age, causing enteritis and mortality. Disease in older turkeys causes impaired growth and poor feed conversion (Guy et al. 1997; Guy et al. 2002; Ismail, Tang & Saif 2003).

TCV is highly infectious and primarily spread by the faecal-oral route. Virus is shed in the faeces of infected poults and can be shed as early as one day post inoculation and up to several weeks after recovery from clinical signs (Breslin et al. 2000; Gomaa et al. 2009b). Vertical transmission of TCV has not been demonstrated however poults may be infected in the hatchery via fomites from infected personnel or equipment (Guy 2013). Domestic house flies can transmit TCV in their faeces up to nine hours after consuming infected material, and are capable of infecting turkey poults three hours after consuming the material (Calibeo-Hayes et al. 2003).

A report of disease in turkeys resembling mud fever was recorded in a New South Wales Department of Agriculture annual report of 1955. No testing was performed and no subsequent reports indicated that the disease was caused by TCV (Department of Agriculture 1955).

Pathogenesis

TCV replicates in the enterocytes lining the apical portions of small intestinal villi and in the epithelium of the bursa of Fabricius (Guy 2013; Naqi, Panigrahy & Hall 1972). One study detected virus in the oviduct of 2 out of 24 experimentally infected turkey hens however, no microscopic lesions were observed in the oviduct (Awe et al. 2013).

Turkey poults inoculated orally with TCV developed depression and diarrhoea with markedly enlarged intestines and pale and flaccid intestinal walls. Microscopically there was mild multifocal enteritis and changes in villus height and crypt depth (Gomes et al. 2010).

Diagnosis

As other enteric pathogens can cause similar lesions and clinical signs, laboratory diagnosis is required for confirmation.

Clinical signs

TCV enteritis presents as sudden onset anorexia followed by depression, dehydration, huddling, loss of condition, foetid, watery diarrhoea and sour crop (Gomaa et al. 2009b; Peterson & Hymas 1951). Morbidity often approaches 100% with varying mortality rates which are highest in younger birds (Cavanagh et al. 2001; Peterson & Hymas 1951). In adult birds there is often decreased weight gain and a drop in egg production (Awe et al. 2013).

Pathology

Birds may be emaciated and the duodenum and jejunum are typically pale and flaccid, the caeca are distended, and the intestines are filled with watery contents (Adams, Ball & Hofstad 1970; Guy 2013). The bursa of Fabricius may be atrophied (Guy 2013).

Histopathologic lesions described are not considered pathognomonic for TCV as they show general damage to the intestines and caeca that can be observed in other enteric diseases (Adams, Ball & Hofstad 1970). Damage to the villous epithelium and hyperactivation of intestinal glands at the ileo-

caecal junction has been identified with desquamated epithelial cells and mucous exudates in the lumen (Teixeria et al 2007).

In experimentally infected 18 day old poults, histologic lesions were observed in the duodenum, ileum and caeca over a 4 day period, after which time they gradually regressed, reaching normality at 21 days post infection. Goblet cells decreased rapidly post inoculation and villi stunting was obvious. The epithelium was separated from the lamina propria by oedema and infiltrated by monocytes and heterophils (Adams, Ball & Hofstad 1970).

Testing

Diagnosis of TCV can be made using virus isolation, electron microscopy (EM), serology, detection of viral antigens or detection of viral RNA by PCR. Viral isolation is achieved by inoculating TCV into the amniotic cavity of embryonated turkey eggs due to the inability to grow TCV in cell culture (Breslin et al. 2000; Teixeira et al. 2007).

RT-PCR has been shown to be a rapid, highly sensitive and specific method for TCV detection (Breslin et al. 2000; Chen et al. 2010; Jindal et al. 2010; Loa et al. 2006). EM has been used to visualise a number of different viruses that may be present however, identification of typical coronavirus particles can be difficult (Guy 2013; Jindal et al. 2010).

ELISAs have been developed for detection of TCV antibodies in sera and have been shown to be highly sensitive and specific as well as effective when processing large numbers of clinical samples (Gomaa et al. 2009a; Guy et al. 2002; Loa et al. 2000).

Historically diagnosis has been by virus isolation and/or detection of viral antigens in tissues by direct and in-direct fluorescent antibody procedures. These procedures are expensive and time consuming and often lack sensitivity (Breslin et al. 2000; Guy et al. 2002; Loa et al. 2006).

4.7.3 Current biosecurity measures

There are current biosecurity measures in place for this disease in the *Conditions for the importation from approved countries of fertile eggs (domestic turkey)*. The source flock must be free from signs of disease in the 90 days prior to egg collection.

Only canned or retorted turkey meat products and turkey meat based flavours that meet specific temperature and time requirements during the manufacturing process are permitted into Australia at this time.

4.7.4 Conclusion

- TCV is present in the US but has not been identified in Australia.
- Coronavirus replicates in the small intestine, caeca and the bursa. It has also been detected in the oviduct. It may be present if remnants of these organs remain after processing or if the carcass is contaminated during processing.
- Coronavirus is inactivated by heat (Biosecurity Australia 2008; Deshmukh & Pomeroy 1974; Jackwood & de Wit 2013). Cooking at the level described in Section 1.2.2 is sufficient to address any biosecurity concerns.

Therefore, the department concluded that further risk assessment of turkey coronavirus was not required.

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4.8 Turkey viral hepatitis

4.8.1 Background

Turkey viral hepatitis (TVH) is highly contagious and causes inflammation of the liver and pancreas in turkeys (Hauck et al. 2013; Snoeyenbos & Basch 1960). TVH was first described in 1959 by groups in Canada and the US that were investigating increased mortality in turkey poults (Mongeau et al. 1959; Snoeyenbos, Basch & Sevoian 1959).

The disease is often subclinical becoming overt when the birds are stressed, resulting in varying rates of illness and death (Honkavuori et al. 2011).

The agent causing TVH has only recently been identified as a picornavirus using pyrosequencing and RT-PCR (Honkavuori et al. 2011). This research supports previous studies which identified a picorna-like virus from TVH affected turkeys (Klein et al. 1991; MacDonald et al. 1982).

TVH is not OIE-listed and is not a notifiable disease in Australia or subject to official control or eradication. TVH has not been reported in Australia.

4.8.2 Technical information

Agent properties

Picornaviridae are a small, non-enveloped family of viruses with a positive sense, single stranded RNA genome (Honkavuori et al. 2011). There are currently 12 genera of Picornaviridae with analysis showing that TVH is distinct from the other genera (Honkavuori et al. 2011).

An isolate of TVH virus survived up to 6 hours at 60 °C and 14 hours at 56 °C. However when exposed to high pH (pH 12) the same isolate was inactivated after one hour at 30 °C while remaining viable at pH 2 for the same time and temperature (Tzianabos & Snoeyenbos 1965b). TVH virus remained viable stored at minus 20 °C for 365 days and at 60 °C for 5 hours (Snoeyenbos, Basch & Sevoian 1959). The virus remained viable in a water bath after 14 hours at 56 °C, but was non-viable after 16 hours (Snoeyenbos, Basch & Sevoian 1959).

TVH virus is resistant to chloroform, creoline, ether, merthiolate and phenol but is inactivated by formalin after six hours at 30 °C (Tzianabos & Snoeyenbos 1965b).

Epidemiology

The true distribution of TVH is not known as the disease is usually subclinical and there are no diagnostic serological tests available (MacDonald et al. 1982). TVH has been isolated in Britain, Canada, Italy and the US (Klein et al. 1991; MacDonald et al. 1982; Mongeau et al. 1959; Snoeyenbos, Basch & Sevoian 1959). The disease has been observed on a regular basis in Californian turkey flocks in the past 12 years (Hauck et al. 2013).

Turkeys are the only natural host for TVH (Guy 2013). Day old chicks and mice were shown to be refractory to infection via inoculation with liver material from infected poults (Snoeyenbos, Basch & Sevoian 1959). White Pekin ducklings, coturnix quail and ring-necked pheasants were also refractory to infection (Tzianabos & Snoeyenbos 1965a).

Originally the disease was thought to occur only in poults younger than 5 weeks of age (Mongeau et al. 1959; Snoeyenbos & Basch 1960). A recent retrospective study of TVH cases identified an age

range of 7 to 61 days however, most cases occurred between 3 and 5 weeks of age (Hauck et al. 2013).

The primary route of infection is considered to be faecal-oral because the virus is isolated from faeces and readily spreads through both direct and indirect contact with affected poults (Andral et al. 1990; Guy 2013).

As a viraemia occurs for a considerable period of the infection, and as the virus has been isolated from an ovarian follicle, vertical transmission is considered possible (Snoeyenbos & Basch 1960). One proposed scenario is that a high number of parent flocks are infected and pass the infections to their progeny, of which only a small number develop lesions and are recognised as infected (Hauck et al. 2013). TVH does not appear to be able to infect embryos through contaminated egg shells (Snoeyenbos & Basch 1960).

Pathogenesis

Characteristic lesions are seen in the liver and pancreas and include focal necrosis of hepatocytes and acinar cells with varying stages of inflammation including giant cells (Hauck et al. 2013).

Lesions were evident as early as four days post inoculation in birds injected with the virus and five days in birds that were in contact with infected poults (Snoeyenbos, Basch & Sevoian 1959). Viral isolation has not been successful at greater than 28 days post inoculation (Tzianabos & Snoeyenbos 1965a).

Diagnosis

Clinical signs

Anorexia, depression, diarrhoea and weight loss may be apparent, however, these signs can be attributable to enteritis which is commonly diagnosed in turkey poults in the US (Honkavuori et al. 2011).

Mortality rates between 1 and 25% in field cases have been reported however, the disease is often subclinical (Snoeyenbos & Basch 1960; Snoeyenbos, Basch & Sevoian 1959).

Pathology

A presumptive diagnosis is often made at post-mortem by the presence of characteristic lesions (gross and/or microscopic) in the liver and pancreas or both (Hauck et al. 2013). Virus isolation may be used for a definitive diagnosis (Guy 2013).

In early experiments, virus isolation in older birds frequently failed and liver lesions were relied upon for diagnosis (Snoeyenbos & Basch 1960). On post-mortem, gross lesions identified included multifocal to coalescing, circular to irregular, tan to pink/grey depressions of the liver and occasionally the pancreas (Klein et al. 1991). Pancreatic lesions are observed usually where there are significant and extensive liver lesions present; however, on rare occasions the pancreas may be the only organ with gross abnormalities (Snoeyenbos & Basch 1960). Similar lesions in the liver may also be caused by other disease agents including avian adenoviruses, *Histomonas meleagridis, Pasteurella multocida,* reovirus and *Salmonella* spp. (Guy 2013).

In a review of studies done in the US, microscopic lesions in the liver were identified in almost all cases, and in the pancreas in 46% of cases (Hauck et al. 2013). Lesions in the liver consisted of focal necrosis of hepatocytes and varying stages of inflammation. Occasionally biliary hyperplasia and

giant cells or syncytia were found in livers of experimentally infected poults (Hauck et al. 2013; Klein et al. 1991). Microscopic lesions of the pancreas were sometimes present and consisted of focal necrosis of acinar cells and varying stages of inflammation (Hauck et al. 2013). The same review found that 72% of cases (from a sample size of 76) had gross liver lesions (Hauck et al. 2013).

Testing

Virus isolation has been achieved from the liver, bile, blood, spleen, kidney, faeces and ovarian follicular contents; however, liver is the preferred sample (Guy 2013; Snoeyenbos & Basch 1960). TVH virus cannot be grown in cell culture and must be inoculated into five to seven day old embryonating chicken eggs via the yolk sac (Guy 2013; Tzianabos & Snoeyenbos 1965a).

There are currently no serological tests available for the diagnosis of TVH (Guy 2013).

4.8.3 Current biosecurity measures

There are current biosecurity measures in place for this disease in the *Conditions for the importation from approved countries of fertile eggs (domestic turkey)*. The source flock must be certified as free from signs of TVH for the 90 days prior to egg collection.

4.8.4 Conclusion

- TVH is present in the US but has not been diagnosed in Australia.
- TVH virus has been isolated from liver, bile, blood, spleen, kidney, faeces and ovarian follicular contents which are generally removed from turkey carcasses at slaughter. However, contamination of the carcass could occur during processing and some remnants of these tissues may remain in the carcass after evisceration.
- TVH may not be inactivated by cooking at the levels described in Section 1.2.2.
- TVH is not an OIE-listed disease and there are no recommendations in the Code on measures for safe trade.

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

4.8.5 Risk assessment

Entry Assessment

- TVH is present in the US (Guy 2013).
- TVH is usually subclinical and there are currently no diagnostic serological tests available so the prevalence is not known (Guy 2013).
- Infection occurs early in the bird's life (three to five weeks) and birds may be culled before processing age. However, adult birds can be infected without showing clinical signs (Hauck et al. 2013). TVH causes inflammation of the liver in poults usually aged between three and five weeks (Hauck et al. 2013; Snoeyenbos & Basch 1960). Clinical signs are not usually observed in poults older than five weeks and it is unlikely that the disease would be picked up at ante-mortem inspection (Snoeyenbos & Basch 1960).

- TVH virus has been isolated from the liver, bile, blood, spleen, kidney, faeces and ovarian follicular contents of infected birds (Guy 2013; Snoeyenbos & Basch 1960). These are generally removed from the carcass at slaughter; however, remnants may remain and contamination may occur at processing.
- The virus survived three to six hours at 60 °C (Tzianabos & Snoeyenbos 1965b). It is likely that the cooking described in Section 1.2.2 will reduce the burden of any TVH virus present due to post-processing contamination.

Conclusion: based on this information, the likelihood of importation of TVH associated with the cooked turkey meat from the US was estimated to be **low**.

Exposure Assessment

- Turkeys are the natural host for TVH and other species are refractory to infection (Guy 2013; Snoeyenbos, Basch & Sevoian 1959). Therefore backyard chickens and wild birds will not become infected through exposure to the waste from domestic consumption of imported turkey meat.
- The only exposure pathway for commercial turkeys is via feed containing meat meal made from waste from imported turkey meat. Rendering will inactivate any TVH virus present.
- It is possible that backyard turkeys may be exposed to the waste from domestic consumption of imported turkey meat. However, given the very limited population of backyard turkeys in Australia this exposure pathway was considered to have a low likelihood.

Conclusion: based on this information, the likelihood of exposure of Australian turkeys to TVH associated with cooked turkey meat from the US was estimated to be **very low**.

Estimation of the likelihood of entry and exposure

The likelihood of entry of this agent to Australia and the corresponding likelihood of its exposure to the Australian turkey population was estimated by using the matrix of rules described in Table 1. As the estimate of the likelihood of entry was low and the likelihood of exposure was very low the estimation of the likelihood of entry and exposure of TVH was estimated to be **very low**.

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

The most likely outbreak scenario following exposure to TVH was considered to be limited establishment in populations of susceptible turkeys, most likely confined to one state.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible turkeys to TVH.

- Turkeys are the only affected species and other poultry species and wild birds are refractory to infection.
- The agent is spread by the faecal-oral route. Infection may be perpetuated on-site if a population of turkeys is maintained but is unlikely to spread between premises unless there is direct contact.
- Commercial turkey operations in Australia practice all-in, all-out farming and cleanout and disinfect housing between batches. This will limit the establishment of the infection on these sites.

Conclusion: based on these considerations, it was estimated that the likelihood of establishment and spread of TVH through the Australian turkey population was **very low**.

Determination of the effects resulting from the outbreak scenario

For the most likely outbreak scenario, the direct and indirect impacts of TVH were estimated at the national, state or territory, district/region and local levels. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of TVH.

Direct effects

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

- Turkeys are the natural hosts of TVH and other species are refractory to infection.
- Although TVH may cause widespread morbidity and mortality, it is usually subclinical and most often detected at post-mortem.

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

- **There are no known effects on the living environment—wild birds are unlikely to become infected or show deleterious effects.**

Indirect effects

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

- TVH is not notifiable in any Australian jurisdiction and there are no control, monitoring or surveillance programs in place.

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

- If TVH was detected in Australian backyard turkeys, it is unlikely that there would be any effects on domestic industry or trade.
- There would be no effects on consumer demand.

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

- There would be no impact on international trade.

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

- There would be no discernible effects on the environment.

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

- There would be no discernible effects on communities.

Conclusion for overall direct and indirect effects: based on the geographic level and magnitude of effects, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **very low** from Table 2. The effect is likely to be minor to directly affected parties and indiscernible at any other level.

Consequence assessment

The estimate of the overall effect associated with the outbreak scenario (very low) was combined with the likelihood of establishment and/or spread for the scenario (very low) using Table 3 to obtain an estimation of likely consequences **negligible**.

4.8.6 Risk estimation and evaluation

Risk estimation is the integration of likelihood of entry and exposure, and likely consequences of establishment and/or spread to derive the risk associated with entry, exposure, establishment and/or spread of TVH introduced by imported cooked turkey meat into Australia.

Using Table 1, 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 was considered necessary for this agent.

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5 Requirements for the importation of cooked turkey meat from the US

5.1.1 Eligibility

Importation under these conditions is restricted to cooked turkey meat from the US only.

5.1.2 Documentation

A written application to import cooked turkey meat must be lodged with the department before any import can occur.

Each consignment must be accompanied by:

a) a valid import permit

b) an official veterinary health certificate in accordance with 'Model veterinary certificates for international trade in live animals, hatching eggs and products of animal origin' as described in Chapter 5.10 of the Code.

The veterinary certificate must provide details of:

- the packaging of the meat including details of the labelling
- the addresses and veterinary approval numbers of establishments at which the turkeys from which the meat was derived were slaughtered
- the facility at which it was prepared
- the establishment at which it was stored before export
- the names and addresses of the exporter and the consignee.

An Official Government Veterinarian means a veterinarian authorised by the competent authority of the US to perform certain official tasks associated with animal health and/or public health, inspections of commodities, and when appropriate, to certify in conformity with the Certification Procedures of Chapter 5.2 of the Code.

Any inadequacies in certification may result in the consignment being returned to the country of origin at the importer's expense or the destruction of the turkey meat without compensation.

5.1.3 Certification

The certificate must include the name and stamp of the Official Government Veterinarian and contain the following declarations:

1. The turkeys from which the meat was derived were born in and have been continuously resident in the US until slaughter and were slaughtered on..... (dates).

2. The turkeys from which the meat was derived passed ante- and post-mortem veterinary inspection under official veterinary supervision, and the meat is considered fit for human consumption.
3. The turkey meat has been cooked to a minimum core temperature of 76.6 °C for at least 30 minutes.

6 Review of processes

6.1.1 Review of conditions

The Department of Agriculture and Water Resources reserves the right to review the import conditions after the first year of trade or when there is reason to believe that the disease or sanitary status of the US has changed.

7 Meeting Australia's food standards

Imported food for human consumption must satisfy Australia's food standards. Australian law requires that all food, including imported food, meets the standards set out in the Australia New Zealand Food Standards Code. Food Standards Australia New Zealand (FSANZ) is responsible for developing and maintaining the Code, including Standard 1.4.2, maximum residue limits (MRLs), available on the ComLaw website. The standards apply to all food in Australia, irrespective of whether it is grown domestically or imported.

If a specific chemical is used on imported foods to control pests and diseases, then any resulting residues must not exceed the specific MRLs in Standard 1.4.2 of the Australia New Zealand Food Standards Code for that food.

If there is no MRL listed in the Australia New Zealand Food Standards Code for a specific food (or a composite, processed food), then there must be no detectable residues in that specific food.

Where an exporting country uses a chemical for which there is no current listed Australian MRL, there are mechanisms to consider establishing an Australian MRL by harmonising with an MRL established by the Codex Alimentarius Commission (Codex) or by a regulatory authority in a recognised jurisdiction. The mechanisms include applications, submissions or consideration as part of a FSANZ proposal to vary the Australia New Zealand Food Standards Code. The application process, including the explanation of establishment of MRLs in Australia, is described at the Food Standards Australia New Zealand website.