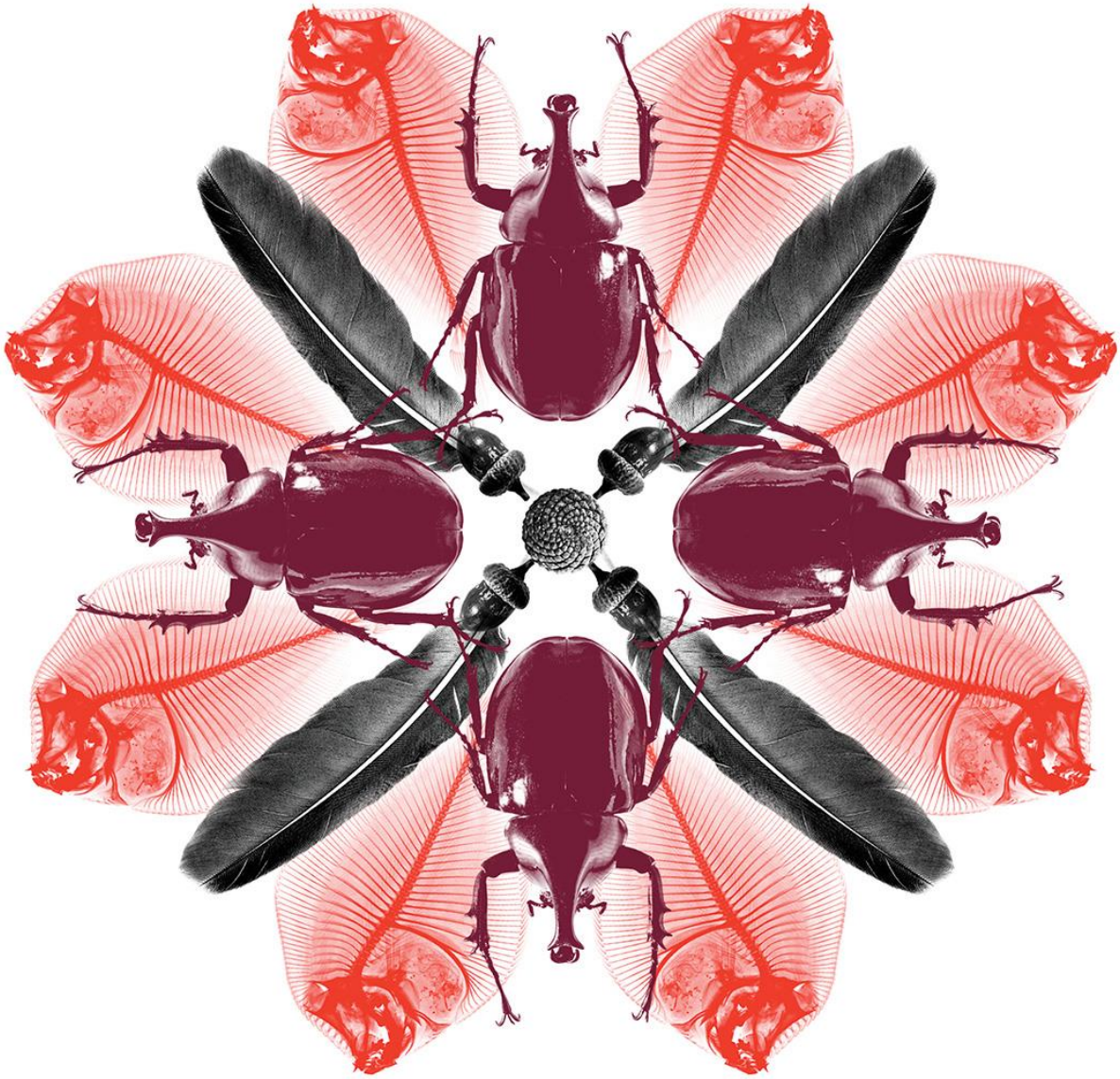
Australian Government
Department of Agriculture, Water and the Environment

Import risk review for cooked duck meat from Thailand

Draft report v2

October 2020



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Summary

The department has prepared this second draft review of biosecurity requirements in response to a request by Thailand for market access to Australia for cooked duck meat.

This is the second draft review, which has been drafted with respect to stakeholder comments after the first draft review period of 60 days. This second version has a new disease chapter on infectious bursal disease virus, an expanded risk assessment for duck virus hepatitis type 1, and several smaller revisions. Therefore, this draft is being released for a second, shorter stakeholder comment period.

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 duck meat products that meet specific temperature and time requirements during the manufacturing process.

Similar to the first draft review, this second draft review proposes that the importation of cooked duck meat to Australia from Thailand be permitted, subject to biosecurity measures.

This draft review identifies hazards that could be present in cooked duck meat from Thailand and therefore proposes the following to achieve Australia's appropriate level of protection (ALOP):

- The duck meat must have been sourced from domestic ducks (*Anas platyrhynchos domesticus* and *Cairina moschata*) that were hatched, raised and slaughtered in Thailand.
- The meat must be limited to muscle meat and overlying skin and fat.
- The ducks from which the meat was derived must have passed ante- and post-mortem veterinary inspection under official veterinary supervision.
- The ducks 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 2012).
- The ducks must have been slaughtered and processed in an abattoir and processing facility approved by the Veterinary Authority of Thailand.
- The duck meat must have been cooked in a commercial process to a minimum core temperature of at least 70°C for a minimum of 8.2 minutes (or time/temperature equivalent approved by the department).

These measures reduce animal biosecurity risks to a level that is consistent with Australia's ALOP.

This draft review was developed as an extension of the *Generic import risk analysis report for chicken meat* (Biosecurity Australia 2008). Disease information relevant to duck meat was reviewed and updated and diseases that occur in ducks but not chickens were also reviewed. Disease agents that were clearly identified as susceptible to the cooking parameters described above, such as avian influenza viruses and Newcastle disease virus, were removed from further assessment at the hazard identification stage.

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 within the 30 day consultation period.

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.

FSANZ 2012, '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 accessed 28 June 2016.

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 appropriate level of protection (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, import of the goods into Australia will not be permitted until suitable risk management measures are identified.

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 described as providing a high level of protection aimed at reducing risk to a very low level, but not to zero.

The department's science-based risk assessment process is consistent with Australian Government policy as well as Australia's rights and obligations under the World Trade Organization'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 *Biosecurity import risk analysis guidelines 2016* located on the Australian Government Department of Agriculture, Water and the Environment website.

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

1.2 Background

Currently, the only duck meat (or similar) products permitted into Australia are foie gras from France, canned or retorted duck meat products and meat-based flavours that meet specific temperature and time requirements during the manufacturing process.

This draft review of the animal biosecurity risks associated with the importation of cooked duck meat from Thailand has been undertaken in response to a request from Thailand's National Bureau of Agricultural Commodity and Food Standards and the Department of Livestock Development for access to Australian markets for cooked duck meat.

Many diseases of ducks 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 duck 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 ducks but not chickens were also reviewed.

1.3 Scope

The scope of this second draft review remains limited to an assessment of the animal biosecurity risks posed by the importation of cooked duck meat from Thailand. The definition of cooking is taken from the *Chicken meat IRA* for control of Newcastle disease virus, which requires heating the meat in a commercial process to a minimum core temperature of 70°C for a minimum of 8.2 minutes (or time/temperature equivalent).

In this second draft review the definition of duck meat is limited to muscle tissue from domestic ducks (*Anas platyrhynchos domesticus* and *Cairina moschata,* or mule ducks, which are a hybrid of *Anas platyrhynchos domesticus* and *Cairina moschata*). It includes soft tissues contained within the muscle body, such as nerves and blood contained within muscle vasculature. It also includes skin and fat overlying the muscle. This draft review does not include an assessment of the risks associated with duck offal, product containing bone or cartilage, or whole duck. In addition, the duck 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 2012).

Dried products such as jerky and powdered products such as duck-meat-based flavours are not included in the scope of this review. There can be significant differences in the thermal inactivation of biosecurity hazards depending on the water content of the product. The thermal inactivation studies cited in this review use moist heat. Consequently, the conclusions of this review can only be applied to moist products. Moist products include but are not limited to roast duck meat and further-processed products such as smallgoods.

This draft review is limited to diseases covered in the *Chicken meat IRA* that infect ducks, as well as diseases that were not considered in the *Chicken meat IRA* because they infect ducks 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.

1.4 Existing conditions

1.4.1 International arrangements

Import conditions exist for foie gras from France, canned or retorted duck meat products and meat-based flavours from all countries, for human consumption.

The import requirements for these commodities can be found on the department's website.

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 capacity of 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.

1.4.2 Domestic arrangements

The Australian 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 jurisdictions. Legislation relating to resource management or animal health may be used by state and territory government agencies to control interstate movement of animals and their products. Once animals and animal products have been cleared by Australian 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 duck 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 or highly pathogenic avian influenza, during eradication of these diseases.

1.5 Duck meat industry

Australia's duck processing industry is highly concentrated with 2 companies located in New South Wales and Victoria accounting for the bulk of production. There are also several smaller duck farms, largely located in Victoria and Western Australia, who process their own birds for local markets.

A small number of backyard ducks are kept in Australia. Most backyard poultry flocks are chickens that are maintained for table egg production and while ducks may be used for egg production they are not as common.

1.6 Next steps

The first draft review was released for 60 days public consultation on 17 July 2019. Comments were received from 10 stakeholders. In response to these comments, a second draft was produced to include a new disease chapter on infectious bursal disease virus, an expanded risk assessment for duck virus hepatitis type 1, and several smaller revisions.

This is the second draft review and is released for 30 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 World Trade Organization Secretariat about the release of the final report. Publication of the final report represents the end of the review process. The conditions recommended in the final report will be the basis of any imports permitted.

Should the final review recommend that the importation of cooked duck meat to Australia from Thailand be permitted, subject to biosecurity measures, the department will verify the systems in place in Thailand prior to issuing any import permits. This will ensure there is official oversight of the processes and that the biosecurity integrity of the processing is maintained from when the duck is slaughtered to when the final product is packaged ready for export. A favourable report will be required before import permits can be issued and trade can commence.

1.7 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.

FSANZ 2012, '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 accessed 28 June 2016.

2 Method

The World Organisation for Animal Health (OIE), in its *Terrestrial Animal Health Code* (OIE 2019c), 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* 2019 (OIE 2019c)
- a review of relevant scientific literature.
- The department considered that the comprehensive *Chicken meat IRA* was still relevant and applicable, and provided a strong framework for a review of the animal biosecurity risks for the importation of cooked duck meat from Thailand. 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 duck meat from Thailand considered only cooked duck meat from Thailand.

2.1 Review of hazard identification

Hazard identification is described in the OIE 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 2019c).

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

- a disease of ducks
- 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 (Thailand).

However, as this draft review is for the hazards associated with cooked duck meat, disease agents that were identified in the *Chicken meat IRA* as being susceptible to inactivation by cooking (definition in Section 1.3 of this document) were not retained for risk assessment.

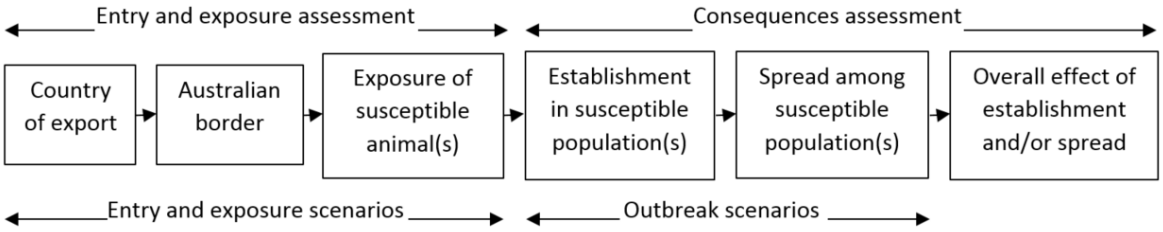
2.2 Risk assessment

Details of the risk assessment process relevant to animals and animal products are provided in Chapter 2.1 of the Code (OIE 2019c).

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 the exposure assessment describes the biological pathways necessary for exposure of animals and estimates the probability of exposure 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 made whether 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.

2.2.1 Evaluating and reporting likelihood

For those hazards retained for risk assessment, the assessment was conducted using a qualitative approach and the nomenclature in Table 1.

Table 1 Nomenclature for qualitative likelihoods

Likelihood	Descriptive definition
High	The event would be very likely to occur
Moderate	The event is equally likely to occur or not occur
Low	The event would be unlikely to occur
Very low	The event would be very unlikely to occur
Extremely low	The event would be extremely unlikely to occur
Negligible	The event would almost certainly not occur

2.2.2 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 duck meat, such as:

- prevalence of the hazard in ducks in Thailand

- ante-mortem inspection
- post-mortem inspection
- tissue distribution of the 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 (Table 1) was then assigned to describe the likelihood of the disease agent being present in the imported cooked duck meat products arriving at the Australian border. The final outcome of the entry assessment was the likelihood of entry of a potential hazard into Australia.

2.2.3 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 duck 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 4 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 applicable.

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 duck 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 the food service industry. Product not consumed would be either dumped (where it may or may not be 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. If raw or semi-cooked product were allowed entry under an approved arrangement for further processing, disposal of all waste in a manner that manages the animal biosecurity risks would be required

Given there would probably be minimal on-shore processing, the likelihood of waste product from processing being made into rendered product, and then into poultry feed, would be lower than described 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 duck meat material should they be exposed to it
- the likelihood that feed containing rendered duck 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 in the environment for 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 (using the nomenclature for qualitative likelihoods in Table 1) 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).

2.2.4 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 described in Figure 2.

Figure 2 Matrix for combining qualitative likelihoods

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

Likelihood of exposure

2.2.5 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. These represent a continuum ranging from no spread to widespread establishment. The *Chicken meat IRA* grouped all likely outbreak scenarios into 4 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.

In this review, for each exposure group, all categories of outbreak scenarios were evaluated for plausibility based on the epidemiology of each disease agent. The most likely outbreak

scenario for each hazard, resulting from the exposure of susceptible animals, was considered (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 using the qualitative descriptors as described in Table 1.

2.2.6 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 and 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.

An outbreak may occur on a small geographical level but have significant national effects, and vice versa. 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

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

2.2.7 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 Figure 3.

Figure 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
	Negligible	*Very low*	*Low*	*Moderate*	*High*	*Extreme*

Overall effect of establishment and spread

2.2.8 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 duck meat from Thailand 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 Figure 4.

Figure 4 Risk estimation matrix

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

Likely consequences

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.

2.3 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 may be imposed pre-border with the purpose of reducing the likelihood of hazards entering Australia, or post-arrival aiming to prevent the exposure and/or establishment and spread of the hazard in susceptible local populations.

2.4 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 2019c).

In conducting import risk analyses and 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 reviews to enable stakeholder assessment and feedback on draft conclusions and recommendations about Australia's animal biosecurity measures.

2.5 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 2019c, *Terrestrial animal health code 2019*, World Organisation for Animal Health, Paris, available at http://www.oie.int/standard-setting/terrestrial-code/access-online/.

3 Hazard identification

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

- diseases identified in the *Chicken meat IRA*, Conditions for the importation from approved countries of fertile eggs (domestic duck) (1999) and Importation of hatching (fertile) duck eggs from approved countries – policy review (2009) (Biosecurity Australia 1999, 2009)
- other diseases identified in the literature as occurring in ducks.

The method of hazard identification and refinement is described in Section 2.1. The hazard identification decision making process is shown in Figure 5. The preliminary list of diseases/disease agents is shown in Table 3. 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 ducks and considered relevant to this draft review.

Hazards that were assessed in the *Chicken meat IRA* as having an unrestricted risk below Australia's ALOP were not further assessed. Similarly, hazards that were assessed in the *Chicken meat IRA* as being susceptible to the cooking parameters as described in the scope (Section 1.3), were not further assessed in this draft review.

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

The following diseases were retained for risk assessment on the basis of the information provided in Table 3:

- Duck hepatitis A virus
- Duck Tembusu virus
- Duck virus enteritis
- Exotic antigenic variant and very virulent strains of infectious bursal disease virus
- Waterfowl parvovirus.

Figure 5 Hazard identification and refinement

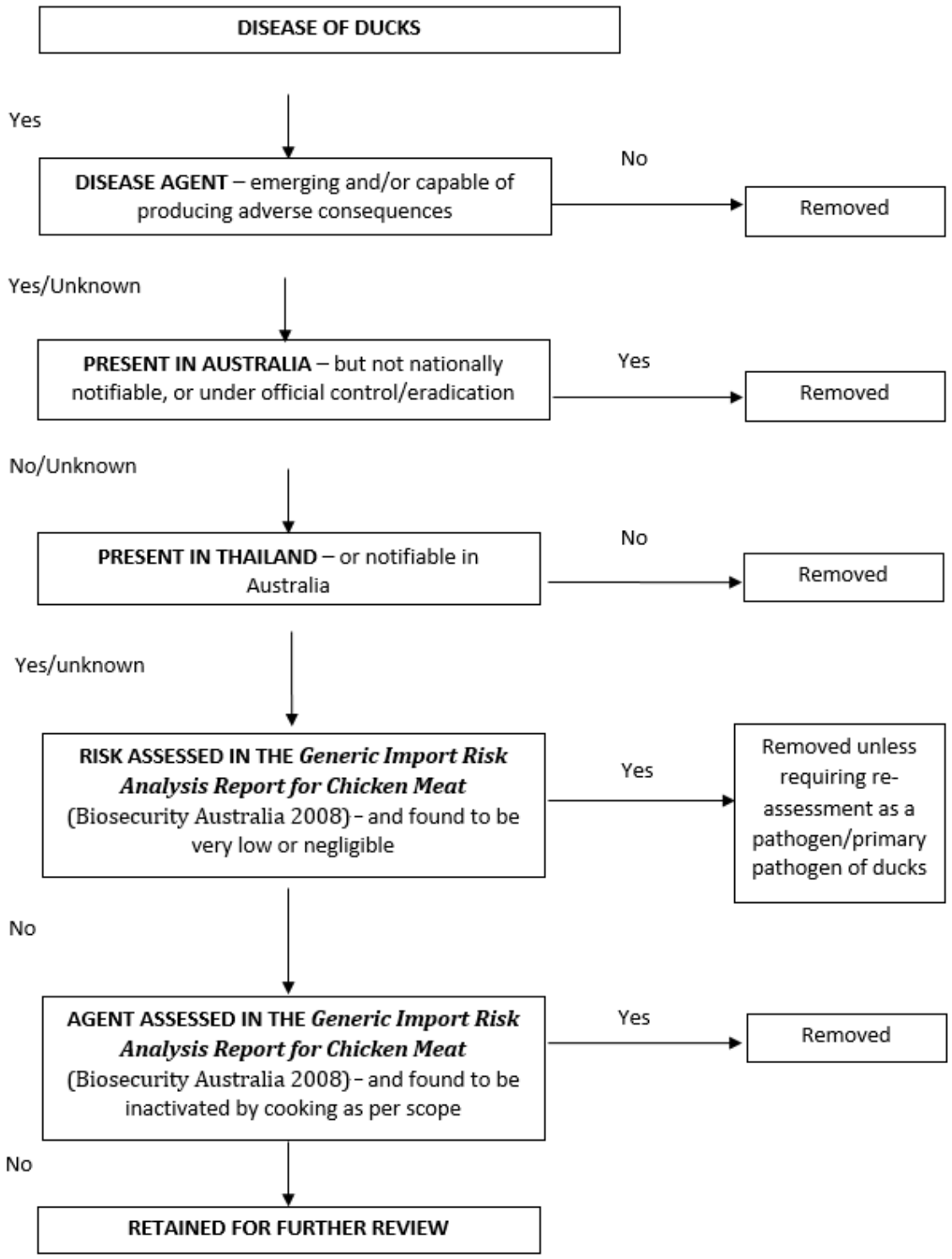


Table 3 Hazard Identification

Disease (disease agent)	**Disease affects or is spread by domestic ducks (*Anas platyrhynchos domesticus* and *Cairina moschata*)**	**Emerging and/or capable of producing adverse consequences**	**Present in Australia**	**Present in Thailand**	**Assessed in *Chicken meat IRA* as either not requiring risk assessment or not requiring risk management**	**Agent assessed in *Chicken meat IRA* and found to be inactivated by cooking as defined in scope**	**Retained for risk review**
Avian infectious laryngotracheitis virus	No	Yes	Yes	Yes	Yes	No	No
Avian nephritis virus	Yes	Yes	Yes	No	Yes	No	No
Avian metaavulavirus 2 and avian paraavulavirus 3	No	Yes	No	Yes	No	Yes	No
Borrelia anserina	Yes	Yes	Yes	No	Yes	No	No
Brachyspira spp	Yes	Yes	Yes	Yes	No	No	No
Campylobacter jejuni	Yes	Yes	Yes	Yes	Yes	No	No
Chlamydophila psittaci	Yes	Yes	Yes	Yes	Yes	No	No
Duck adenovirus-1 (Egg drop syndrome virus/Atadenovirus)	Yes	Yes	Yes	No	No	No	No
Duck adenovirus-2 (Adenovirus Group 1)	Yes	Yes	Yes	No	No	No	No
Duck virus hepatitis type 1 (Duck hepatitis A virus/ DHAV/avihepatovirus)	Yes	Yes	No	No	No	No	Yes
Duck Tembusu virus	Yes	Yes	No	Yes	No	No	Yes
Duck virus hepatitis type 2 (Duck astrovirus -1/ DAstV-1)	Yes	Yes	No	No[a]	No	No	No[b]
Duck virus hepatitis type 3 (Duck astrovirus -2/DAstV-2)	Yes	Yes	No	No[c]	No	No	No[b]

Duck virus enteritis	Yes	Yes	No	Yes	No	No	Yes
Eastern equine encephalitis/Western equine encephalitis viruses	Yes	Yes	No	EEE Yes; WEE No	Yes	No	No No
Enterohaemorrhagic Escherichia coli	Yes	No	Yes	Yes	Yes	No	No
Fowl pox	Yes	Yes	Yes	Yes	Yes	No	No
Haemophilus paragallinarum/ Avibacterium paragallinarum	No	Yes	Yes	Yes	Yes	No	No
Highly pathogenic avian influenza virus	Yes	Yes	No (Controls)	No	No	Yes	No
Infectious bursal disease virus serotype 1 (very virulent and exotic antigenic variant strains)	Uncertain[d]	Yes	No	Yes	No	No	Yes[d]
Japanese encephalitis virus	Yes	Yes	Yes	Yes	Yes	No	No
Low pathogenicity avian influenza virus	Yes	Yes	No (Controls)	No	No	Yes	No
Marek's disease	No	Yes	Yes	Yes	Yes	No	No
Muscovy duck reovirus	Yes	Yes	No	No	No	No	No
Mycobacterium avium	Yes	Yes	Yes (Controls)	Yes	Yes	No	No
Mycoplasma gallisepticum	Yes	Yes	Yes	Yes	Yes	No	No
Mycoplasma synoviae	Yes	Yes	Yes	Yes	Yes	No	No
Mycoplasma anatis	Yes	Yes	No	No	No	No	No
Newcastle disease	Yes	Yes	No (Controls)	Yes (Controls – Lentogenic strain vaccines	No	Yes	No

				are applied in all poultry sectors to prevent disease)			
Ornithobacterium rhinotracheale	Yes	Yes	Yes	Yes	Yes	No	No
Pasteurella multocida	Yes	Yes	Yes	Yes	Yes	No	No
Riemerella anatipestifer	Yes	Yes	Yes	Yes	Yes	No	No
Salmonella enteritidis	Yes	Yes	Yes (controls)	Yes	No	Yes	No
Salmonella Gallinarum	Yes	Yes	No[e] (controls)	No	No	Yes	No
Salmonella Pullorum	Yes	Yes	No[e] (controls)	Yes	No	Yes	No
Salmonella Typhimurium, antibiotic resistant strains	Yes	Yes	No	Yes	No	Yes	No
Waterfowl parvoviruses	Yes	Yes	No	Yes	No	No	Yes
West Nile virus	Yes	Yes	No	Yes	Yes	No	No

a Only ducks appear to be affected by duck virus hepatitis type 2 (duck astrovirus type 1 [DAstV-1]). Outbreaks only appear to have occurred in England and potentially China. The earliest described outbreaks occurred in ducks kept on open fields in eastern England, initially reported in 1965 and up to 1969, then again in 1983/84, which were the last reported outbreaks in England (Gough et al. 1985; OIE 2018b; Woolcock & Tsai 2013). In 2008 there was a severe outbreak of duck hepatitis in China. Virus present in liver extracts had very high amino acid sequence identity (Fu et al. 2008) to the DAstV-1 sequenced by Todd et al. (2009) and very different to DAstV-2 (Todd et al. 2009).
b Astroviruses and avihepatoviruses are non-enveloped viruses with a single stranded, positive sense genome; both are resistant to low pH and are heat tolerant. Duck viral hepatitis type 1 (consisting of DHAV-1, DHAV-2, DHAV-3 genotypes) is more pathogenic and widespread compared to duck virus hepatitis type 2 (duck astrovirus type 1 [DAstV-1]) and duck virus hepatitis type 3 (duck astrovirus type 2 [DAstV-2]). Duck virus hepatitis type 2 (duck astrovirus type 1 [DAstV-1]) is more closely related to chicken astroviruses (CAstVs), and duck virus hepatitis type 3 (duck astrovirus type 2 [DAstV-2]) is more closely related to turkey astrovirus type 2 (TAstV-2) (Todd et al. 2009). Inactivation studies have shown that to obtain the 6 log10 sterility assurance level, TAstV-2 needs to be heated for 1.8 minutes at 82.2ºC (Koci & Schultz-Cherry 2002). The heat treatment required by the scope of the review would, similar to duck viral hepatitis type 1, likely result in some inactivation of any virus present but is unknown whether it would completely inactivate this virus. However, given the similarities between the 3 types of duck viral hepatitis' in regards to agent properties, transmission and pathogenesis, the required risk management for duck virus hepatitis type 1 (duck hepatitis A virus, which is believed to be more prevalent and widespread globally) would also apply to duck virus hepatitis type 2 (duck astrovirus type 1 [DAstV-1]) and duck virus hepatitis type 3 (duck astrovirus type 2 [DAstV-2]).
c Duck virus hepatitis type 3 (duck astrovirus type 2 [DAstV-2]) has only been described in the United States of America (OIE 2018b; Woolcock 2008).

d Available literature suggests that IBDV serotype 1 is capable of infecting ducks in some instances and producing a limited antibody response, probably due to poor virus replication. Experimental infection studies in ducks have failed to isolate virus. No clinical or microscopic signs have been observed in ducks to date. Virus was isolated from faeces and caecal tonsils of wild ducks in 1 study. There is no available literature to support the conclusion that significant replication of pathogenic IBDV serotype 1 occurs in ducks (Eddy 1990; Eterradossi & Saif 2013; Hollmén et al. 2000; McFerran et al. 1980; McNulty, Allan & McFerran 1979; Okoye, Iguomu & Nwosuh 1990; Oladele 2010; Oluwayelu et al. 2007; Tsai et al. 1996; Wilcox et al. 1983; Woo-Jin et al. 2008; Yamada, Matsuo & Uchinuno 1982). However, given the importance of the disease and questions raised by stakeholders with supporting reference material about whether ducks can be infected, the disease was subjected to further analysis.
e *Salmonella* Gallinarum and *Salmonella* Pullorum are not present in commercial poultry.

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4 Risk reviews

4.1 Duck hepatitis A virus

4.1.1 Background

Duck hepatitis A virus (DHAV) includes 3 antigenically unrelated genotypes, 1, 2 and 3. Duck hepatitis A virus type 1 (DHAV-1) is the most prevalent and widespread of the 3 types. There is limited information available on types 2 and 3, however, the clinical presentation is reported to be similar to type 1. Therefore, this draft risk review will focus on DHAV-1.

DHAV-1 is a highly contagious, acute and lethal pathogen that affects young ducklings. Normally, after 3 weeks of age, ducklings begin to develop age-related resistance, and at 6 weeks of age are no longer susceptible. However, DHAV-1 was determined to be the cause of egg drop, reduced feed consumption and ovary-oviduct infection in laying ducks in China (Zhang et al. 2018). DHAV-1 is 1 of 5 viruses that causes liver disease in ducks, however DHAV-1 is considered to be the most pathogenic and causes significant losses in the duck industry (Lin et al. 2016; Song et al. 2014). This type can cause high mortality and morbidity levels with a mortality rate greater than 80% in ducklings under 3 weeks of age (Lin et al. 2016).

The first recorded identification of duck hepatitis was in 1945, on Long Island, the United States of America (Fabricant, Rickard & Levine 1957). It has now been officially reported in many countries in Europe, Asia and North America. Outbreak investigations have been reported for cases in Canada, Egypt, Japan, Korea, Poland, Taiwan, the United States of America and Vietnam (Doan et al. 2016; Erfan et al. 2015; Friend & Trainer 1974; Hassaan, Shahin & Eid 2018; Kamomae et al. 2017; Kozdru, Czekaj & Lorek 2014; MacPherson & Avery 1957; Soliman et al. 2015; Tseng, Knowles & Tsai 2007). DHAV-2 has been identified in Taiwan and DHAV-3 in China, Vietnam and South Korea (Doan et al. 2016; Li et al. 2013).

Duck virus hepatitis is an OIE-listed disease and nationally notifiable in Australia (Department of Agriculture 2019; OIE 2019b).

4.1.2 Technical information

Epidemiology

Susceptible species

Ducklings are the primary hosts; mature ducks are susceptible to infection but asymptomatic. Field observations indicate chickens and turkey are resistant (Woolcock & Tsai 2013). A serological survey of bean and white fronted geese in Germany showed none had antibodies to DHAV (Hlinak et al. 1998).

In an unpublished study investigating species sensitivity to the virus, Cherry Valley and Tsaiya ducklings were found to be highly sensitive breeds, Pekin ducklings and hybrid Pekin and Tsaiya ducklings were relatively sensitive to disease while mule ducklings showed intermediate sensitivity and Muscovy ducklings were resistant (Woolcock & Tsai 2013).

Modes of transmission

In the field, DHAV-1 can spread quickly through a flock, infecting all susceptible ducklings. Although this virus is considered extremely contagious, there have been exceptions within farms with adjoining duck pens reporting both negligible mortality and 65% mortality (Woolcock & Fabricant 1991).

The natural routes of transmission are airborne or via contaminated feed or water. The infection route is the pharynx or upper respiratory tract (Toth & Norcross 1981). DHAV-1 was found in 1 study to be lethal when ducklings were infected with an aerosol form of the virus (Priz, cited in

Woolcock & Tsai 2013). Oral vaccination of ducklings has resulted in successful immunisation (Tripathy & Hanson 1986).

Incubation period
The incubation period is generally 1–2 days with all mortalities occurring within 3 to 4 days. Generally ducklings die within an hour of clinical signs appearing (Woolcock & Tsai 2013).

Ducklings that have recovered may excrete the virus for up to 8 weeks in faeces, and have immunity with the serum containing virus neutralising antibodies (Kamomae et al. 2017; Reuss, cited in Woolcock & Tsai 2013).

Distribution and prevalence
There have been no official reports of DHAV-1 in Thailand; however, a duck hepatitis virus has been isolated from 9 to 14 day old ducklings from Chonburi and Rayong provinces (Sirivan, Tantaswasdi & Chuenchai 1997). Experimental vaccination of ducks with a DHV vaccine resulted in protection but the unvaccinated ducklings were susceptible to infection with 60–80% mortality (Sirivan, Tantaswasdi & Chuenchai 1997).

There is variation amongst strains, and reports of the pathogen in China have indicated that the diversity of strains is dependent upon where they are isolated (Li et al. 2013). The virus strains can be allocated into 3 regional groups dependent upon the phylogenetic analysis of the VP1 gene. Interestingly, Asian DHAV-1 clusters with Egyptian DHAV-1, with the closest nucleotide homology to the Asian strains being 98%. The difference in virulence between the strains was identified when embryonated eggs were inoculated with the Egyptian strain of DHAV-1. These eggs did not display lesions, while reports of inoculations with the Asian strain of DHAV-1 indicate that lesions are usually observed (Erfan et al. 2015).

Australian status
DHAV-1 has not been reported in Australia and there is no active surveillance. However, duck virus hepatitis is nationally notifiable.

Agent properties
DHAV-1 is a single-stranded, positive-sense RNA virus and is classified as the type strain of the genus *Avihepatovirus* in the family Picornaviridae (Tseng, Knowles & Tsai 2007; Yang et al. 2008). The pathogen is 1 of 3 genotypes and was originally identified in 1950 in chicken embryos after which it was classified as an enterovirus. It was not until after 2006 when the complete genome was determined that the virus was classified in the new genus *Avihepatovirus* (ICTV 2018).

Published studies on the thermal stability of DHAV-1 are dated and show some discrepancies in survival time (Davis 1987; Hanson, Rhoades & Schricker 1964). The most recent publication by Davis (1987) found that the virus is comparatively stable at temperatures below 40–45°C but was rapidly inactivated at higher temperatures. At 56°C, the half-life of the virus was 1.26 minutes. Hanson et al. (1963) found that the virus was rapidly inactivated at 56°C, with 99% of the activity lost in 30 minutes and none detectable after 90 minutes, as measured by inoculating chicken embryos with virus. Furthermore, early experiments at APHA Weybridge Laboratory showed that a strain of virus resisted heating to 56°C for 60 minutes but was inactive after heating at 62°C for 30 minutes (Woolcock & Tsai 2013). Whilst these results appear to infer similar information about the virus's thermal stability, they are in contrast to another study which indicated a longer survival time; Dvorakova and Kozusnik (cited in Hanson, Rhoades & Schricker 1964) reported that the virus withstood 56°C for at least 18 hours.

At lower temperatures, the virus appears to remain viable for extended periods of time. In laboratory conditions the virus has been shown to survive for 9 years at -20°C and for longer than 2 years at 2–4°C (Asplin 1961; Hanson, Rhoades & Schricker 1964). In more natural conditions, the virus has been recorded to survive greater than 37 days in moist faeces in a cool shed (Asplin 1961). Furthermore, Hanson et al. (1964) found that the virus was able to survive at 37°C for 7 days.

The virus appears to be susceptible to degradation under extreme pH levels. Davis (1987) found that the virus lost infectivity at pH 2 and pH 12 and titres fell to undetectable levels within 30 minutes. Exposure to 3% chloramine, 2% caustic soda or 1% formaldehyde at 15–20°C for 2 hours will also inactivate DHAV due to pH sensitivity (Dvorakova, Pollard, cited in Woolcock 2003).

Phylogenetic analysis shows that avihepatoviruses are more closely related to the genus *Parechovirus* than to other picornaviruses (Ding & Zhang 2007; Kim et al. 2006). Thermal inactivation studies performed on parechoviruses, namely Ljungan virus (LV) and *Parechovirus 1*, show the viruses can be inactivated by heat but are relatively heat resistant compared to other food borne viruses. LV was still found to be infectious after exposure at 70°C for 20 min, albeit at significantly reduced titres, was resistant to inactivation at acidic pHs, detergents and oxidizing environments, complete inactivation requiring heating to 90°C or laboratory purification of the sample prior to lower thermal treatments (Ekström et al. 2007). In another study, *Parechovirus 1* was the only food borne virus to not be completely inactivated at 73°C within 3 minutes (Tuladhar et al. 2012). The conclusion was that parechovirus inactivation by common food processing methods should not be simply assumed (Tuladhar et al. 2012).

The above information on thermal inactivation of parechoviruses and DHAV-1 suggest that whilst cooking as defined in this review's scope (70°C for a minimum of 8.2 minutes) is likely to result in some inactivation of DHAV-1 virus, it may not fully inactivate all virus.

4.1.3 Presence in duck meat

Song et al. (2014) researched the invasive properties of a virulent strain (VS) and an attenuated strain (AS) in different organs in ducklings and the levels of the 2 strains in the different organs were compared. RNA extracts from the heart, spleen, lung, kidney, brain, intestine and muscle of infected ducklings at 36 hours post infection were subjected to quantitative reverse transcriptase polymerase chain reaction (RT-PCR). The test showed high levels of VS and AS RNA in the liver, spleen and intestine, moderate levels in the kidney, muscle, lung and heart, and low levels in the brain. VS and AS showed similar tissue tropism in the liver, spleen and intestine, suggesting that the 3 organs are targeted by these 2 DHAV-1 strains, whilst the intestine might be the initial infection and proliferation site during natural infection. Moreover, although there was a higher number of AS virus copies inoculated than VS, significantly more VS virus RNA copies than AS were detected in each organ. These results demonstrated that VS replicated more efficiently than AS in ducklings.

The findings by Lin et al. (2016) were similar to those of Song et al. (2014). The viral loads of DHAV-1 in liver and spleen were significantly higher than those in pancreas, heart, kidney, brain, bursa of Fabricius and the thymus, which indicated that the liver and spleen of ducklings are the major target organs for the 2 viruses.

4.1.4 Pathogenesis

Infection in ducklings occurs mainly through the mucous membranes of the digestive and respiratory organs. The virus spreads via the blood circulation to several organs, especially the

liver and brain. The liver becomes enlarged with petechial and ecchymotic haemorrhages throughout and there is extensive hepatocyte necrosis and hyperplasia (Trefilov et al. 2018).

Within the first hours of infection, virus titre in the blood circulation rises rapidly, then gradually decreases by 48–72 hours. The virus titre in the liver and brain also increases, usually within 48–72 hours of infection (Trefilov et al. 2018). Liver function decreases, allowing toxic products to spread throughout the body. Ducklings die as a result of irreversible changes in the liver and other organs. The toxins produced typically cause opisthotonus, especially in fat ducklings (Trefilov et al. 2018).

DHAV-1 has been isolated predominately in the allantoic liquids of duck embryos and the liver (Chen et al. 2013). The spleen, kidneys and bursa of Fabricius are also reported to show signs of disease (Jin et al. 2008; Sheng et al. 2014).

Lin et al. (2016) determined that the age of the ducklings did not correlate with DHAV-1 viral loads in tested organs. Ducklings tested between 3 and 19 days old died regardless of the specific age when they reached a critical level of DHAV-1 (Lin et al. 2016).

Although wild aquatic fowl are considered to be mechanical carriers of the virus, studies have not confirmed this hypothesis. Testing 520 birds of 6 species found no serological confirmation of DHAV-1 infection (Asplin, cited in Woolcock & Tsai 2013). Furthermore, none of the 36 wild ducks of 4 species demonstrated evidence of DHAV-1 even though the virus had been found in domestic ducks from the same pond (Ulbrich, cited in Woolcock & Tsai 2013). However, wild duck embryonating eggs, when taken from an infected area, have been found to be susceptible to infection, confirming that although the species is susceptible, they do not appear to be extensive carriers of the virus (Woolcock & Tsai 2013).

4.1.5 Diagnosis

Clinical signs

DHAV-1 causes acute and highly contagious hepatitis in ducklings that usually results in mortality within 3 to 4 days of infection. Soon after infection ducklings become lethargic and stop moving, have partially closed eyes and squat before falling on their sides. They then kick spasmodically, draw their heads back, and usually die within an hour of clinical signs starting (Fabricant, Rickard & Levine 1957; Kim et al. 2007).

Ducklings less than 1 week of age are affected most severely with mortality reaching up to 95%. One to 3–week–old ducklings usually suffer up to 50% mortality, while ducklings 4–5 weeks of age have low or negligible mortality (Kim et al. 2007). Mao et al. (2017) infected adult ducks (160 days old) and noted that they were asymptomatic, though they had been vaccinated at 1 day of age.

Pathology

The liver contains ecchymotic haemorrhages, is enlarged, and has the primary lesions. The kidney and spleen are often swollen, with the former having congested renal blood vessels and the latter showing signs of necrosis (Woolcock & Tsai 2013). As the disease progresses, the organs will appear differently to those in the acute stage of infection. For example, the liver will no longer be enlarged, but have irregular, pale mottling (Fabricant, Rickard & Levine 1957; Woolcock 2003).

Histological changes seen from DHAV-1 are necrosis of hepatic cells, subcutaneous oedema and bile duct hyperplasia with different degrees of haemorrhaging and inflammatory cell response. Ducklings that survive have been observed to have regeneration of the liver parenchyma (Fitzgerald, Hanson & Simon 1969; Woolcock 2003).

Testing

DHAV-1 can be isolated from the liver or allantoic liquids of duck embryos (Chen et al. 2013). The pathogen may then be confirmed by inoculation of the isolate into susceptible ducklings, into allantoic sacs of susceptible embryonating duck eggs or primary cultures of duck embryo liver cells (Woolcock 1986; Woolcock & Tsai 2013). Other detection techniques include RT-PCR, multiplex polymerase chain reaction and VP1 ELISA (Enzyme-linked immunosorbent assay) (Chen et al. 2013; Liu et al. 2010; Wen et al. 2014).

4.1.6 Treatment

There is no effective treatment for DHAV-1 infection.

4.1.7 Control

Both inactivated vaccines and live attenuated vaccines are available to protect ducks against DHAV-1.

Live attenuated DHAV-1 vaccines are used before the start of egg laying to immunise breeder ducks or to actively immunise susceptible ducklings. Breeder ducks, primed with live attenuated vaccine or previously exposed to live DHAV-1, may also be vaccinated using an inactivated DHAV-1 vaccine. Well-vaccinated breeder ducks may transmit passive immunity in eggs for 8–9 months (Woolcock & Tsai 2013).

4.1.8 Current biosecurity measures

For imports of fertile duck eggs into Australia, the flock from which the eggs were derived are required to be sample tested free from DHAV-1. The eggs are hatched under quarantine in Australia and the hatched ducklings undergo further testing.

4.1.9 Conclusion

- DHAV-1 is an OIE-listed disease and is nationally notifiable in Australia.
- DHAV-1 can cause high mortality and morbidity levels with a mortality rate greater than 80% in ducklings under 3 weeks of age. Slaughter age ducks (7-8 weeks and onwards) are resistant to infection.
- Ducklings that have recovered may excrete the virus for up to 8 weeks in faeces, and have immunity with the serum containing virus neutralising antibodies.
- There has been no official report of DHAV-1 in Thailand; however, a duck hepatitis virus (DHV) was isolated from 9 to14 day old ducklings from Chonburi and Rayong provinces.
- Whilst cooking as defined by this review's scope at section 1.3 is likely to result in some inactivation of DHAV-1 virus, it may not fully inactivate all virus.

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

4.1.10 Risk assessment

Entry assessment

- There are no official reports of DHAV-1 in Thailand, however, a Duck Hepatitis virus has been isolated from ducklings in the Chonburi and Rayong provinces.
- The disease causes high economic losses in ducklings due to high mortalities. Most severe infections occur in ducklings less than 1 week of age with mortality reaching up to 95%. One to 3-week-old ducklings usually suffer up to 50% mortality, while ducklings 4–5 weeks of age have low or negligible mortality. Adult ducks are asymptomatic.

- Given the wide-scale mortalities in ducklings, it is considered unlikely that the virus would be circulating in commercial flocks or production systems without knowledge by the company or Competent Authority.
- Gross changes may also be expected on ante- and post-mortem inspection in younger (7-8week) slaughter age ducks that have previously been infected, but not necessarily at older ages (18week).
- Ducklings that recover can continue to faecally excrete the virus for up to 8 weeks.
- Infected ducklings show gross pathological lesions on affected organs, especially the liver. There is no clear documentation to support if lesions persist in recovered ducklings of slaughter age (greater than 7 weeks). The liver is the major target organ for DHAV-1 but the virus has been isolated from other areas including muscle.
- Inactivation studies show that DHAV-1 was inactivated rapidly at 56°C with 99% of activity lost in 30 minutes and none detectable after 90 minutes (Hanson, Rhoades & Schricker 1964). Further studies on the virus showed the half-life of DHAV at 56°C to be 1.26 minutes but this could be extended by the addition of salts to the virus solution (Davis 1987). Closely related Ljungan virus was still infective at 70°C for 20 minutes, albeit at significantly reduced titres, and was not inactivated at 73°C for 3 minutes.

Conclusion: Based on this information and using the nomenclature for qualitative likelihoods in Table 1, the likelihood of importation of DHAV-1 associated with cooked duck meat from Thailand was estimated to be **very low**.

Exposure assessment

- The virus is extremely contagious, markedly heat resistant and can survive in the environment for a long period of time.
- Infection in ducklings occurs mainly through the mucous membranes of the digestive and respiratory organs. There have been no reports of oral transmission through infected meat; however, transmission by mouth cannot be discounted. The infective dose of DHAV-1 has not been described.
- There is no information on the susceptibility of wild birds or Australian wild aquatic birds to DHAV-1. Chickens and turkeys are resistant.
- As only cooked products consisting of muscle meat and overlying fat and skin would be imported, most imported product will move from the retailer/ distributer to household consumers or to the food industry. Product not consumed would be disposed of, which may result in exposure to wild birds and non-avian species. Backyard (low biosecurity) poultry could also be exposed to waste if fed the products sold directly to households. Limiting the scope to muscle meat and overlying fat and skin that has been cooked reduces the type and volume of waste that may be created. Bone, trimmings and offal would not be imported, reducing the risk of exposure to waste.

Conclusion: Based on this information and using the nomenclature for qualitative likelihoods in Table 1, the likelihood of exposure of Australian ducks to DHAV-1 associated with cooked duck meat from Thailand was estimated to be **very low**.

Estimation of the likelihood of entry and exposure

The estimate of the likelihood of entry was very low and the likelihood of exposure was very low. Using the matrix described in Figure 2, the likelihood of entry and exposure of DHAV-1 was estimated to be **extremely low**.

Likelihood of establishment and/or spread associated with the outbreak scenario
The most likely outbreak scenario following exposure to DHAV-1 was considered to be limited establishment in populations of backyard ducklings.

Factors considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible ducklings to DHAV-1 were:

- Horizontal transmission via the oral route by consumption of infected product. If the duckling or duck recovers then it may continue to shed the virus for up to 8 weeks, further transmitting via the faecal-oral route.
- Due to its resistance in the environment, DHAV-1 can survive for long periods of time and can persist in buildings and on poorly cleaned and disinfected surfaces which results in transmission between subsequent flocks.
- Exposure to native ducks either via feeding infected product or horizontal transmission via infected ducklings. The spread of DHAV-1 via wild populations has not been reported.

Conclusion: Based on these considerations and using the nomenclature for qualitative likelihoods in Table 1, it was estimated that the likelihood of establishment and spread of DHAV-1 in backyard duck populations was **low**.

Determination of the effects resulting from the outbreak scenario
For the most likely outbreak scenario, the direct and indirect impacts of DHAV-1 were estimated at the national, state or territory, district/region and local levels. Adverse effects are evaluated in terms of 7 (2 direct and 5 indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of DHAV-1.

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

- DHAV-1 is highly contagious and fatal in ducklings less than 1 week of age with mortality reaching 95%. One to 3 week old ducklings usually suffer up to 50% mortality, while ducklings 4 to 5 weeks of age have low or negligible mortality. Adult ducks are asymptomatic.

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

- There are no reports of DHAV-1 in wild ducks or other waterfowl species. However, wild duck embryonating eggs, when taken from an infected area, have been found to be susceptible to infection, confirming that although the species is susceptible, they do not appear to be extensive carriers of the virus.

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

- DHAV-1 is an OIE-listed disease and is nationally notifiable in Australia.
- An outbreak would likely provoke control and eradication measures. These include active surveillance, disinfection of infected flocks and elimination of carrier birds. A vaccination program may be introduced.

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

- Australia's duck processing industry is highly concentrated with 2 companies located in New South Wales and Victoria accounting for the bulk of production. There are also several smaller duck farms, largely located in Victoria and Western Australia, who process their own birds for local markets. There are also backyard ducks scattered around the country. There are no significant producers of duck eggs for commercial purposes (PoultryHub 2018). Because of the structure of the major producers, biosecurity can largely be controlled to prevent or respond to potential impacts. Smaller duck farms and backyard ducks are more likely to be impacted by an outbreak due to low biosecurity controls and resources, however the scattered nature of their locations will limit spread.
- Pekin and Muscovy ducks are among the major breeds used for meat production, Pekin ducks being more sensitive to infection whilst Muscovy ducks are more resistant.
- There would be potential for minor shortages of duck meat for commercial customers due to direct losses of stock.
- There would be little 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

- Australia would lose its country freedom status for DHAV-1, potentially restricting international trade. Only 5% of processed duck meat produced in Australia is exported thus the impact of this occurrence would be minor.

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

- As there has not been any discernible effects on wild duck or waterfowl species internationally, it is expected that 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

- DHAV-1 may cause significant mortalities in young ducklings. Following an outbreak, affected grower groups would need to be culled, horizontal transmission controlled with proper disinfection protocols and on-going age group segregation to avoid subclinical infection in older duck groups. Given there are only 2 major, vertically integrated farm companies in Australia, the loss of a grower group (depending on size) would have noticeable financial impact on the market. However, as they operate all-in all-out systems, an affected farm would be expected to only lose the grower groups exposed to the virus, with income losses on those grower ducks. Current surveillance and monitoring measures would be expected to quickly identify, isolate and eradicate the disease.
- Currently, duck manure is often spread onto farming land (PoultryHub 2018) and either a full or partial cleanout of litter occurs between grower groups (Animal Health Australia, Farm Biosecurity Manual for the Duck Meat Industry May 2010). This could pose risks of exposure to other groups.

Conclusion for overall direct and indirect effects: Based on the geographic level and magnitude of effects, and using the rules outlined in Table 2, the overall effect of establishment and/or spread for the outbreak scenario was estimated to be **low**. The effect would likely be recognised on a regional level and be of minor significance within affected zones. The effect would likely be significant to directly affected parties.

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 Figure 3 to obtain an estimation of likely consequences of **very low**.

4.1.11 Risk estimation

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 DHAV-1 introduced by imported cooked duck meat from Thailand into Australia.

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

Because the unrestricted risk estimate achieves Australia's ALOP, no specific risk management is considered necessary for this hazard.

However, this risk estimation relies on the assumption that DHAV-1 is unlikely to be circulating unnoticed in a commercial production system rearing ducklings; the presence of DHAV-1 on a commercial farm or operation would significantly change the entry assessment.

Therefore, to address this issue it is proposed that the following general declaration (or equivalent) be provided on export health certification:

A statement that no clinical signs, epidemiological or other evidence of infection with contagious diseases were present in the source flock(s) prior to slaughter.

4.1.12 References

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4.2 Duck Tembusu virus

4.2.1 Background

Duck Tembusu virus (DTMUV) is a flavivirus, a genus within the family Flaviviridae, which causes a highly infectious disease in ducks. It was first identified in April 2010 in Shanghai, China and spread rapidly to most of the duck producing regions of China, especially in the south-east provinces (Su et al. 2011; Yan et al. 2011). The disease is characterised by a dramatic fall in egg production in both egg laying and breeding ducks, and by diarrhoea, neurological disorders and death in ducklings under 2 weeks of age (Su et al. 2011; Sun et al. 2014; Thontiravong et al. 2015). Since 2010, the disease has been reported in ducks in Malaysia and Thailand (Homonnay et al. 2014; Thontiravong et al. 2015).

Flaviviruses are positive sense, single-stranded RNA viruses that occur throughout the world. Over 70 different members in the genus Flavivirus have been discovered. Many flaviviruses are transmitted to humans and animals by infected mosquitoes or ticks (Liu et al. 2013).

Tembusu viruses (TMUV) have been reported to infect mosquitoes, chickens, ducks, geese (family Anatidae, genera *Anser*, *Branta* and *Chen*), pigeons (C*olumba livia*) and sparrows (Passeridae) with DTMUV being reported only in a variety of duck breeds, especially the Pekin ducks, Muscovy ducks and domesticated Mallards (Zhang et al. 2017). TMUV is not known to be a zoonotic disease although TMUV antibodies have been detected in workers involved with the duck industry (Tang et al. 2013; Wang et al. 2016).

Infection due to DTMUV is not OIE-listed nor is it nationally notifiable or subject to official control or eradication in Australia.

4.2.2 Technical information

Epidemiology

DTMUV can spread very rapidly within and between flocks and there is clear evidence supporting both direct transmission and spread via a mosquito vector. Direct transmission and airborne transmission were observed to be the primary method of spread of DTMUV (Homonnay et al. 2014; Li et al. 2015b). This is unusual in that this group of flavivirus usually relies on a mosquito vector for spread (Liu et al. 2013). However, evidence of direct transmission is very strong. In China outbreaks have occurred during times when mosquitoes are not active (Yan et al. 2011). Further, high levels of DTMUV have been identified in tracheal cells, enabling direct transmission of virus between birds in close proximity to each other (Cha et al. 2013).

There have also been reports of faecal-oral transmission of DTMUV (Thontiravong et al. 2015).

Mosquito vectors do have a role in transmission as DTMUV genes have been detected in mosquitoes and DTMUV outbreaks in Thailand have been most frequent at times of high mosquito activity (Tang et al. 2015; Thontiravong et al. 2015). TMUV is experimentally transmissible to chickens through at least 1 member of the *Culex* genus, *C. vishnui* (O'Guinn et al. 2013). *C. vishnui* has not been identified in Australia.

The identification of the virus in other avian species may indicate an ability of the virus to adapt quickly to new hosts (Liu et al. 2013). As flaviviruses are RNA viruses, they are prone to transcription errors that make them more likely than DNA viruses to be modified by the process of replication (Carter & Saunders 2007).

Infection and morbidity rates are very high (up to 90%) and, depending on the management conditions of flocks, mortality typically ranges from 5 to 15% and occasionally up to 30%, especially when there is secondary bacterial infection (Zhang et al. 2017).

Distribution and prevalence

Infection due to DTMUV was first reported in China in 2010. The cause of the outbreaks in China was initially identified as a new flavivirus, Baiyangdian virus, and the disease was called Duck Egg Drop Syndrome (DEDS) (Su et al. 2011). DEDS has been responsible for economic losses through decreased egg production and the deaths of laying ducks in mainland China (Jiang et al. 2012; Liu et al. 2013; Yan et al. 2011). In 2011, DEDS also occurred in several chicken and goose farms in China and a Tembusu-like virus, having homology to the DTMUV, was identified as the cause (Liu et al. 2012).

Outbreaks characterised by neurological signs and significant losses in young Pekin ducks were reported in Malaysia in 2012 with the strain of DTMUV identified being named Perak virus (Homonnay et al. 2014). This strain is around 90% homologous to the strain affecting ducks in Thailand, indicating that Perak virus is the same species as the virus affecting ducks in Thailand and China (Thontiravong et al. 2015). Flaviviruses are defined as being of the same species if they are more than 84% homologous (Kuno et al. 1998). There were earlier reports of a TMUV named Sitiawan virus causing encephalitis in chickens in Malaysia (Kono et al. 2000).

In Thailand, outbreaks of disease due to DTMUV were first reported in layer and broiler duck farms in 2013 with investigations indicating infection occurring as far back as 2010 (Chakritbudsabong et al. 2015; Thontiravong et al. 2015). A strain of DTMUV, KPS54A61, was later identified as causing duck mortalities and reduced egg production in some provinces in Thailand. More recently, it has been proposed that this virus is the same as the DTMUV found in the ducks in China, to which it is 98% homologous (Chakritbudsabong et al. 2015).

Australian status

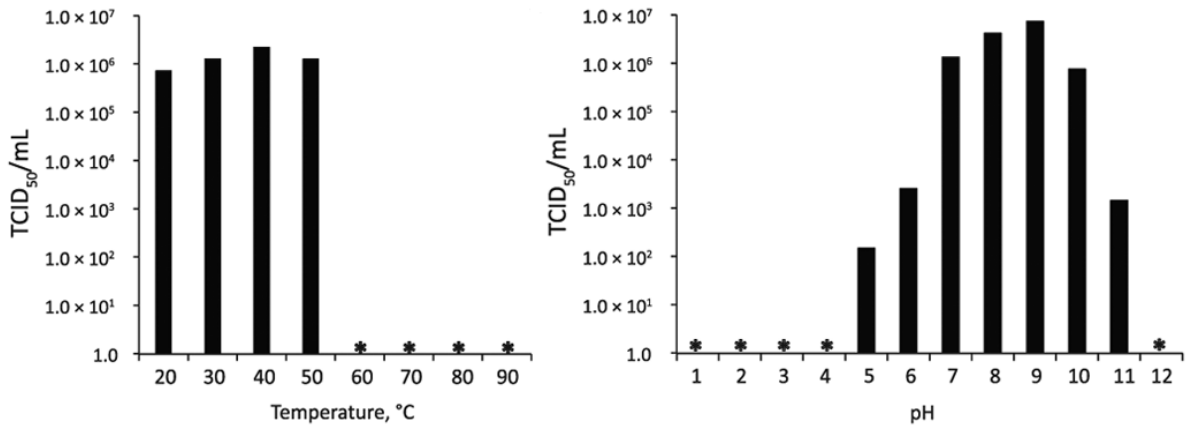
Tembusu viruses, including DTMUV, have not been reported in Australia, there is no active surveillance, and tembusu viruses of ducks are not nationally notifiable.

Agent properties

There is no information on the chemo-physical properties of the TMUV. However, flaviviruses share remarkable similarities in virion properties, structure, and genome organisation but vary in biological and antigenic properties (Goo et al. 2016; Kuhn et al. 2002). As the flaviviruses are enveloped viruses and composed of 17% lipids by weight, they are stable in an alkaline environment of pH 8 under laboratory (in vitro) conditions but are sensitive to treatment with organic solvents and detergents, and to high temperature.

In general, flaviviruses are typically inactivated by temperatures above 56°C for at least 30 minutes, in solutions of pH less than or equal to 6, by ultraviolet light and by gamma-radiation, and are known to be susceptible to a range of disinfectants (Charrel et al. 2016). Because it was thought that Zika virus, also a flavivirus, was more stable than other flaviviruses, Müller et al. (2016) assessed the environmental stability of Zika virus to heat and change in pH. The virus was stable at temperatures up to 50°C but lost all infectivity at temperatures of greater than 60°C (Figure 6). They also observed that Zika virus infectivity was highest after adjusting the stock to a pH of approximately 9 but adjusting to pH 12 or to less than pH 4 abrogated infectivity (Figure 6).

Figure 6 Inactivation and environmental stability of Zika virus



(from Müller et al. 2016).
Left graph - Zika virus was incubated for 5 min at indicated temperatures. Temperatures >60°C inactivated the virus. Right graph - Stocks were adjusted to indicated pH values and incubated for 10 min. pH levels <4 or >11 inactivated the virus.

Bovine viral diarrhoea virus, a pestivirus which is also a genus within the family Flaviviridae, is enzootic in most cattle populations throughout the world and is present throughout the body of persistently infected cattle. The virus in whole and ground muscle meat was consistently inactivated when cooked to temperatures greater than or equal to 75°C (Bratcher et al. 2012).

4.2.3 Presence in duck meat

There is no specific information available on DTMUV in duck meat.

Diseased meat ducks, especially 20–40 day old ducklings, typically develop nervous system disorders including unsteady standing, falling and quivering. Older ducks show less severe clinical signs, except for breeder or egg-laying ducks which show severe drop in egg production. Histology has revealed evidence of viral encephalitis. However, necropsy of female infected ducks consistently showed severe ovarian haemorrhage, ovaritis, with occasional ruptured ovarian follicles and peritonitis. Enlarged spleens and leg muscle hemorrhage were occasionally noted (Han et al. 2016). High levels of DTMUV have also been identified in tracheal cells (Cha et al. 2013).

Wu et al. (2014) observed that DTMUV in adult male and female ducks was detectable in most of the parenchymatous organs as well as the oviduct and intestinal tract from days 1 to 7 after inoculation. Viral titres were maintained at high levels for at least 9 days in the spleen, kidney, bursa of Fabricius, brain and ovary. However, no virus was detected in any of these organs or tissues 18 days after inoculation. Wu et al. (2014) concluded that DTMUV causes systemic infection in male and female ducks, and is capable of replication in most organs, with the exception of the ovaries and testes.

4.2.4 Pathogenesis

Flaviviruses enter cells by receptor-mediated endocytosis after attaching to the cell surface. The E protein involved in this process also plays a role in the immune response, including as a target for antibodies (Liu et al. 2013; Murphy et al. 1999). The virus replicates rapidly in cells with cytopathic effects (Murphy et al. 1999). Cells favoured by flaviviruses include cells involved in immunity such as macrophages, monocytes and dendritic cells (Liu et al. 2013). In particular, the spleen has been identified as one of the main target organs for virus replication, based on the appearance at post-mortem (Jiang et al. 2012). There is also a post-mortem reduction in

lymphocyte numbers in the spleen (Wang et al. 2014). In the case of the ovary, the virus mainly affects the follicle cells, causing degeneration and necrosis of these cells and haemorrhage and congestion in the follicular cavity itself (Wang et al. 2014).

DTMUV is present in the blood 4 hours after infection and ducks begin shedding the virus in the faeces around 8 hours after infection (Wang et al. 2014). Clinical signs may be evident 2 to 3 days later (Yan et al. 2011).

4.2.5 Diagnosis

Clinical signs

Sudden onset and rapid spread are features of the disease caused by DTMUV (Zhang et al. 2017). Anorexia is usually the first clinical sign, becoming evident within 2 to 3 days of experimental inoculation, or 5 days following contact with infected ducks (Yan et al. 2011). Within 10 days, egg production drops to lower than 10% (Liu et al. 2013). Other clinical signs include rhinorrhoea, diarrhoea, pyrexia and reduced growth. Neurological signs such as paralysis, ataxia and behavioural changes are also common (Jiang et al. 2012; Liu et al. 2013; Zhang et al. 2017). In drakes, the pathological effects on the testes can lead to an absence of spermatogenesis (Wu et al. 2014).

The age of the infected duck can influence the severity of signs, with very young ducks showing more severe clinical signs and reduced clearance of the virus (Li et al. 2015a). In the outbreaks in Thailand, clinical signs were evident in broiler ducks over 3 weeks of age and in laying ducks when in lay (Thontiravong et al. 2015). The Malaysian Perak virus outbreak was characterised by neurological signs including lameness, ataxia and progressive paralysis, which were most likely to be observed in young ducks (4 to 7 weeks of age) (Homonnay et al. 2014).

Morbidity can approach 90% or even 100% in infected populations (Liu et al. 2013; Zhang et al. 2017). Mortality in the Chinese outbreaks was reported to range from 5% to 30%, depending on the supportive treatment provided, with seroconversion occurring in the surviving birds (Liu et al. 2013). A mortality rate of 7.5% was observed in an experimentally infected flock of 80 9-week-old shelducks (most ducks in genus *Tadorna*) (with 10% mortality in a smaller experiment carried out earlier) (Yan et al. 2011). In the outbreaks in Thailand, mortality was as high as 50% in some groups, with a strong correlation with secondary bacterial infection (Thontiravong et al. 2015).

Pathology

Post-mortem examinations of ducks in the Chinese outbreaks revealed ovarian pathology such as ovaritis, ovarian haemorrhage and regression, with or without ruptured follicles and peritonitis (Liu et al. 2013). Lymphocyte infiltration in the ovaries and portal area interstitial inflammation were also observed (Tang et al. 2012). However, there were often lesions affecting other organs, including severe splenic necrosis or enlargement, pulmonary haemorrhage, moderate necrosis of the brain tissue, and nephrosis (Liu et al. 2013; Yan et al. 2011). Similar ovarian and splenic pathology was seen in ducks in Thailand, with multifocal gliosis and perivascular cuffing identified on histopathological examination of the cerebellum and spinal cord (Thontiravong et al. 2015). In the Perak virus outbreaks in Malaysia, non-purulent panencephalomyelitis was the main feature of the microscopic pathological lesions found in the brain and spinal cord of infected Pekin ducks (Homonnay et al. 2014).

Chicken embryos died 3 to 5 days after experimental inoculation of eggs with the virus. Severe cutaneous haemorrhage was evident in the embryos on examination (Thontiravong et al. 2015).

Testing

In endemic regions, presumptive diagnosis can often be made based on the clinical signs, rapid spread through the flock and post-mortem findings (Zhang et al. 2017).

Real-time RT-PCR and reverse transcription loop-mediated isothermal amplification assays can be used to definitively diagnose the virus (Liu et al. 2013). Both methods can detect DTMUV within 30 minutes, with the latter having the advantage of being inexpensive and usable in the field compared to RT-PCR, which is more expensive and requires a higher level of skill to perform (Jiang et al. 2012). Both the RT-PCR and reverse transcription loop-mediated isothermal amplification assay tests have high sensitivity and specificity, with the specificity determined through analysis with other flaviviruses with no evidence of a cross-reaction (Jiang et al. 2012). ELISA tests have also been used, but like RT-PCR, these have the disadvantage of being expensive and requiring laboratory facilities (Wang et al. 2014). A latex-agglutination assay has also been employed and is reported to be specific for DTMUV (Wang et al. 2014).

Duck blood remains positive for viral nucleic acid for up to 4 weeks following infection, while faeces can be positive for up to 13 days, suggesting they might be used for fluid sampling (Wang et al. 2014). The spleen has been identified as one of the preferred targets for replication of the virus and therefore a good site for post-mortem tissue sampling due to the high viral loads present in infected birds, although the virus can no longer be detected in the spleen by 18 days after infection (Jiang et al. 2012; Wu et al. 2014). The kidneys, brain, ovaries and bursa of Fabricius can also be used, with similar constraints on the sampling period (Wu et al. 2014).

4.2.6 Treatment

There is no effective treatment for DTMUV infection.

4.2.7 Control

Currently there is no commercial vaccine available. In recent years there has been promising research in the development and testing of vaccines for protection against DTMUV. These include an inactivated vaccine injected subcutaneously twice in the neck of ducks, a live attenuated vaccine and an oral DNA vaccine (Huang et al. 2018; Li et al. 2014; Zhang et al. 2017). Combination vaccines are also being developed and trialled, including a trivalent vaccine to prevent avian influenza (H5N1), DTMUV, and duck enteritis virus infections in ducks using the CRISPR/Cas9 system (Zou et al. 2017).

4.2.8 Current biosecurity measures

There is no biosecurity measure against DTMUV for the importation of fertile duck eggs, however the virus has not been identified in countries approved for sourcing.

4.2.9 Conclusion

- DTMUV is not an OIE-listed disease nor is it nationally notifiable in Australia.
- DTMUV is an emerging disease, with outbreaks resulting in significant morbidity and mortality first reported in China in 2010. Since then outbreaks have been reported in Thailand and Malaysia.
- DTMUV is present in Thailand but has not been identified in Australia.
- DTMUV can spread rapidly within and between duck flocks, causing up to 100% morbidity and 30% mortality. It spreads via mosquito vectors, direct transmission and/or airborne transmission.
- There is no treatment or commercially produced vaccine currently available.

- Prevention and control rely mainly on early diagnosis using both serologic and molecular methods, disinfection of premises, removal of birds from the infected environment and depopulation.
- DTMUV causes systemic infection in male and female ducks, and is capable of replication in most organs. Cells favoured by DTMUV include cells involved in immunity such as macrophages, monocytes and dendritic cells.
- DTMUV is no longer present in ducks that survive within 18 days of onset of infection.
- Given that flaviviruses are typically inactivated by temperatures above 56°C for at least 30 minutes, Zika virus is inactivated after 5 minutes at 60°C and bovine viral diarrhoea virus is inactivated when core temperature reaches 75°C, it is expected that DTMUV would be inactivated when heating duck meat to a minimum core temperature of 70°C for a minimum of 8.2 minutes.

Based on the above information cooking as defined in the scope at section 1.3 of this document (70°C for 8.2 minutes) will inactivate any DTMUV that may be present in meat of ducks. Therefore, the department concluded that further risk assessment of duck Tembusu virus was not required.

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4.3 Duck virus enteritis (duck plague)

4.3.1 Background

Duck virus enteritis (DVE) or duck plague is caused by a contagious herpesvirus, the duck virus enteritis virus (DVEV). DVE is an acute, though sometimes chronic disease of waterfowl of the family Anatidae (ducks, swans and geese). Natural infections occur in ducks from 7 days of age to adults, and causes significant economic losses (Hansen & Gough 2007). Ducklings infected with a low virulence strain often suffer from secondary bacterial infections due to the virus having an immunosuppressive effect (Campagnolo et al. 2001; Shawky, Sandhu & Shivaprasad 2000). Laying ducks can suffer from significant reductions in egg production and adult males may suffer from prolapse of the penis (Woolcock & Tsai 2013).

DVE was first identified in 1923 in the Netherlands, and in the 1940s was termed 'duck plague' when the disease could not be reproduced in species other than Anatidae (Woolcock & Tsai 2013). The virus has subsequently been confirmed in Austria, Belgium, Canada, China, Denmark, England, France, Germany, Hungary, India, Poland, Thailand, the United States of America and Vietnam (Dhama et al. 2017).

DVE is not an OIE-listed disease, having been removed from the list in 2010 due to its limited spread across country borders. DVE is nationally notifiable.

4.3.2 Technical information

Epidemiology

Susceptible species

With natural infection, members of the Anatidae family are the only species susceptible to DVEV; however, susceptibility varies greatly among the different species (Friend 1999). High mortalities have been reported in a variety of domestic ducks (*Anas platyrhynchos*) and Muscovy ducks (*Cairina moschata*). Despite the widespread distribution and frequent occurrence of duck plague in captive and wild waterfowl in North America, wild waterfowl have only been infrequently infected.

Mallards are more resistant to the lethal effects yet are considered to be a possible natural reservoir of infection (Dhama et al. 2017). In the northern hemisphere, Mallards are usually migratory, but in the southern hemisphere, where they have been introduced, they are not known to be migratory.

Other types of aquatic birds do not become infected. Outbreaks in domestic ducks generally occur when they have access to aquatic environments co-habited by wild ducks, geese and/or swans (Woźniakowski & Samorek-Salamonowicz 2014).

The virus only replicates in cell cultures obtained from the Anseriformes and Galliformes orders. The virus can be modified to grow in chickens less than 2 weeks of age as well as embryonating chicken eggs (Campagnolo et al. 2001; Hansen & Gough 2007).

Modes of transmission

Transmission occurs by direct contact between susceptible and infected birds, with oral transmission being the most common route. Transmission via indirect contact can also occur with contaminated water and movement of infected birds being the most likely cause of exposure in both domestic and wild populations. Wild ducks have been implicated as the carriers, thus playing a crucial role in DVEV transmission within and between continents. Both domestic and wild populations have been found to shed the virus periodically when recovering from disease (Campagnolo et al. 2001; Dhama et al. 2017; Woolcock & Tsai 2013).

Incubation period
Ducks as young as 1 week old can be infected by DVEV (Dhama et al. 2017). The birds can become infected as a result of direct exposure to infected birds or indirectly from contaminated environments, including water. The incubation period of DVEV ranges from 3 to 7 days, with a maximum of 14 days (Campagnolo et al. 2001; Woolcock & Tsai 2013). After overt clinical signs appear, death occurs within 1 to 5 days. The mortality and morbidity of the flock can range between 5 and 100% depending on the health of the flock and the virulence of the strain. Those that survive can become carriers of the infection for several years, with infection latent in the trigeminal ganglion, where upon re-activation, shedding occurs periodically (Burgess & Yuill 1983).

A study by Burgess et al. (1979) found that all species tested (including Mallard, black, pintail and gadwall ducks) had a high frequency of DVEV shedding. This study also determined that oral erosions did not signify the occurrence of shedding, rather that they are an indicator of persistent infection, which may occur latent in the trigeminal ganglion. Long term persistence of the virus has been suggested with reports of DVEV being isolated from Pekin ducks 45 days after initial infection. Butterfield and Dardiri (1969) also recovered DVEV 17 days after infection from the cloacae of clinically healthy waterfowl (Burgess, Ossa & Yuill 1979; Converse & Kidd 2001).

Distribution and prevalence
DVEV is considered endemic in domestic waterfowl in many Asian countries including Thailand and Vietnam (Hansen & Gough 2007; Woolcock & Tsai 2013).

Australian status
DVEV has not been reported in Australia and there is no active surveillance. However, infection with DVEV is nationally notifiable.

Agent properties
The disease is caused by DVEV, or Anatid herpesvirus 1, which is a double-stranded DNA virus. It is classified as the type strain of the genus *Mardivirus*, in the subfamily Alphaherpesvirinae and family Herpesviridae (Converse & Kidd 2001). Differences in virulence occur amongst strains, though immunologically they are identical (Akter et al. 2004; Dhama et al. 2017).

Herpesviruses are enveloped virus, with the envelope containing considerable amounts of lipid. Because of this structure, herpesviruses are susceptible to chemical agents, especially those that are lipid solvents (Maris 1995).

Salts and proteins affect the thermostability of herpesviruses, being thermolabile in tissue cultures, extremely thermosensitive in isotonic solutions, but thermostable at 50°C by simple dilution in distilled water, and by 1 M Na_2SO4 or Na_2HPO4 (Wallis & Melnick 1965). Strains of herpesvirus hominis have been reported to resist thermal inactivation in the presence of sodium ions (Wallis & Melnick 1965).

Using heat during inactivation experiments, 10 minutes at 56°C or 90 to 120 minutes at 50°C, nullifies the infectivity of DVEV (Sandhu 2003). Laboratories handling live viruses generally recognise that herpesviruses are inactivated within 30 minutes at 56°C (Public Health Agency of Canada 2011).

A live attenuated DVEV strain used in a vaccine was observed to be non-infective above 42°C within 2 hours and had become unstable within 2 hours above 65°C (Makhija & Kumar 2017).

Short-term pasteurisation of human breast milk, that is, 5 seconds at 72°C, was able to completely destroy the infectivity of cytomegalovirus, a virus in the order Herpesvirales (Hamprecht et al. 2004). Furthermore a cell culture supernatant containing koi herpesvirus was

inactivated completely when heated to temperatures above 50°C for 1 minute (Kasai, Muto & Yoshimizu 2005).

4.3.3 Presence in duck meat

The virus replicates mainly in the mucosa of the digestive tract, then spreads to the bursa of Fabricius, thymus, spleen and liver where it continues to replicate. More specifically, DVEV replicates in the epithelial cells, lymphocytes and macrophages in these organs (Dhama et al. 2017).

Immunoperoxidase staining of a DVEV protein, UL51, enabled the identification of DVEV antigen in the bursa of Fabricius, thymus, spleen, liver, oesophagus, small intestine and large intestine of DVEV-infected ducks, and less so in the Harder's glands, glandularis ventriculus, cerebrum, kidney, lung, pancreas and myocardium. The DVEV protein was also expressed in the cytoplasm of various types of cells, most abundantly in the cytoplasm of lymphocytes, reticulum cells, macrophages, epithelial cells and hepatocytes. However, throughout the infection process UL51 was not seen in the muscle (Shen et al. 2010).

4.3.4 Pathogenesis

The pathologic response is dependent upon age, stage of infection, sex, species affected as well as virus virulence and intensity (Leibovitz 1969; Leibovitz & Hwang 1968; OIE 2018a). Mortality rates differ between isolates of DVEV as well as between and amongst species. The original O strain of the virus studied by Jansen (1961) had 100% mortality, though a virus recovered from a 1959 outbreak in the Netherlands caused less than 80% mortality, which could suggest that virus virulence can decrease over time (Hansen & Gough 2007).

Replication of DVEV occurs within the host cell's nucleus. During the late stages of infection, the replicated DNA is packaged into capsids so that it can move into extracellular spaces (Johnson & Baines 2011).

Initial lesions observed in ducks were in the stratified squamous mucosa of the gastrointestinal tract (Proctor 1975). Lesions then develop in the submucosal lymphoid tissue, intestinal villous lamina propria and intestinal crypt epithelium. Infection then proceeds towards the thymus, bursa of Fabricius, spleen and liver. Lesions in these tissues have also been described (Dhama et al. 2017). After viral replication, the epithelial cells containing the viral antigens begin to degenerate (Proctor 1975).

Degenerative changes of parenchymatous organs and generalised haemorrhages are the result of vascular damage induced by DVEV. It has been suggested that lymphoid depletion caused by the necrosis and apoptosis of lymphocytes may lead to the virus causing immunosuppression, which would explain the high presence of secondary infections seen in natural outbreaks (Dhama et al. 2017).

4.3.5 Diagnosis

Diagnosis is based on a combination of clinical signs, gross pathology and histopathology, supported by identification of the virus by either isolation or polymerase chain reaction (PCR) (OIE 2018a).

Clinical signs

High mortality is commonly the first observation in domestic flocks as there are few clinical signs. Sometimes carcasses are found floating on the water surface (Dhama et al. 2017).

Further signs of infection are extreme thirst, anorexia, ataxia, nasal discharge, ruffled feathers, watery diarrhoea, half-closed and swollen eyelids, droopiness, solid vents, inability to stand and tremors of head, neck and body. Clinical signs may develop as the infection progresses (Akter et

al. 2004; OIE 2018a; Woolcock & Tsai 2013). Sick birds may maintain an upright stance by using their wings for support, but their overall appearance is one of weakness and depression (OIE 2018a).

Young ducklings may develop conjunctivitis, nasal exudate, lacrimation, blood-staining of the vent, dehydration and weight loss (Akter et al. 2004; OIE 2018a; Woolcock & Tsai 2013).

Pathology

Internally, infection is characterised by lesions of lymphoid organs, tissue haemorrhages, vascular damage, digestive mucosal eruptions and degenerative changes in parenchymatous organs (Woolcock & Tsai 2013; Zhang et al. 2010).

Lesions can range from 1 to 10mm in length and are found through the entire digestive tract from the oral cavity to the cloaca. A haemorrhagic surface covers the wall of the lumen of bursa of Fabricius, and there are haemorrhages in the liver in the early stages before the liver becomes a dark bronze colour later in the infection process (OIE 2018a; Woolcock & Tsai 2013).

DVEV affects ducklings slightly differently to mature birds, with prominent lymphoid lesions and less tissue haemorrhage, while mature domestic ducks have the primary lesions on the reproductive tract as well as tissue haemorrhages (Woolcock & Tsai 2013). This difference may account for the higher rate of secondary bacterial infections in ducklings.

Parenchymatous and lymphoid organs as well as the gastrointestinal tract will show mucosa with necrotic degenerative changes and disseminated intravascular coagulopathy. Petechiae may be found over the surface of the intestines, kidney, pancreas, liver and lungs, with extravasations of blood found in visceral organs and the myocardium. The ovarian follicles of laying females may be discoloured and deformed, and haemorrhaging of the ovary which fills the abdominal cavity may be present (Woolcock & Tsai 2013).

The vascular damage caused by DVEV results in degenerative changes for the affected tissues. Those without gross lesions may have microscopic changes such as haemorrhages in capillaries within the intestinal villi as well as in venules near the liver and lungs (Woolcock & Tsai 2013).

Testing

The OIE Terrestrial Manual provides recommendations for diagnostic tests for DVEV in ducks (OIE 2018a).

Clinical and gross findings may provide a presumptive diagnosis; however, detection of viral antigen or virus isolation is necessary for confirmation (Woolcock & Tsai 2013). The real-time PCR and conventional PCR are sensitive tests for identifying DVEV antigen.

The virus isolation test, along with PCR, is best for confirmation of clinical cases using samples of liver, spleen or kidney tissue. However, the usefulness of the virus isolation test is severely limited for confirming population freedom from infection.

Antigen tests not described in the OIE Manual include the antigen-capture ELISA which is reported to be a sensitive and reliable method for detection of the DVEV antigen (Dhama et al. 2017; Jia et al. 2009).

Another method of detecting DVEV is a loop-mediated isothermal amplification assay. It is a simple yet specific method that is superior to the PCR method and can be used for on-farm diagnosis (Ji et al. 2009).

The recommended tests for determining population freedom are the microtitre plate virus neutralisation assay and neutralisation assay in duck embryos. These are also the recommended

tests for determining the post vaccination immune status in individual animals or populations (OIE 2018a).

4.3.6 Treatment

There is no effective treatment for DVEV infection.

4.3.7 Control

In most European countries and the United States of America, both live attenuated and inactivated vaccines are used in broiler and breeder ducks over 2 weeks old (Dhama et al. 2017). The live attenuated vaccines are not ideal because of the risk of latency. However, there is no evidence of spread by contact from vaccinated to unvaccinated ducklings.

4.3.8 Current biosecurity measures

There are current biosecurity measures in place for this disease in the *Policy review: importation of hatching (fertile) duck eggs from approved countries* (Biosecurity Australia 2009). However, as the duck egg policy review required that the youngest bird not be less than 40 weeks old when eggs are collected for export, these measures are not relevant for meat ducks which are slaughtered from 7 to 8 weeks of age.

4.3.9 Conclusion

- DVE is present in ducks in Thailand but has not been identified in Australia. It is not an OIE-listed disease but is nationally notifiable in Australia.
- DVEV is transmissible by direct contact with oral transmission being the most common route. Transmission via indirect contact can also occur through contaminated water.
- Whilst DVEV is not found in muscle tissue it is found in most organs within the abdominal and thoracic cavities, head and in some white blood cells. There is the potential for infected white blood cells to be present in muscle tissue.
- Using heat during inactivation experiments, 10 minutes at 56°C or 90 to 120 minutes at 50°C, renders DVEV inactive.
- In general, laboratories handling live viruses recognise that herpesviruses are inactivated within 30 minutes at 56°C.

Based on the above information cooking as defined in the scope at section 1.3 of this document (70°C for 8.2 minutes) will inactivate any DVEV that may be present in meat of ducks. Therefore, the department concluded that further risk assessment of duck virus enteritis was not required.

4.3.10 References

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4.4 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 2018c). 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, evidence of natural infection has been detected in ducks on limited occasions (Jeon et al. 2008; McFerran et al. 1980). IBD was first recognised as a disease in chickens in 1957 and was named Gumboro disease after the area in the United States where IBDV was first identified (Cosgrove 1962).

There are 2 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 2 major groups antigenically—classic and variant, and into 3 groups pathogenically—attenuated (vaccine), classic virulent and very virulent. Pathogenic strains are confined to serotype 1 and reassortants of serotypes 1 and 2 (Ismail & Saif 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 only considering IBD viruses that are exotic to Australia, including the very virulent IBDV (vvIBDV) 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

Epidemiology

Susceptible species

Yamada et al. (1982) conducted research on 121 specific pathogen free white Pekin ducks ranging in age from 1 to 180 days that were experimentally infected with 1 of 2 strains of IBDV serotype 1 either orally or nasally. Yamada et al. (1982) also infected duck and chicken embryonated eggs. Virus material was prepared from infected bursae of Fabricius of chickens. Virus could multiply in duck embryos and fibroblast cells, and antibodies were detected using Agar-gel precipitation tests. All experimentally infected ducks remained clinically normal. All ducks tested negative 11 weeks post infection. The antibody titre of the serum samples from 30- and 180-day-old ducks began to rise one week after infection. Antibodies were detected over the 11-week period of observation and antibody responses were lower in 30-day-old ducks than in 180-day-old ducks. The antibody responses to oral inoculation in 180-day-old ducks were the same as in chickens. No virus could be isolated from any of the ducks from body tissues or intestinal contents. Liver, spleen, kidney and heart showed no microscopic changes that could be attributed to IBDV. McNulty et al. (1979) also determined that IBDV can be grown in duck embryo cells.

Serotype 1 isolates have been obtained from both healthy (collected from Pekin duck faeces) and dead waterfowl (Jeon et al. 2008; McFerran et al. 1980). McFerran et al. (1980) identified a serotype 1 IBDV in fowl and ducks, virtually identical to a commercial chicken IBDV vaccine

strain. Jeon et al. (2008) examined 13 wild spot-billed ducks and 17 mallard ducks from Korea that were found dead (cause of death pesticides or road kill). The caecal tonsils of 2 mallard ducks tested positive for IBDV by RT-PCR and virus isolation was completed using chicken embryos.

Jeon et al. (2008) collected samples of caecal tonsil and kidney from 107 wild birds found dead of apparently non-infectious causes (e.g. killed by car) throughout the Republic of Korea. The samples were tested for IBDV using RT-PCR and 5 birds were positive including 2 out of 20 mallard ducks. The authors analysed the strains and concluded they were closely related to the IBDV strains isolated from domestic chickens in endemic regions. The authors used a strain isolated from a black-billed magpie (closely related to the strain isolated from the mallard ducks) to experimentally infect specific pathogen free (SPF) chickens with IBDV via the intranasal route and produced typical IBD in the chickens. The authors also noted that this showed a probable role of wild birds in the epidemiology of IBDV in endemic areas, however, the blacked-billed magpie is commonly found around chicken farms in the Republic of Korea and could not exclude the possibility the bird was infected though contact with contaminated sources from the poultry industry.

Wahome (2018) as part of an unpublished thesis surveyed village poultry in Kenya for IBDV. The survey included 32 indigenous ducks free ranged and in mixed flocks and found that 6% (2/32) of ducks and 65% of chickens (63/97) were positive for antibodies to IBDV using ELISA, and for adult birds (8 months old and over), 85.71% (18/21) of chickens and 6.67% (1/15) of ducks were found to be seropositive. The author concludes that ducks and turkeys play an important role in the natural maintenance and spread and IBDV. However, based on the resistance of IBDV and local farming practices (including limited vaccination of birds, selling suspected infected and sick birds, and no biosecurity measures by poultry traders between farm visits) it appears more likely that IBDV is being maintained by the local chicken population through those practices. Together with high amounts of IBDV in the environment, and close contact with chickens and related workers, it is likely that ducks are being exposed and seroconverting as a result.

McFerran et al. (1980) were unable to detect antibodies in ducks from which IBDV was isolated above. Okoye et al. (1990), using an IBDV serotype 1 isolate to experimentally infect ducks, did not detect neutralising or precipitating antibodies, viral antigen in the bursa, virus in the bursa, spleen and liver, nor any clinical signs or gross or microscopic lesions. Wang et al. (2007) recovered an IBDV isolate from a pool of ducks and used it to infect chickens but this did not produce any mortalities nor significant clinical signs of IBDV or pathology other than damage to the bursa. Geetha et al. (2008) observed seropositivity to IBDV in 2 of 183 samples from ducks prior to slaughter. Ibu et al. (2000) did not detect IBDV antibodies in the wild ducks they sampled.

Karunakaran et al. (1992) reported clinical signs and high mortality (>50%) of ducklings in a duck breeding unit, attributed to IBDV. However, this is the only report of clinical signs (which were only partly consistent with IBDV)—let alone mortalities in ducks—due to IBDV found in the literature including in controlled experimental conditions where high doses of virus have been directly inoculated into ducklings. Given the report only considered Newcastle disease and mycotoxins as potential differentials to exclude, it is unclear whether other possibilities more commonly known to cause disease in ducks such as duck hepatitis were investigated as the actual causative agent.

Modes of transmission
IBDV is extremely hardy and highly contagious. In chickens it is 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 chickens nor of vertical transmission (Eterradossi & Saif 2013).

Distribution and prevalence
IBDV occurs in all major poultry producing areas worldwide including Thailand.

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). 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).

Hollmén et al. (2000) detected positive titres (greater than or equal to 1:16) in 75% (between 19% and 96% at different locations) of eider ducks in the Baltic Sea. Eddy (1990) detected neutralising antibodies to experimental and natural (sourced from ducks at the time of slaughter) IBDV exposures in the UK. In that study, the ducks were inoculated by a simultaneous combination of intranasal and intramuscular inoculation and then again 3 weeks later, yet no clinical signs were observed. Oluwayelu et al. (2007) detected precipitating antibodies in naturally exposed ducks in Nigeria with 19.1% being positive and the highest titre being 1:64.

Australian status
Antibodies to IBDV serotype 1 have been detected in Australian flesh-footed shearwaters, silver gulls and black ducks (Wilcox et al. 1983).

Agent properties
IBDV is a member of the Birnaviridae family, Avibirnavirus genus (Dobos et al. 1979; ICTV 2014b; Ignjatovic & Prowse 1997). It is a single shelled, non-enveloped virion with a genome consisting of 2 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 1 study, reduction of the infectivity by 1 log10 took 18.8 minutes at 70°C, 11.4 minutes at 75°C and 3 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 1997).

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 log10 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 log10 within 2 hours at 23°C and 4°C, while SDF and Virkon applied at 23°C and 4°C reduced IBDV by 5 log10 within 15 minutes (Guan et al. 2014).

4.4.3 Presence in duck meat

There is no specific information available on IBDV in duck meat.

4.4.4 Pathogenesis

Detailed pathogenesis in chickens is described in the *Chicken meat IRA* (Biosecurity Australia 2008). Only details as they compare to research in ducks are presented below.

Oladele (2010) experimentally infected groups of chickens, turkeys and ducks with IBDV via conjunctival instillation. No changes were observed in ducks other than a weak antibody response. The authors concluded that the bi-phasic gross pathological changes including bursal enlargement observed in chickens (but not in turkeys and ducks) as well as the bi-phasic viraemia, lymphopeania and increased antigen concentrations reported in earlier studies have further revealed the importance of 2-phase systemic changes observed only in chickens in the pathogenesis of IBD for the establishment of a successful clinical disease. Oladele (2010) further concluded that the absence of gross pathology in ducklings portrayed unresponsiveness or refractoriness in this species to IBDV infection.

4.4.5 Diagnosis

Clinical signs

Clinical signs in chickens are described in the *Chicken meat IRA* (Biosecurity Australia 2008). IBDV infection in ducks does not produce clinical signs.

Pathology

Pathology in chickens is described in the *Chicken meat IRA* (Biosecurity Australia 2008). IBDV infection in ducks produces very little, if any, pathology.

Two groups of SPF white Pekin ducks inoculated twice (3 weeks apart) by nasal instillation for the first group and intramuscular injection for the second group showed seroconversion but no clinical signs. Tsai et al. (1996) found no clinical disease, mortality or gross lesions in the bursa of Fabricius in 1 and 30 day old experimentally infected ducklings, however, mild to moderate histopathologic lesions were observed in the 1–day–old ducklings.

In 2010 Oladele experimentally infected (conjunctival instillation) 5-week-old chickens, turkeys and ducks with serotype 1 IBDV. While clinical signs were observed only in chickens, muscular haemorrhage and increase in red blood cells, which was adopted in place of a direct measurement of bursal enlargement, was observed in both turkeys and chickens. No changes were observed in ducks other than the detection of antibodies, which occurred in 2 of 8 ducks (5/8 turkeys and 10/10 chickens) using ELISA, while none were positive using agar gel immunodiffusion (AGID) (precipitating) (0/8 turkeys and 10/10 chickens). The author concludes that antibody response in ducks is evidence of infectivity, though very poor. Okoye et al. (1990) did not detect any clinical signs or evidence of pathology in ducks experimentally infected with IBDV serotype 1.

Testing
Presence of the virus is confirmed by detection of the virus in tissues using immunological or molecular methods. Demonstration of specific antibodies to IBDV also demonstrates either current or prior infection.

Exposure to IBDV can be confirmed by antibody identification in unvaccinated birds or by detecting viral antigen or genomic RNA in tissues (OIE 2019a). 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 2019a). 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 2019a).

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 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 2019a). 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 2000, 2002).

4.4.6 Treatment
There is no effective treatment for IBDV infection.

4.4.7 Control
Vaccination is used routinely for the control of IBDV in chickens, however, Australian vaccines are not protective against exotic antigenic strains or very virulent strains (Ignjatovic, Sapats & Gould 2001).

4.4.8 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*. These include certification that IBDV is not present and that any clinical evidence of disease or deaths above 0.5% have been investigated.

Current biosecurity measures are also in place for this disease from the *Chicken meat IRA*. 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).

4.4.9 Conclusion

- IBD is primarily a disease of chickens, however, detection of antibodies in ducks, or the inability to, has occurred in several research papers. In all cases where antibodies were detected and quantified there were low titres compared to those detected in chickens.
- Surveys have shown that antibodies to IBDV exist in most avian species.
- Available literature suggests that IBDV serotype 1 is able to infect ducks and produce an antibody response in some instances. However, no clinical or pathological signs have been observed to date (other than mild to moderate histopathological lesions in a single experimental study involving 1–day–old ducklings).
- Virus has been isolated in 1 study of wild ducks from faeces and caecal tonsils. Experimental infection studies in ducks have failed to isolate virus.

Based on the above information, while it is possible for ducks to be infected and produce a low level antibody response, this is uncommon and results from exposure to large amounts of virus in experimental settings. As infection does not appear to result in replication and shedding of viruses in all but the rarest cases the department concluded that the risk of entry of IBDV in cooked duck meat from Thailand was negligible and therefore further risk assessment of IBDV was not required.

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4.5 Waterfowl parvoviruses

4.5.1 Background

Waterfowl parvoviruses are members of the Parvoviridae family. The International Committee on Taxonomy of Viruses (ICTV) currently classifies the waterfowl parvoviruses, Muscovy duck parvovirus (MDPV) and goose parvovirus (GPV), as a single species (namely, Anseriform dependoparvovirus 1), which is classified into the genus Dependoparvovirus in subfamily Parvovirinae due to similar genetic properties and evolutionary origins (ICTV 2014a).

Variants of both GPV and MDPV have appeared in waterfowl and include the duck parvovirus (DPV), a recently identified variant of the GPV (Jordan et al. 2016; Li et al. 2018; Woźniakowski, Kozdruń & Samorek-Salamonowicz 2009; Zádori et al. 2006). A variant of the MDPV was also recently identified in Muscovy ducks in China and found to be a recombination between classical MDPV and GPV strains (Wang et al. 2017).

The GPV and MDPV are antigenically related (Mahardika et al. 2016; Yu et al. 2016) and possess approximately 85% homology in overall protein sequences. Nonetheless, GPV and MDPV are distinct, and as a result, any antibody-driven cross protection between these 2 related viruses is limited (Yu et al. 2016).

Waterfowl parvoviruses are highly contagious with high mortalities in young birds, often causing between 70 and 100% mortality in goslings and ducklings less than 4 weeks old (Zsak 2016). GPV causes Derzsy's disease, also known as goose hepatitis, goose plague or gosling plague, a disease characterised by watery diarrhoea, lethargy, anorexia, prostration, and weight loss in young Muscovy ducklings and goslings (Zsak 2016). MDPV affects only young Muscovy ducklings and mule ducks (Woolcock et al. 2000) and causes ascites, enteritis, myocarditis and hepatitis with high morbidity and mortality (Woźniakowski, Kozdruń & Samorek-Salamonowicz 2009). DPV causes similar clinical signs in mule ducks and Pekin ducks (Cherry Valley breed) (Chen et al. 2016). Young birds that survive infection and infected older birds show degenerative skeletal muscle myopathy and growth retardation characterised by the beak atrophy and dwarfism syndrome (Zsak 2016).

Vaccinating breeding flocks provides a high level of maternal antibody protection in the progeny until approximately 2 weeks of age (Zsak 2016).

Infection due to waterfowl parvoviruses is not OIE-listed nor is it nationally notifiable or subject to official control or eradication in Australia.

4.5.2 Technical information

Epidemiology

Susceptible species

Waterfowl parvovirus infects birds within the Anatidae family, namely geese, Muscovy ducks, swans (*Cygnus* spp), mule ducks and more recently Pekin ducks, especially those of the Cherry Valley breed (Li et al. 2018). GPV primarily infects geese and Muscovy ducks, and can be highly contagious in goslings under the age of 4 weeks (Yu et al. 2016). However, other breeds of ducks have shown susceptibility to variants of GPV. In 1989/1990 outbreaks, Muscovy ducks, mule ducks and Tsaiya ducks were susceptible to GPV (Chang et al. 2000; Liao et al. 1991). In 2014, a Malaysian study revealed that GPV was detected in Pekin ducks from Sarawak (Syamsiah et al. 2017). GPV antibodies have been detected in the sera of wild geese (Hlinak et al. 1998). Since 2015, a number of DPV strains have been identified in Pekin and mule ducks in northern China (Chen et al. 2016; Li et al. 2018).

Geese are resistant to MDPV infection; however, both GPV and MDPV can cause severe disease in Muscovy ducks and several hybrid duck breeds (Yu et al. 2016).

Evidence suggests that wild geese are implicated in the epidemiology of the disease as a high prevalence of GPV antibodies were detected in the sera of wild geese (bean geese and white-fronted geese) in Germany (Hlinak et al. 1998). In addition, in 2004, epidemiological investigations of the outbreaks in the United Kingdom and Sweden led to a concern that some migratory wild geese species (including the Canada goose (*Branta canadensis*)) may have been the source of introduction of GPV (Gough et al. 2005; Jansson et al. 2007). Although over 50 taxa of waterbirds have been identified in Australia (Kingsford & Porter 2009), the susceptibility of Australian wild waterfowl species to waterfowl parvoviruses is not known. However, the Canada goose (*Branta canadensis*), suspected to be a source of GPV in the UK and Sweden and originally introduced into Australia as an exotic waterfowl species, have been occasionally sighted in the wild (DPIRD 2017).

Modes of transmission

Waterfowl parvoviruses are excreted in large amounts in the faeces of infected waterfowl and can subsequently spread rapidly to susceptible birds by both direct and indirect routes (Yu et al. 2016). Goslings and ducklings can be infected through vertical transmission via eggs laid by infected breeder geese and ducks (Chen et al. 2016; Zádori et al. 2006; Zsak 2016). Infected ducklings hatching from eggs where the virus was vertically transmitted, are often the source of the virus when outbreaks of GPV occur in countries free of the disease (Zsak 2016).

GPV may cause subclinical infection in older geese which pass on the virus to their progeny. Recovered birds are thought to act as carriers, and latency may be established with vertical (egg) transmission of the virus and potentially further horizontal transmission (Irvine & Holmes 2010; Palya 2010).

Horizontal infection is faecal-oral and occurs between flock-mates through the digestive tract via virus contaminated water and feed (Zádori et al. 2006). There is no reliable scientific information about transmission via the respiratory system or the conjunctiva.

Due to its resistance in the environment, parvovirus can persist in buildings and on poorly cleaned and disinfected surfaces. This can result in transmission to subsequent flocks (Palya 2010). Machinery and other farm equipment can also spread the disease as the virus is very difficult to remove using conventional cleaning agents.

Incubation period

In unprotected goslings infected with GPV in their first 10 days of life, clinical signs develop 5 to 7 days post infection. The signs include inappetence, listlessness, anorexia, polydipsia, and diarrhoea. Deaths peak around 11 to 12 days post infection (Zádori et al. 2006). In susceptible neonatal birds, death may occur in 2 to 5 days (Irvine & Holmes 2010). Older geese and Muscovy ducks may develop subclinical infection (Irvine & Holmes 2010).

In 3 day old Muscovy ducklings experimentally infected with GPV, mortality reached 100% within 10 days and the virus was distributed to many tissues within 2 days after inoculation by mouth (Takehara et al. 1998). GPV DNA was detected by PCR in the duodenum and rectum from day 3 to day 28 post inoculation. This suggests that birds may excrete virus in their faeces for a long period of time (Takehara et al. 1998).

Egg transmission will lead to embryo death during incubation or hatching in an infected state, depending on the level of yolk-sac antibody (Zádori et al. 2006). There are reports that latency may be established with vertical transmission of the virus (Irvine & Holmes 2010).

Distribution and prevalence

Currently, the majority of northern hemisphere countries with intensively farmed waterfowl have GPV (Zádori et al. 2006). It has been reported to cause disease in geese and Muscovy ducks in China, France, Germany, Hungary, North America, Poland, Thailand, Taiwan and Vietnam (Chang et al. 2000; Deemagarn & Tangdee 2015; Irvine & Holmes 2010; Jordan et al. 2016; Mahardika et al. 2016; Syamsiah et al. 2017; Yu et al. 2016)

It was suspected that imports of large numbers of goslings and goose eggs from Thailand might have been involved in a 1982 outbreak of GPV in Taiwan (Chang et al. 2000).

MDPV was first isolated from Muscovy ducks in the west of France in 1989. The United States of America also experienced an outbreak of MDPV in 1997 (Woolcock et al. 2000). The disease has been reported in Europe and Asia including China, France, Germany, Indonesia, Japan, Malaysia and Thailand (Li et al. 2018; Mahardika et al. 2016; Sirivan, Tantaswasdi & Chuenchai 1997; Takehara et al. 1994; Woolcock et al. 2000).

Australian status

Waterfowl parvovirus has not been reported in Australia, there is no active surveillance, and waterfowl parvovirus is not nationally notifiable.

Agent properties

Parvoviruses can survive for long periods in the environment and show marked resistance to environmental and thermal stress, and pH extremes (Jordan et al. 2016; Ridpath & Mengeling 1988).

Parvoviruses, being non-enveloped and hydrophilic, have high resistance to disinfection. The GPV particles are resistant to various organic solvents and disinfectants (including chloroform, ether, fluorocarbon, 1:1000 formalin, 0.5% phenol, and sodium deoxycholate) and they cannot be inactivated by low pH (pH 3) or heat treatment typically used for disinfection applications (56°C for 30 minutes or 65°C for 30 minutes) (Schettler 1973; Takehara et al. 1994). The department is not aware of any inactivation studies specifically for GPV or MDPV.

4.5.3 Presence in duck meat

Transmission of GPV or MDPV by feeding infected duck meat or by feed contaminated with infected duck meat has not been reported. However, virus has been demonstrated in the organs and muscles of Muscovy ducks that were experimentally inoculated with attenuated GPV by mouth (Takehara et al. 1998). In this experiment, GPV DNA was detected by PCR at 2, 6, 10, 14 and 28 days post inoculation and viral titre in cardiac muscle reached 850 plaque forming units (PFU)/ml and 1316 PFU/ml at days 10 and 14 post inoculation respectively. GPV DNA was detected in the skeletal muscle up to 28 days post inoculation. The infective dose of GPV and MDPV has not been established.

4.5.4 Pathogenesis

Once the virus enters the gastrointestinal tract it replicates in the cells of the intestinal wall. From there the virus spreads through the body to other organs including the heart, liver and skeletal muscle (Zádori et al. 2006; Zsak 2016).

GPV genome has been detected by PCR in tissue samples from the respiratory tract, digestive tract, muscle (musculus longus colli and major adductor magnus), bone marrow and brain of Muscovy ducklings which were inoculated with the virus. GPV in these tissues was detected from day 4 to 10 post inoculation (Limn et al. 1996). A study to measure the distribution of GPV within tissues after oral infection found that virus was consistently detected in the blood, heart,

liver, spleen, kidney, bursa of Fabricius, thymus and Harder's glands by 8 hours post inoculation, and titres had significantly decreased at 6 and 9 days (Yang et al. 2009).

In a later study in 2016, 40 liver and spleen tissue samples from ducklings retrieved from farms affected with GPV outbreaks in China were all positive for the virus (Yu et al. 2016).

MDPV was isolated in allantoic fluid after the inoculation of a pool of liver, heart and skeletal muscle tissue samples from ducks found dead during an outbreak in California (January 1998) into the chorioallantoic sacs of embryonating Muscovy duck eggs (Woolcock et al. 2000).

A study showed DPV DNA present in hatching eggs, duck embryos and newly hatched ducklings. Of the newly hatched ducklings, 58.33% (21/36) were seropositive. Further, isolates were obtained from a 12 day old duck embryo and a newly hatched duckling. DPV infection did not reduce the fertilisation rate and hatchability (Chen et al. 2016).

4.5.5 Diagnosis

Clinical signs

Infection with GPV can result in 100% mortality in its acute form and geese remain susceptible to GPV throughout their lives (FAO 2002; Zádori et al. 2006). However, clinical sensitivity to GPV in geese is usually less than 30 days of age, compared to 40 to 50 days of age in Muscovy ducklings infected with MDPV (Zádori et al. 2006) . If birds are infected during the first week of life, high mortalities (up to 100%) often occur, but once the animals are 4 to 5 weeks old it is likely infection will be subclinical (FAO 2002; Jordan et al. 2016; Zádori et al. 2006). In 1 outbreak in China, 5% of total tissue samples and 39% of total faecal swabs collected from ducklings with no clinical signs were positive for GPV (Yu et al. 2016). Clinical findings in MDPV infected ducklings resemble those of GPV-infected goslings (Mahardika et al. 2016).

During an outbreak of MDPV in California in October 1997, weakness, lateral recumbency and inability to walk were observed in Muscovy ducklings 1 to 4 weeks old. There was also increased mortality (10 to 50%) and morbidity (30 to 80%) in these ducklings (Woolcock et al. 2000).

There have been recent reports that GPV has also caused duck beak atrophy and dwarfism syndrome in ducklings (Palya 2010; Yu et al. 2016).

Pathology

Post-mortem findings include pale thigh and leg muscles, pale myocardium, fibrinous exudate on the liver capsule, pulmonary oedema and congestion, and ascites. Older survivors can have pathological changes such as rounded hearts, and chronic passive congestion of the liver, and ascites (Mahardika et al. 2016; Poonia et al. 2006)

Testing

Clinical diagnosis is not sufficient to confirm disease and laboratory confirmation is necessary to diagnose GPV and MDPV (Xiao et al. 2017).

As none of the waterfowl parvoviruses are OIE-listed, there is no recommended diagnostic test. PCR and serology are the most commonly used laboratory tests (Palya 2010).

Immunoelectron microscopy, ELISA, immunofluorescence antibody testing and agar gel precipitin have been used for serology to detect GPV or MDPV antibodies (Irvine & Holmes 2010; Woolcock et al. 2000). In 1 study, sera from older ducklings (35 day old and 63 day old) were positive on ELISA, but no antibodies were detected in 10 day old ducklings. In the same study 3 of 10 sera taken from 21 day old ducklings had low titres of antibody which may indicate the start of a serological response (Woolcock et al. 2000). Antibodies may persist for years in older Muscovy ducks that have subclinical infection (Irvine & Holmes 2010).

There are no tests available that can differentiate between GPV and MDPV antibodies because the 2 viruses share many antigenic similarities (Woolcock et al. 2000).

Electron microscopy, PCR or RT-PCR can detect the virus (Yu et al. 2016).

4.5.6 Treatment

There is no effective treatment for waterfowl parvovirus infection.

4.5.7 Control

Control for GPV infection relies on effective flock biosecurity, hygiene and eliminating carrier birds (Irvine & Holmes 2010).

Many outbreaks have been attributed to birds hatched from eggs contaminated with the virus from GPV infected breeding flocks. Therefore, eggs should be imported only from countries that can guarantee breeder flock freedom from GPV. Geese that have survived an outbreak should not be used for breeding purposes as recovered birds may act as carriers (Irvine & Holmes 2010; Zsak 2016).

Live and inactivated vaccines are used in some countries to protect goslings during the first few weeks of life. The preferred vaccines are based on live attenuated viruses (Jordan et al. 2016). Vaccine used in breeder geese results in the transfer of immunity to newly hatched goslings for 2 to 3 weeks (FAO 2002).

There are conflicting reports of whether GPV vaccine is effective against both GPV and MDPV. Whilst some of the literature suggests that GPV strain vaccines are ineffective against MDPV (Zádori et al. 2006); others report that 'vaccinating Muscovy ducklings with strains of GPV provided them with a high level of protection against a challenge with MDPV' (Woolcock et al. 2000).

Studies on a recently developed vaccine based on a live attenuated Muscovy duck parvovirus, produced by Merial, showed that vaccination with a live attenuated MDPV strain provided immunity to Muscovy ducks against GPV as well as MDPV (Maurin-Bernaud et al. 2014). This vaccine is now registered for use within the European Union (EMA 2015).

4.5.8 Current biosecurity measures

Australia currently requires that for foie gras for human consumption from France, during the 12 to 21 day stuffing period prior to slaughter, certification that there have been no outbreaks of Derzsy's disease on the properties of origin of the ducks and/or geese from which the livers were derived.

With regards to importing fertile duck eggs, Australia requires:

- Either the country of origin is free from Derszy's disease or serological testing of the parent flock within 21 days before the first day of collection of eggs for export to Australia has been performed, with negative results;
- The source flock is free from clinical signs of GPV and MDPV (Muscovies only) for the 90 day period prior to collection of the eggs for export to Australia and has not come into contact with any birds showing evidence of these diseases;
- Any clinical evidence of disease in the source flock in the 21 days since the last day of collection of the eggs exported to Australia has been investigated and the results indicate disease caused by GPV or MDPV has not occurred; and
- Serology of imported birds in quarantine for goose parvovirus has been performed 6 weeks post hatching, with negative results.

4.5.9 Conclusion

- Waterfowl parvoviruses have been reported to cause disease in geese and ducks in Thailand.
- Waterfowl parvoviruses have not been reported in Australia.
- Transmission of GPV or MDPV by feeding infected duck meat or by feed contaminated with infected duck meat has not been reported.
- Virus has been demonstrated in the organs and muscles of Muscovy ducks that were experimentally inoculated with attenuated GPV by mouth.
- The virus is markedly resistant to environmental and thermal inactivation and may survive for long periods of time in infected duck carcasses. It is likely that the virus will resist cooking at the time and temperature defined for this review.
- GPV and MDPV are not OIE-listed disease agents and there are no recommendations in the OIE Code on disease testing and measures for safe trade.
- Waterfowl parvovirus is not nationally notifiable.
- Vaccines are available to manage the disease.

Based on the above information cooking as defined in the scope at section 1.3 of this document (70°C for 8.2 minutes) is likely to reduce the viral load but not fully inactivate the waterfowl parvoviruses that may potentially be present in meat of ducks. Therefore, the department concluded that further risk assessment of the waterfowl parvoviruses was required.

4.5.10 Risk Assessment

Entry assessment

- Both MDPV and GPV have been reported in Thailand. Whilst there are no reports of MDPV affecting species other than Muscovy ducks and their hybrid breeds, GPV has been reported in Pekin ducks.
- Although GPV causes severe disease in young ducklings, older ducklings greater than 4 to 5 weeks of age may not show clinical signs but may be shedding the virus. Older birds may also be subclinically infected. Furthermore, recovered birds may act as carriers and be a source of infection. Clinical signs will usually be observed in young ducklings and as the disease is likely to cause severe morbidity and mortality it would almost certainly be identified on affected farms and limit their ability to supply meat.
- Infection of slaughter age ducks (greater than 7 weeks) are likely to be subclinical and may not be identified on ante-mortem inspection. Gross lesions may not be readily visible on post-mortem inspection of these ducks.
- The virus spreads through many organs including skeletal muscle. The department is not aware of any inactivation studies specifically for GPV or MDPV and it has been reported that parvovirus is resistant to thermal inactivation (56°C for 30 minutes or 65°C for 30 minutes) and may not be inactivated by low pH. Therefore, it is likely that while the viral load would be reduced, some virus would resist cooking at the time and temperature defined for this review, 70°C for 8.2 minutes.

Conclusion: Considering the above factors, the likelihood of entry in imported cooked duck meat from Thailand was assessed to be **moderate.**

Exposure assessment

- The virus is markedly resistant and could remain viable for a long period of time in the environment.

- Vertical transmission through infected eggs is the most commonly reported source of outbreaks in disease-free countries. There have been no reports of oral transmission through infected meat; however, transmission by mouth cannot be discounted given that oral transmission of GPV has been demonstrated experimentally. The infective dose of GPV and MDPV has not been described.
- The impact of the viruses in Australian wild waterfowl is not known. Evidence suggests that some Northern Hemisphere species of wild geese were implicated in the epidemiology of the disease.
- As only cooked products consisting of muscle meat and overlying fat and skin would be imported, most imported product would move from the retailer/distributor to household consumers or to the food service industry. Product not consumed would be disposed of, which may result in exposure to wild birds and non-avian species. More likely in products sold directly to households, backyard (low biosecurity) poultry could also be exposed to waste. Limiting the scope to muscle meat and overlying fat and skin that has been cooked reduces the type and volume of waste that may be created. Bone, trimmings and offal would not be imported, reducing the risk of exposure to waste.
- A possible exposure pathway for some waste to Australian commercial ducks and geese is via feed for ducks containing meat meal made from waste from imported cooked duck meat. It is unknown whether rendering, especially the continuous low temperature type (70 to 95°C) will totally inactivate any parvovirus if present. However the Australian Standard for Hygienic Rendering of Animal Products (AS 5008:2001) requires processing plants to verify that their process inactivates spore forming bacteria (Primary Industries Standing Committee & Primary Industries Ministerial Council 2007). In poultry diets, meat and bone meal is typically limited to less than 5% of the diet because of the high calcium, phosphorus, and lysine content of the meal. The possibility of exposure via this pathway is further reduced by the restriction on the products that can be imported as outlined in the scope.

Conclusion: Based on this information, the likelihood of exposure of domestically kept geese and ducks to waterfowl parvovirus infected duck meat was assessed to be **low**.

Estimation of likelihood of entry and exposure

The estimate of the likelihood of entry was moderate and the likelihood of exposure was low. Using the matrix described in Figure 2, the likelihood of entry and exposure of a waterfowl parvovirus was estimated to be **low**.

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

The most likely outbreak scenario following exposure to a waterfowl parvovirus was considered to be establishment in susceptible wild and low biosecurity geese and duck populations with vertical and horizontal spread to more than 1 state or territory.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible duck and geese to a waterfowl parvovirus:

- Vertical transmission through infected eggs is the most commonly reported source of an outbreak. GPV may cause subclinical infection in older geese and pass on the virus to their progeny. Recovered birds may be carriers.
- Waterfowl parvoviruses are excreted in large amounts in the faeces of infected waterfowl and can subsequently spread rapidly to susceptible birds by both direct and indirect routes.
- Due to its resistance in the environment, parvovirus can persist in buildings and on poorly cleaned and disinfected surfaces which results in transmission between subsequent flocks.

- The role of wild geese and ducks in the spread of the disease is undefined. Evidence suggests that 2 species of wild geese in the northern hemisphere may be implicated in the epidemiology of the disease. Although over 50 taxa of waterbirds have been identified in Australia (Kingsford & Porter 2009), the susceptibility of Australian wild waterfowl species to waterfowl parvoviruses is not known. However, the Canada goose (*Branta canadensis*), suspected to be a source of GPV in the UK and Sweden and originally introduced into Australia as an exotic waterfowl species, have been occasionally sighted in the wild (DPIRD 2017).
- Waterfowl parvoviruses are highly contagious and fatal in goslings, Muscovy ducklings, and Pekin ducklings, often causing between 70 and 100% mortality in goslings less than 4 weeks old.

Conclusion: Considering the above factors, the likelihood of establishment and spread of a waterfowl parvovirus through the Australian duck or goose population was assessed to be **moderate.**

Determination of the effects resulting from the outbreak scenario

Direct effects

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

- GPV is highly contagious and fatal in goslings and Muscovy ducklings, often causing between 70 and 100% mortality in goslings less than 4 weeks old. MDPV causes similar clinical signs but causes disease only in Muscovy ducks and their hybrids.
- If the birds are 4 to 5 weeks old or older it is likely the infection will be subclinical.

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

- There are no reports of DPV in wild ducks. Wild geese may be infected with GPV, however, there is no evidence of adverse effects on the environment.

Indirect effects

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

- GPV and MDPV are not notifiable in any Australian jurisdiction and there are no control, monitoring or surveillance programs in place.
- An outbreak would likely provoke similar control and eradication measures to those that have been introduced in other affected countries. These include active surveillance, disinfection of infected flocks and elimination of carrier birds. A vaccination program may be introduced.

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

- The Australian duck farming industry is dominated by 2 vertically integrated producers, 1 in New South Wales and the other in Victoria. Both breed and grow ducks under intensive and biosecure conditions. There are also several smaller duck farms, especially in Victoria and Western Australia, as well as backyard ducks scattered around the country. There are no significant producers of duck eggs for commercial purposes (PoultryHub 2018). Because of the structure of the major producers, biosecurity can largely be controlled to prevent or respond to potential impacts. However, smaller duck farms and backyard ducks are more likely to be impacted by an outbreak due to low biosecurity controls and resources.
- Pekin and Muscovy ducks are among the major breeds used for meat production, and the Pekin duck is the predominant breed used for meat production in Australia (Stein 2012).

- Commercial customers would be unlikely to be affected by shortages of duck eggs. Commercial customers could be affected by shortages or price increases of duck meat in some outbreak scenarios.
- 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 are unlikely to be any impacts on international trade. Only 5% of processed duck meat produced in Australia is exported (PoultryHub 2018).

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

- As there has not been any discernible effects on wild duck or goose species in the northern hemisphere, it is expected that 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

- In young ducks and goslings, GPV and MDPV may cause significant mortalities and morbidities, and it is likely that breeding flocks and potentially other flocks in an operation would be destroyed following an outbreak due to their carrier state. Loss of stock may affect the livelihood and income of those directly affected. There are only 2 major duck breeders in Australia employing a total of around 300 people. Therefore, affected areas may experience very minor issues while movement restrictions are in place.

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.** 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.

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 (**moderate**) using Figure 3 to obtain an estimation of likely consequences of **low**.

4.5.11 Risk estimation

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 waterfowl parvoviruses being introduced by cooked duck meat imported into Australia from Thailand.

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

Therefore, under the defined scope and method of this review, the unrestricted risk estimate achieves Australia's ALOP and the department concluded that additional risk management was not necessary for waterfowl parvoviruses. The department considers the controls imposed by the scope and method of this review to be acceptable risk management measures for waterfowl parvoviruses.

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5 Requirements for the importation of cooked duck meat from Thailand

5.1 Eligibility

Importation under these conditions is restricted to cooked duck meat from Thailand only. These conditions do not apply to dried or powdered products (jerky and duck-meat-based flavours for example).

5.2 Documentation

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

Each consignment must be accompanied by:

1) a valid import permit
2) 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 OIE Code.

The veterinary certificate must provide details of:

- the packaging of the cooked duck meat product including details of the labelling
- the addresses and Veterinary Authority of Thailand approval numbers of establishments at which the ducks from which the meat was derived were slaughtered
- the facility at which the cooked duck meat product was prepared
- the establishment at which the cooked duck meat product was stored before export
- the names and addresses of the exporter and the consignee.

An Official Government Veterinarian means a veterinarian authorised by the Veterinary Authority of Thailand 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 OIE 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 duck meat without compensation.

5.3 Certification

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

1) A statement that all of the establishments where the ducks from which the meat was derived were slaughtered, processed and stored have current approval from the Veterinary Authority of Thailand and are operating in accordance with the laws of Thailand.
2) A statement that the ducks were slaughtered and processed in accordance with the regulatory requirements of Thailand for items intended for human consumption.
3) A statement that no clinical signs, epidemiological or other evidence of infection with contagious diseases were present in the source flock(s) prior to slaughter.
4) A statement that the ducks 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.

5) A statement that the ducks from which the meat was derived were hatched in and have been continuously resident in Thailand until slaughter and the date/s of slaughter provided.
6) A statement that the duck meat is sourced from domestic ducks (*Anas platyrhynchos domesticus* and *Cairina moschata*) only.
7) A statement that the duck meat is limited to muscle meat and overlying skin and fat.
8) A statement that the duck meat has been cooked in a commercial process to a minimum core temperature of 70°C for at least 8.2 minutes or equivalent according to the time/temperatures specified in Annex 1 and records confirm that these time/temperature parameters were met.
9) A statement that the temperature recording equipment was confirmed to be in good working order prior to each day's production and was independently calibrated at least once every 12 months.
10) A statement that all establishments that handled, prepared, processed or stored the cooked duck meat destined for Australia have approved and documented internal processes to keep such meat isolated from any meat and meat products that are not eligible for export to Australia and to ensure the integrity and health status of the product is maintained.
11) A statement that the cooked duck meat was packed for transport to Australia in a clean container and sealed with an official government seal.

5.4 Annex 1

Heat treatment considered equivalent to a core temperature of least 70°C for not less than 8.2 minutes includes the following time/temperature combinations. The heat treatment must be applied as a minimum core temperature and minimum time at that core temperature in a commercial heating process. It does not apply to dried or powdered products:

1) 71°C for not less than 6.6 minutes
2) 72°C for not less than 5.2 minutes
3) 73°C for not less than 4.2 minutes
4) 74°C for not less than 3.3 minutes
5) 75°C for not less than 2.6 minutes
6) 76°C for not less than 2.1 minutes
7) 77°C for not less than 1.7 minutes
8) 78°C for not less than 1.3 minutes
9) 79°C for not less than 1.1 minutes
10) 80°C for not less than 1 minute.

6 Review of processes

6.1 Review of conditions

The department 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 Thailand has changed or if there is evidence of new or emerging diseases. The department may also review the import conditions if there is any change to the nature or understanding of a disease, agent or exposure pathways.

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. 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.

Glossary

Term or abbreviation	Definition
AGID	Agar gel immunodiffusion
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
ALOP	Appropriate level of protection
AS	Attenuated strain
bean geese	*Anser fabalis*
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
Cherry Valley duck	A breed of domestic duck
chicken	*Gallus gallus domesticus*
Chicken meat IRA	*Generic import risk analysis report for chicken meat 2008*
OIE Code	OIE Terrestrial Animal Health Code 2019
CRISPR	Clustered regularly interspaced short palindromic repeats
DEDS	Duck egg drop syndrome
the department	The Department of Agriculture, Water and the Environment
DHAV-1	Duck hepatitis A virus type 1
DNA	Deoxyribonucleic acid
domestic duck	*Anas platyrhynchos domesticus*
DPV	Duck parvovirus
DTMUV	Duck Tembusu virus
DVE	Duck virus enteritis
DVEV	Duck virus enteritis virus
ELISA	Enzyme-linked immunosorbent assay
endemic	Belonging to, native to, or prevalent in a particular geography, area or environment
FSANZ	Food Standards Australia New Zealand
FMDV	Foot and mouth disease virus
GPV	Goose parvovirus
host	An organism that harbours a parasite, mutual partner, or commensal partner, typically providing nourishment and shelter
ICTV	International Committee on Taxonomy of Viruses
import permit	Official document authorising importation of a commodity in accordance with specified sanitary import requirements
mallard	*Anas platyrhynchos*
MDPV	Muscovy duck parvovirus
mule duck	*Cairina moschata* and *Anas platyrhynchos domesticus* cross breed
Muscovy duck	*Cairina moschata*
OIE	World Organisation for Animal Health

Term or abbreviation	Definition
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
Pekin duck	A breed of domestic duck
pest	Any species, strain or biotype of plant, animal, or pathogenic agent injurious to plants or animals
PFU	Plaque forming unit
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
Tsiay duck	A breed of domestic duck
TMUV	Tembusu virus
unrestricted risk	Risk estimate in the absence of sanitary measure(s)
VS	Virulent strain
white-fronted geese	*Anser albifrons*