Import risk analysis: Chicken and duck meat for human consumption

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Ministry for Primary Industries
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6011
New Zealand
Tel: 64 4 894 0100
Fax: 64 4 894 0731

Science and Risk Assessment
Ministry for Primary Industries

Ministry for Primary Industries
Manatū Ahu Matua

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Approved for general release

Stephen Cobb
Acting Manager, Risk Analysis (Animals & Aquatic)
Ministry for Primary Industries

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Contributors to this risk analysis

1. Author

Stephen Cobb	Principal Adviser, Risk Analysis (Animals & Aquatic)	MPI, Wellington
Helen Smith	Senior Adviser, Risk Analysis (Animals & Aquatic)	MPI, Wellington

2. Internal Peer Review

Lincoln Broad	Senior Adviser, Risk Analysis (Animals & Aquatic)	MPI, Wellington
Lucy Johnston	Senior Adviser, Animal Imports	MPI, Wellington
Matthew Stone	Director, Animal and Animal Products	MPI, Wellington
Marnie Thomas	Manager, Animal Imports	MPI, Wellington
Stuart MacDiarmid	Principal Adviser, Risk Analysis, International Organisations	MPI, Wellington
Lisa Oakley	Manager, Risk Analysis (Animals & Aquatic)	MPI, Wellington

3. External Scientific Review

David E Swayne	Laboratory Director, Southeast Poultry Research Laboratory	OIE Collaborating Centre for Research on Emerging Avian Diseases
Nigel Horrox	International Poultry Health & Food Safety Consultant	Thorpe House, Driffield, East Yorkshire, United Kingdom
Kerry Mulqueen	Veterinary Consultant	Nominated by the Poultry Industry Association of New Zealand

1. Executive Summary

The biosecurity risks associated with the importation of chilled or frozen meat and meat products derived from chickens (*Gallus gallus*) or ducks (Pekin ducks, *Anas platyrhynchos domestica* or *Anas peking*, Muscovy ducks *Cairina moschata,* or a hybrid of these known as mulard or moulard ducks) have been examined.

From an initial list of 116 organisms/groups of organisms possibly associated with chickens and ducks, a preliminary hazard list identified 47 organisms/groups of organisms that required further consideration.

Of these preliminary hazards, 14 were considered to be potential hazards in imported whole chicken carcases and 6 of these were considered to be potential hazards in imports limited to chicken meat.

16 of the preliminary hazards were considered to be potential hazards in imported whole duck carcases and 10 of these were considered to be potential hazards in imports limited to duck meat.

Following a risk assessment for each of these potential hazards, options to manage the risk associated with the following hazards in chicken meat have been presented:

- Newcastle disease virus
- Highly pathogenic avian influenza virus
- Infectious bursal disease virus
- *Salmonella arizonae*

For imported whole chicken carcases, risk management measures have also been presented for avian paramyxovirus-2 and exotic strains of infectious bronchitis virus.

Options to manage the risk associated with the following hazards in duck meat have been presented:

- Newcastle disease virus
- Highly pathogenic avian influenza virus
- Duck hepatitis virus
- Derzsy's disease virus (Muscovy ducks and their hybrids only)
- *Salmonella arizonae*

For imported whole duck carcases, risk management measures have also been presented for avian paramyxovirus-2 and duck virus enteritis virus.

2. Introduction

This risk analysis examines the biosecurity risks associated with the import of chicken and duck meat and meat products from all countries.

3. Scope

This qualitative risk analysis will be used to formulate requirements to be met for effective management of biosecurity risks in an import health standard issued under Part 3 section 22 of the Biosecurity Act. The purpose of Part 3 of the Biosecurity Act 1993 is to provide for the effective management of risks associated with the importation of risk goods.

The scope of this risk analysis is the assessment of the likelihood and consequences of organisms that may be associated with the importation of chicken and duck meat and meat products being introduced into New Zealand as a result of these imports, and the various options to manage these risks. The risk analysis is undertaken in accordance with the principles and obligations under the World Trade Organisation Agreement on the Application of Sanitary and Phytosanitary Measures. Requirements in import health standards, formulated with reference to this risk analysis, manage the risk of introducing organisms not established in New Zealand, or under regulatory control here.

In New Zealand, the Food Act 1981 and the Animal Products Act 1999 manage risks to public health associated with food. All imported foods must meet food safety and suitability requirements under the Food Act. Risks in imported food from organisms of concern to public health that are known to already be present in New Zealand are managed under the Food Act, unless the commodity concerned represents a pathway to animal populations in New Zealand that is different to the risks associated with importation through carriage inside humans (in which case requirements may be considered under the Biosecurity Act). Such organisms are specifically excluded from the scope of this risk analysis.

Consignments of product imported into New Zealand for human consumption in New Zealand must comply with the Food Act 1981. These requirements are independent of the import health standard requirements.

4. Commodity definition

The commodity considered in this import risk analysis is defined as chilled or frozen meat[1] and meat products[2] derived from chickens (*Gallus gallus*) or ducks (Pekin ducks, *Anas platyrhynchos domestica* or *Anas peking*, Muscovy ducks *Cairina moschata,* or a hybrid of these known as mulard or moulard ducks) that have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate effective Good Manufacturing Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes.

The commodities covered in this risk analysis include:

[1] Skeletal muscle with naturally included or inherent tissue or bone. This definition excludes animal by-products, offal, and giblets.

[2] Products prepared from or with meat that has undergone treatment such that the cut surface shows that the product no longer has the characterisitics of fresh meat (e.g. cooked or cured).

1. whole chicken or duck carcases that have been subject to routine evisceration procedures. These may be uncooked, unskinned, and may include the head and feet;
2. bone-in chicken or duck products such as wings or legs;
3. boneless chicken or duck meat products such as breasts, boned-out thighs;
4. reconstituted[3] chicken or duck meat products comprised of meat and skin.

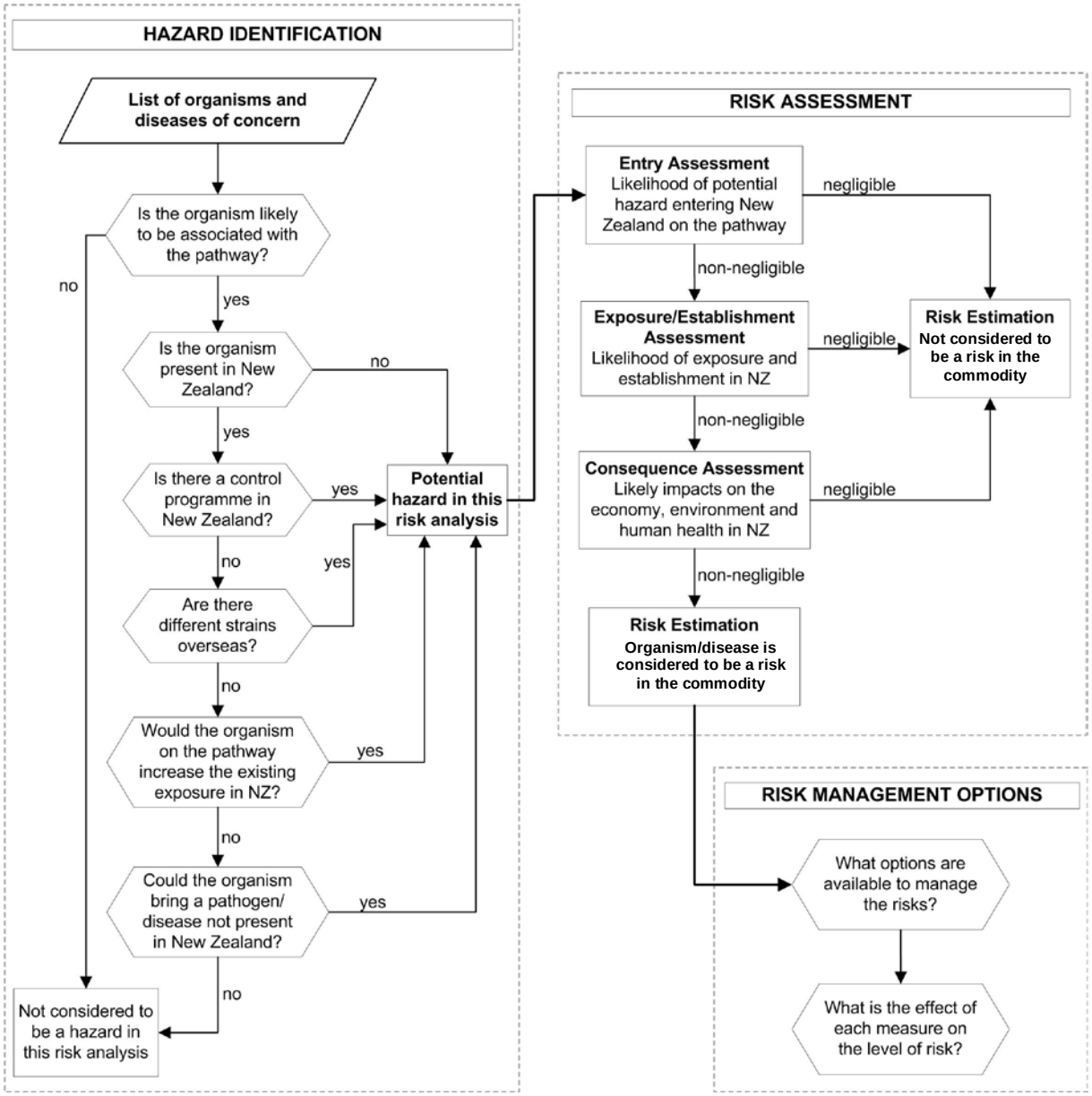
5. Risk analysis methodology

The methodology used in this risk analysis follows the guidelines as described in *Biosecurity New Zealand Risk Analysis Procedures – Version 1*[4] and in Section 2 of the *Terrestrial Animal Health Code* of the World Organisation for Animal Health (OIE 2011).

The risk analysis process used by the Ministry for Primary Industries (MPI) is summarised in Figure 1.

[3] Reconstituted meat is a liquefied meat product used as a meat supplement in foods such as chicken nuggets and food for domestic animals. For the purposes of this risk analysis reconsitituted meat products are comprised only of chicken or duck meat and skin.
[4] See www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf.

Figure 1. The risk analysis process



5.1. PRELIMINARY HAZARD LIST

The first step in the risk analysis is hazard identification. This process begins with the collation of a list of organisms that might be associated with chicken or duck meat (the preliminary hazard list). The diseases/agents of interest are those that could be transmitted in chicken or duck meat or meat products and could infect domestic, feral, or wild animals that occur in New Zealand, or humans. In this case the preliminary hazard list was compiled from *Diseases of Poultry*, 12th Edition, 2008, Ed Y.M. Saif, Blackwell Publishing. The diseases/agents identified in the preliminary hazard list are shown below in Table 1.

Organisms in the preliminary hazard list requiring further consideration are subjected to further analysis to determine whether they should be identified as potential hazards and all organisms identified as potential hazards are subjected to risk assessment.

Table 1. List of organisms, diseases of concern

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Viral diseases						
Newcastle disease	Avian paramyxovirus serogroup 1 (APMV-1)	Yes	Exotic strains	Yes	Yes	Yes
Avian paramyxoviruses 2-9	APMV-2 to 9	No	Exotic serogroups/ strains	Yes	Yes	Yes
Turkey rhinotracheitis (TRT), swollen head syndrome (SHS), and avian rhinotracheitis (ART)	Avian metapneumovirus	Yes	Exotic	Yes	Yes	Yes
Infectious bronchitis	Infectious bronchitis virus (IBV)	Yes	Exotic strains	Yes	No	Yes
IBV is primarily an infection of chickens (Cavanagh 2005). There is a single report of a group III coronavirus being recovered from a hand-caught mallard although this was not associated with disease in this individual and was distinct form IBV (Jonassen *et al.* 2005).						
Laryngotracheitis	Laryngotracheitis virus (LTV)	Yes	Present	N/A	N/A	Yes
Strains of LTV associated with clinical disease are considered to be present in New Zealand (Howell 1992) although more virulent strains of this virus have been reported overseas (Devlin *et al.* 2011).						
Influenza	Influenzavirus A	Yes	Exotic serogroups/ strains	Yes	Yes	Yes
Infectious bursal disease	Infectious bursal disease virus (IBDV)	Yes	Exotic	Yes	Yes?	Yes
There is limited evidence of naturally occurring IBDV infection in ducks, with a single report recording the recovery of virus from a Pekin duck (McFerran *et al.* 1980). Yamada *et al.* (1982) did record a serological reaction to IBDV in experimentally infected ducks although this was not associated with either disease or gross or histopathological lesions and virus could not be recovered from experimentally infected birds. Similarly Okoye *et al.* (1990) were unable to experimentally infect ducks with IBDV. More recently, Oladele *et al.* (2009) described limited IBDV-specific immunohistochemical staining in experimentally challenged ducks.						
Chicken infectious anaemia	Chicken infectious anaemia virus	No	Present	N/A	N/A	No
Chicken infectious anaemia virus is regarded as present in New Zealand (Anonymous 2005).						

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Viral diseases (continued).						
Duck circovirus infection	Duck circovirus (DuCV)	No	Unknown	No	Yes	Yes
DuCV has only been recovered from ducks (Hattermann *et al.* 2003; Soike *et al.* 2004). There are no reports associating this agent with chickens.						
Group I adenovirus infections	Fowl adenovirus (FAdV)	No	Some species of *Aviadenovirus* genus present	Yes	Yes	Yes
Quail bronchitis (QBV) and Chicken embryo lethal orphan (CELO) viruses are indistinguishable and are considered the type strain of group I, serotype 1 avian adenovirus (FAdV-1) (Reed & Jack, 2008; Smyth & McNulty, 2008; DuBose & Grumbles, 1959).						
FAdV-1, 8, and 12 are recognised as present in New Zealand (Saifuddin *et al.* 1992, Saifuddin 1990).						
Egg drop syndrome (EDS)	EDS virus	No	Present	N/A	N/A	No
Disease outbreaks mostly occur in laying hens although ducks and geese are also thought to be natural hosts of the virus (Adair and Smyth 2008). EDS is recognised in New Zealand (Howell 1992).						
Avian adenovirus splenomegaly (AAS)	AAS virus (AASV)	No	Exotic	Yes	No	Yes
AASV is closely related to haemorrhagic enteritis virus (HEV) of turkeys. Until recently, turkeys, pheasants, and chickens were the only known natural hosts for HEV and related viruses although it is now thought that guinea fowl and psittacines may also be naturally infected (Pierson and Fitzgerald 2008).						
Pox	Fowl pox virus (FPV)	No	Present	N/A	N/A	No
Chicken, turkey, pigeon and canary pox virus infections are considered common in New Zealand (Anonymous 1995).						
Viral arthritis	Avian reoviruses	No	Present	N/A	N/A	No
Reovirus (viral arthritis) is recognised in New Zealand (Howell 1992).						
Other reovirus infections	Avian reoviruses	No	Exotic	Yes	Yes	Yes
Turkey coronavirus enteritis	Turkey coronavirus (TCV)	No	Exotic	Yes	No	Yes
Turkeys are believed to be the only natural host for TCV although SPF chickens have been successfully infected under experimental conditions (Guy 2008a).						
Rotavirus infection	Rotavirus	No	Present	N/A	N/A	No
Rotaviruses have been described in New Zealand poultry (Saifuddin *et al.* 1989).						
Astrovirus infection	Astrovirus	No	Exotic	Yes	Yes	Yes
Avian enterovirus-like infection	Enterovirus-like viruses (ELVs)	No	Exotic	Yes	No	Yes
Duck hepatitis viruses are considered separately below. Infections with ELVs have been described in turkeys, chickens, guinea fowl, partridges, pheasants, ostriches, and psittacine species (Guy *et al.* 2008).						
Duck hepatitis	Duck hepatitis virus (DHV) types 1, 2, and 3	Yes	Exotic	No	Yes	Yes
Although experimental infection of chickens with DHV-1 has been described, natural infection of chickens with DHV-1,2, or 3 has not been reported (Woolcock 2008).						
Duck virus enteritis (DVE)	Duck enteritis virus	No	Exotic	No	Yes	Yes
Natural susceptibility to DVE is limited to members of the family *Anatidae* (ducks, geese, and swans) (Sandhu and Metwally 2008).						

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Viral diseases (continued).						
Haemorrhagic nephritis enteritis of geese	Goose haemorrhagic polyomavirus (GHPV)	No	Exotic	No	No	No
Natural infection with GHPV has only been described in geese (Guerin 2008).						
Derzsy's disease	Goose parvovirus	No	Exotic	No	Yes	Yes
Geese, Muscovy ducks, and some hybrid breeds are the only species in which natural clinical disease has been observed. Other breeds of domestic poultry are refractory to experimental infection (Gough 2008).						
Avian nephritis	Avian nephritis virus (ANV)	No	Present	N/A	N/A	No
Avian nephritis viruses types 1-3 are recognised as present in New Zealand (Howell 1992).						
Arbovirus infections	Eastern equine encephalitis (EEE) virus	EEE, WEE, and WN listed	Exotic	Yes	Yes	Yes
	Western equine encephalitis (WEE) virus					
	Highlands J (HJ) virus					
	Israel turkey meningoencephalitis (IT) virus					
	West Nile (WN) virus					
Turkey viral hepatitis	Aetiology thought to be a picornavirus	No	Exotic	No	No	No
Turkey viral hepatitis has been recognised only in turkeys. Chickens and ducks have been shown to be refractory to infection (Guy 2008b).						
Avian encephalomyelitis	Avian encephalomyelitis virus	No	Present	N/A	N/A	No
Avian encephalomyelitis has been described in New Zealand (Howell 1992; Christensen 2010).						
Hepatitis-splenomegaly syndrome	Avian hepatitis E virus (HEV)	No	Exotic	Yes	No	Yes
Natural HEV infections are limited to chickens (Meng *et al.* 2008).						
Marek's disease	Marek's disease virus (MDV)	No	Exotic strains?	Yes	No?	Yes
Ducks are probably refractory to infection although MDV-inoculated ducks developed antibodies (Schat and Nair 2008).						
Leukosis/sarcoma group	Members of the alpharetrovirus family	No	Present	Yes	No	No
Avian leukosis subgroup J was diagnosed using ALV-J ELISA in 1998/99 in commercial meat-type chicken flocks in New Zealand (Stanislawek 2001). Chickens are the natural hosts for this group of viruses which have not been isolated from other avian species except pheasants, partridges, and quail (Fadly and Nair 2008).						

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Viral diseases (continued).						
Reticuloendotheliosis	Reticuloendotheliosis virus	No	Present	N/A	N/A	No
Although clinical disease is unusual in New Zealand, serological investigations indicate that infection with reticuloendotheliosis virus is widespread (Howell *et al.* 1982).						
Dermal squamous cell carcinoma	Unknown	No	Present	N/A	N/A	No
Disease is recognised to be present in New Zealand (Mulqueen 2012)						
Multicentric histiocytosis	Unknown	No	Unknown	Yes	No	Yes
Disease is described as a condition of young broiler chickens (Hafner and Goodwin 2008).						
Bacterial diseases						
Pullorum disease and fowl typhoid	*Salmonella* Gallinarum-Pullorum	Yes	Exotic	Yes	Yes	Yes
Paratyphoid infections	*Salmonella* spp.	No	Some members exotic	Yes	Yes	Yes
Arizonosis	*Salmonella* arizonae serovar $18Z_4Z_{32}$	No	Exotic	Yes	Yes	Yes
Campylobacteriosis	*Campylobacter jejuni* and others	No	Exotic strains?	Yes	Yes	Yes
Colibacillosis	Avian pathogenic *Escherichia coli* (APEC)	No	Exotic strains	Yes	Yes	Yes
Fowl cholera	*Pasteurella multocida*	Yes	Present	N/A	N/A	No
Fowl cholera due to *P. multocida* was removed from New Zealand's list of notifiable organisms on 21 September 2001 (Poland 2001). Suspected exotic disease investigations have recorded diagnoses of fowl cholera due to *P. multocida* in ducks and commercial poultry (Anonymous 2000; Bingham 2006). Diagnostic laboratories have recovered *P. multocida* from cases of fowl cholera in chickens, turkeys, and quail (Orr 2000; Varney 2004c; Varney 2007). Although the virulence of an individual isolate appears to depend upon a number of factors, based on the clinical presentation of this disease described in New Zealand, there is no evidence to support claims that overseas strains of avian *P. multocida* are likely to be more virulent than those seen here (MAF Biosecurity New Zealand 2010). Furthermore, Christensen and Bisgaard (2000) have stated that no country can be considered free of fowl cholera, because *P. multocida* has a broad habitat, including mucosal surfaces of a wide range of domestic and wild birds and mammals and that processed poultry products are not considered to present a major risk of transmission of infection, due to the delicate nature of *P. multocida*.						
Riemerella anatipestifer infection	*Riemerella anatipestifer*	No	Exotic strains	Yes	Yes	Yes
Ornithobacterium rhinotracheale infection	*Ornithobacterium rhinotracheale*	No	Exotic	Yes	Yes	Yes
Bordetellosis (turkey coryza)	*Bordetella avium*	No	Exotic	Yes	Yes	Yes
Infectious coryza	*Avibacterium paragallinarum*	No	Present	N/A	N/A	No
Avibacterium paragallinarum infection was diagnosed in New Zealand poultry in 2011 and is believed to have been present in this country for many years (Bingham 2011).						

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Bacterial diseases (continued).						
Mycoplasma gallisepticum infection	*Mycoplasma gallisepticum*	Yes	Exotic strains?	Yes	Yes	Yes
Mycoplasma gallisepticum is considered present in New Zealand (Black 1997). However, exotic strains of this organism may be more virulent than those present in this country (MAF Biosecurity New Zealand 2010).						
Mycoplasma meleagridis infection	*Mycoplasma meleagridis*	No	Exotic	No	No	No
M. meleagridis is a specific pathogen of turkeys. Chickens have been shown to be refractory to infection (Chin *et al.* 2008).						
Mycoplasma synoviae infection	*Mycoplasma synoviae*	Yes	Present	N/A	N/A	No
Mycoplasma synoviae is considered present in New Zealand (Black 1997).						
Mycoplasma iowae infection	*Mycoplasma iowae*	No	Exotic	Yes	No	Yes
The natural host of *M. iowae* is the turkey, although isolation from chickens is not uncommon (Bradbury and Kleven 2008).						
Other mycoplasma infections	*Mycoplasma* spp.	No	Exotic?	Yes	Yes	Yes
Ulcerative enteritis (quail disease)	*Clostridium colinum*	No	Present	N/A	N/A	No
Ulcerative enteritis recognised in New Zealand (Black 1997).						
Necrotic enteritis	*Clostridium perfringens* type A and type C	No	Present	N/A	N/A	No
Necrotic enteritis recognised in New Zealand (Black 1997).						
Botulism	*Clostridium botulinum*	No	Present	N/A	N/A	No
Surveillance of wild birds has confirmed the presence of botulism in New Zealand (Alley 2002a).						
Gangrenous dermatitis	*Clostridium perfringens* type A, *Clostridium septicum*, *Staphylococcus aureus*	No	Present	N/A	N/A	No
Gangrenous dermatitis recognised in New Zealand (Black 1997).						
Staphylococcosis	*Staphylococcus aureus* and *Staphylococcus* spp.	No	Present	N/A	N/A	No
Staphylococcosis recognised in New Zealand (Black 1997).						
Streptococcosis	*Streptococcus* spp.	No	Present	N/A	N/A	No
Streptococcosis recognised in New Zealand (Black 1997).						
Enterococcosis	*Enterococcus* spp.	No	Present	N/A	N/A	No
Enterococcosis recognised in New Zealand (Black 1997).						

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Bacterial diseases (continued).						
Erysipelas	*Erysipelothrix rhusiopathiae*	No	Present	N/A	N/A	No
Erysipelas recognised in New Zealand (Black 1997; Alley 2002a)						
Avian intestinal spirochaetosis	*Brachyspira* spp.	No	Exotic strains	Yes	Yes	Yes
Currently nine species of *Brachyspira* are described, with the four main pathogenic species in birds being *B. intermedia*, *B. pilosicoli*, *B. alvinipulli*, and *B. hyodysenteriae* (Hampson and Swayne 2008). *B. pilosicoli* and *B. hyodysenteriae* have been isolated in New Zealand. Neither *B. intermedia* nor *B. alvinipulli* have been identified (Midwinter and Fairley 1999).						
Tuberculosis	*Mycobacterium avium*	No	Present	N/A	N/A	No
There is no evidence that the strains of *M. avium* associated with avian tuberculosis in New Zealand are less virulent than strains found in commercial poultry overseas (MAF Biosecurity New Zealand 2010).						
Other bacterial diseases	*Acinetobacter* spp.	No	Present (Varney 2005)	N/A	N/A	No
	Actinobacillus / *Gallibacterium* spp.	No	Present (Wilson 2002)	N/A	N/A	No
	Arcanobacterium pyogenes	No	Present (Varney 2004a)	N/A	N/A	No
	Aegyptianella spp.	No	Exotic	Yes	Yes	Yes
	Aerobacter aerogenes	No	Present (Spiller 1964)	N/A	N/A	No
	Aeromonas spp.	No	Present (Julian *et al.* 2002)	N/A	N/A	No
	Arcobacter spp.	No	Present (McFadden *et al.* 2005)	N/A	N/A	No
	Bacillus spp.	No	Present (Wraight 2003)	N/A	N/A	No
	Bacteroides spp.	No	Present (McDougall 2005)	N/A	N/A	No
	Borrelia spp.	No	Exotic	Yes	Yes	Yes
	Citrobacter spp.	No	Present (Julian *et al.* 2002)	N/A	N/A	No
	Coenonia anatina	No	Exotic	No	Yes	Yes
	Coenonia anatina causes an exudative septicaemia in ducks and geese (Vandamme *et al.* 1999).					
	Actinomyces (Corynebacterium) pyogenes	No	Present (McLachlan 2010)	N/A	N/A	No

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Bacterial diseases (continued).						
Other bacterial diseases (continued)	*Enterobacter* spp.	No	Present (Thompson 1999)	N/A	N/A	No
	Flavobacterium spp.	No	Present (Ubiquitous – Quinn *et al.* 1994)	N/A	N/A	No
	Hafnia spp.	No	Present (Gartrell *et al.* 2007)	N/A	N/A	No
	Helicobacter spp.	No	Present (Varney and Gibson 2006)	N/A	N/A	No
	Klebsiella spp.	No	Present (Varney 2004b)	N/A	N/A	No
	Lactococcus spp.	No	Present (Stone 2005)	N/A	N/A	No
	Lawsonia intracellularis	No	Present (Smits *et al.* 2002)	N/A	N/A	No
	Listeria monocytogenes	No	Present (Varney 2005)	N/A	N/A	No
	Long-segmented filamentous organisms (LSFOs)	No	Unknown	Yes	Unknown	Yes
	Moraxella spp.	No	Present (Vermunt and Parkinson 2000)	N/A	N/A	No
	Mycobacterium avium subsp. *paratuberculosis*	Yes	Exotic strains	No	No	No
	Natural infections of poultry with *Mycobacterium avium* subsp. *paratuberculosis* have not been reported (Barnes and Nolan 2008).					
	Neisseria spp.	No	Present (Alley 2002b)	N/A	N/A	No
	Nocardia spp.	No	Present (Orchard 1979)	N/A	N/A	No
	Oerskovia spp.	No	Unknown	No	No	No
	Infections with *Oerskovia* spp. recorded in pigeons (Barnes and Nolan 2008).					
	Pelistega spp.	No	Unknown	No	No	No
	Pelistega europaea is a newly described bacterium associated with respiratory disease in pigeons (Barnes and Nolan 2008).					

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Bacterial diseases (continued).						
Other bacterial diseases (continued)	*Peptostreptococcus* spp.	No	Present (Graham 1998)	N/A	N/A	No
	Planococcus spp.	No	Exotic	Yes	No	Yes
	Planococcus spp.are usually associated with the marine environment. *P. halophilus* has been associated with multifocal hepatic necrosis in a layer flock (Abdel Gabbar *et al.* 1995) but no reports suggesting an association with ducks have been found.					
	Plesiomonas spp.	No	Present (Staples 2000)	N/A	N/A	No
	Proteus spp.	No	Present (Orr 1995)	N/A	N/A	No
	Pseudomonas spp.	No	Present (Coats 1998)	N/A	N/A	No
	Rothia spp.	No	Present (Thompson 1999)	N/A	N/A	No
	Streptobacillus moniliformis	No	Present (Sakalkale *et al.* 2007)	N/A	N/A	No
	Vibrio spp.	No	Present (Staples 2000)	N/A	N/A	No
Avian chlamydiosis	*Chlamydia psittaci*	Yes	Exotic strains	Yes	Yes	Yes
Fungal diseases						
Aspergillosis	*Aspergillus* spp.	No	Present	N/A	N/A	No
Aspergillus spp. (including *Aspergillus fumigatus*) are listed as organisms known to be present in New Zealand on the NZFUNGI database (see: http://nzfungi.landcareresearch.co.nz/html/mycology.asp).						
Candidiasis (thrush)	*Candida* spp.	No	Present	N/A	N/A	No
Candidiasis is recognised in New Zealand (McCausland 1972).						
Dermatophytosis (favus)	*Microsporum gallinae*	No	Present	N/A	N/A	No
Dermatophytes have been recovered from a number of animal species in New Zealand (Carman *et al.* 1979). An extensive literature search has found no record of *M. gallinae* recovery and there are no published reports of dermatophyte examinations being carried out on poultry specimens. However, the diagnosis of dermatophytosis in poultry in New Zealand is not uncommon (Christensen 2010).						
Dactylariosis	*Dactylaria gallopava*	No	Present	N/A	N/A	No
Although clinical dactylariosis has not been reported in New Zealand, *Dactylaria gallopava* is an environmental fungal organism which causes sporadic opportunistic infections and is found in New Zealand (see http://nzfungi.landcareresearch.co.nz/html/mycology.asp).						
Histoplasmosis	*Histoplasma capsulatum*	No	Exotic	No	No	No
No reports of natural infection of poultry with *H. capsulatum* can be found and birds are not considered to be susceptible to infection with *H. capsulatum*. It is now recognised that the organism prefers to grow in soils enriched with avian manures and, although the disease is avian-associated, the reservoir of infection is in the soil, not birds (Jacob *et al.* 2003).						

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Fungal diseases (continued).						
Cryptococcosis	*Cryptococcus neoformans*	No	Present	N/A	N/A	No
Cryptococcus neoformans is recognised in New Zealand (Varney 2005).						
Zygomycosis (Phycomycosis)	Fungi belonging to the genera *Mucor*, *Rhizopus*, *Absidia*, *Rhizomucor*, and *Mortierella*.	No	Present	N/A	N/A	No
All genera are recognised in New Zealand (see http://nzfungi.landcareresearch.co.nz/html/mycology.asp).						
Macrorhabdosis (Megabacteria)	*Macrorhabdus ornithogaster*	No	Present	N/A	N/A	No
Megabacteriosis has been described in New Zealand (Christensen *et al.* 1997).						
Parasitic diseases						
Nematodes and acanthocephalans	Various	No	Some exotic	Yes	Yes	Yes
Cestodes and trematodes	Various	No	Some exotic	Yes	Yes	Yes
Coccidiosis	*Eimeria acervulina, E. brunetti, E. hagani, E. maxima, E. mitis, E. mivati, E. necatrix, E. praecox, E. tenella* associated with chickens. *Eimeria* spp., *Wenyonella* spp., and *Tyzzeria* spp. recognised in ducks.	No	Some species present, others may be exotic	Yes	Yes	Yes
E. acervulina, *E. brunetti*, *E. maxima*, *E. necatrix*, and *E. tenella* are recognised in New Zealand (McKenna 1998).						
Cryptosporidiosis	*Cryptosporidium baileyi*, *C. meleagridis*	No	Present	N/A	N/A	No
Cryptosporidiosis has been described in New Zealand poultry (Anonymous 1999).						
Cochlosoma anatis infection	*Cochlosoma anatis*	No	Exotic	Yes	Yes	Yes
Histomoniasis (blackhead)	*Histomonas meleagridis*	No	Present	N/A	N/A	No
Histomoniasis described in New Zealand (Black 1997).						
Trichomoniasis	*Trichomonas gallinae*	No	Present	N/A	N/A	No
Canker due to *Trichomonas gallinae* is considered common in New Zealand (Anonymous 1975).						

Table 1 (continued).

Disease	Agent	OIE notifiable	NZ status	Associated with chickens?	Associated with ducks?	Requires further consideration
Parasitic diseases (continued).						
Hexamita	*Spironucleus meleagridis*	No	Exotic?	No	No	No
Hexamitiasis is recognised in turkeys, and has also been associated with pheasants, quail, chukar partridge, and peafowl (McDougald 2008).						
Leucocytozoonosis	*Leucocytozoon simondi, L. caulleryi, L. sabrezi, L. schoutedeni*	No	Some species may be exotic	Yes	Yes	Yes
Leucocytozoonosis due to an organism that morphologically resembles *L. simondi* has been described in yellow-eyed penguins (*Megadyptes antipodes*) (Hill 2008).						
Avian malaria	*Plasmodium spp.*	No	Exotic species?	Yes	Yes	Yes
About 65 species of *Plasmodium* have been described in birds, although only 35 of these are considered valid (Bermudez 2008). *Plasmodium cathemerium*, *P. elongatum*, *P. relictum* and *Plasmosium* sp. have been recognised in New Zealand birds (McKenna 1998).						
Haemoproteus infections	*Haemoproteus spp.*	No	Exotic species?	Yes	Yes	Yes
Around 128 species of Haemoproteus have been reported from birds (Bermudez 2008). *Haemoproteus danilewsky* is recognised in New Zealand (McKenna 1998).						
Sarcocystosis	*Sarcocystis spp.*	No	Exotic species?	Yes	Yes	Yes
Sarcocystis hovathi (*S. gallinarum*) is associated with chickens whereas *S. rileyi* (*Balbiani rileyi*, *S. anatina*) is associated with ducks (Bermudez 2008). *Sarcocystis* sp. has been described in New Zealand (McKenna 1998).						
Toxoplasmosis	*Toxoplasma gondii*	No	Present	N/A	N/A	No
Only sporadic cases of toxoplasmosis in chickens have been reported (Bermudez 2008). A single species of *T. gondii* is the cause of toxoplasmosis in all hosts and *T. gondii* is recognised as present in New Zealand (Hartley and Rofe 2002).						

5.2. HAZARD IDENTIFICATION

For each organism identified as requiring further consideration in Table 1, the epidemiology is discussed, including a consideration of the following questions:

1. Could the imported commodity act as a vehicle for the introduction of the organism?
2. If the organism requires a vector, could competent vectors be present in New Zealand?
3. Is the organism exotic to New Zealand?
4. If it is present in New Zealand,
    i. is it "under official control", which could be by government departments, by national or regional pest management strategies or by a small-scale programme, or
    ii. are more virulent strains known to exist in other countries?

For any organism, if the answer to question 1 is "yes" (and the answer to question 2 is "yes" in the cases of organisms requiring a vector) and the answers to either questions 3 or 4 are "yes", it is identified as a potential hazard requiring risk assessment.

Under this framework, organisms that are present in New Zealand cannot be considered as potential hazards unless there is evidence that strains with higher pathogenicity are likely to be present in the commodity to be imported. Therefore, although there may be potential for organisms to be present in the imported commodity, the risks to human or animal health are no different from risks resulting from the presence of the organism already in this country.

During external scientific review of this risk analysis, the likelihood of surface contamination of poultry during processing was raised as a concern. The MPI position with regard to cross-contamination of carcasses with viruses is that although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern viruses will not multiply on the carcase surface (MAF 1999). Furthermore, the commodity considered in this risk analysis will originate from slaughter and processing plants which operate effective Good Manufacturing Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes[5] which will effectively manage the risk associated with cross-contamination.

5.3. RISK ASSESSMENT

In line with the MPI and OIE risk analysis methodologies, for each potential hazard requiring risk assessment the following analysis is carried out:

a) Entry assessment - the likelihood of the organism being imported in the commodity.

b) Exposure assessment - the likelihood of animals or humans in New Zealand being exposed to the organism.

c) Consequence assessment - the consequences of entry, establishment or spread of the organism[6].

d) Risk estimation - a conclusion on the risk posed by the organism based on the release, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is classified as a risk.

It is important to note that all of the above steps may not be necessary in all risk assessments. The MPI and OIE risk analysis methodologies make it clear that if the likelihood of entry is negligible for a potential hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same situation arises where the likelihood of entry is non-negligible but the exposure assessment concludes that the likelihood of exposure to susceptible species in the importing country is negligible, or where both entry and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

[5] For example, based on Codex Alimentarius guidelines for the control of *Campylobacter* and *Salmonella* in chicken meat, CAC/GL 78-2011.

[6] Detailed analysis of the estimated consequences is not necessary if there is sufficient evidence, or it is widely agreed, that the introduction of a hazard will have unacceptable consequences. In such cases, risk assessment will primarily focus on the likelihood of entry, establishment and exposure (See: www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf)

5.4. RISK MANAGEMENT

For each organism classified as a risk, a risk management step is carried out, which identifies the options available for managing the risk. Where the *Code* lists recommendations for the management of a risk, these are described alongside options of similar, lesser, or greater stringency where available. In addition to the options presented, unrestricted entry or prohibition may also be considered for all risks. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when an import health standard (IHS) is drafted. As obliged under Article 3.1 of the WTO Agreement on the Application of Sanitary and Phytosanitary Measures (the SPS Agreement) the measures adopted in IHSs will be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3 (where measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate following a risk assessment).

5.5. RISK COMMUNICATION

After an import risk analysis has been written, the Imports Standards Team of MPI analyses the options available and proposes draft measures for the effective management of identified risks. These are then presented in a draft IHS which is released together with a risk management proposal that summarises the options analysis, the rationale for the proposed measures and a link to the draft risk analysis. The package of documents is released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to these documents are reviewed before a final IHS is issued.

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6. Avian paramyxovirus-1

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Family: *Paramyxoviridae*, Subfamily: *Paramyxovirinae*, Genus: *Avulavirus* (Alexander and Senne 2008). Nine serogroups of avian paramyxoviruses are recognised, APMV-1 to APMV-9. Newcastle disease (ND) is caused by viruses belonging to serogroup APMV-1, considered below. Viruses belonging to serogroups APMV-2 to APMV-9 are considered in Chapter 7.

The first attempts to classify APMV-1 viruses based on pathogenicity examined chicken embryo mortality after allantoic inoculation. Based on this system, velogenic strains cause mortality at less than 60 hours, mesogenic strains cause mortality between 60 and 90 hours, and lentogenic strains cause mortality after greater than 90 hours (Alexander and Senne 2008). Other tests to determine pathogenicity assess clinical signs or death in infected birds; the intracerebral pathogenicity index (ICPI) in day-old chicks or the intravenous pathogenicity index (IVPI) in six-week-old chickens.

For the ICPI test, diluted virus is injected intracerebrally into each of ten chicks hatched from eggs from a specific pathogen-free (SPF) flock. These chicks must be over 24-hours and under 40-hours old at the time of inoculation. The birds are examined every 24 hours for 8 days. At each observation, the birds are scored: 0 if normal, 1 if sick, and 2 if dead. Birds that are alive but unable to eat or drink should be killed humanely and scored as dead at the next observation. Dead individuals must be scored as 2 at each of the remaining daily observations after death. The ICPI is the mean score per bird per observation over the 8-day period. The most virulent viruses will give indices that approach the maximum score of 2.0, whereas lentogenic and asymptomatic enteric strains will give values close to 0.0 (Alexander 2008). Whilst the ICPI test is considered to be a sensitive measure of virulence, minor variations in the number of birds sick and time of onset may result in markedly different ICPI values for viruses of low virulence (Alexander 1988a).

More recently, the molecular basis of viral pathogenicity has been demonstrated. To replicate, virus must first gain entry to the host target cell. Entry is enabled by a viral protein (the fusion protein) fusing with the host cell membrane. During viral replication a precursor glycoprotein is produced which then has to be cleaved into the fusion protein for the progeny virus to be infectious (Rott and Klenk 1988). The structure of the precursor glycoprotein cleavage site determines the pathogenicity of the virus. Virulent strains have a cleavage site containing multiple basic amino acids, which can be cleaved by a wide range of host proteases enabling these strains to replicate in many different cell types. Low virulence strains have fewer basic amino acids in the cleavage site so are cleaved by a more limited range of host enzymes and their replication is limited to the intestinal tract (Alexander and Senne 2008).

The amino acid sequence at the precursor glycoprotein cleavage site is considered to be an excellent guide to real or potential virulence of viral isolates (Alexander and Senne 2008), although other factors have been described that influence viral virulence (Huang *et al.* 2004; Römer-Oberdörfer *et al.* 2006).

The *Code* (OIE 2011) defines ND as an infection of poultry caused by a virus (NDV) of avian paramyxovirus serotype 1 (APMV-1) that meets one of the following criteria for virulence:

i. the virus has an intracerebral pathogenicity index (ICPI) in day-old chicks (*Gallus gallus*) of 0.7 or greater; or

ii. multiple basic amino acids have been demonstrated in the virus (either directly or by deduction) at the C-terminus of the F2 protein and phenylalanine at residue 117, which is the N-terminus of the F1 protein. The term 'multiple basic amino acids' refers to at least three arginine or lysine residues between residues 113 and 116. Failure to demonstrate the characteristic pattern of amino acid residues as described above would require characterisation of the isolated virus by an ICPI test.

6.1.2. OIE list

Listed.

6.1.3. New Zealand status

Apathogenic and mildly pathogenic (ICPI < 0.2) strains of APMV-1 occur, which have a precursor glycoprotein cleavage site containing no more than two basic amino acids (Pharo *et al.* 2000; Stanislawek *et al.* 2001; Stanislawek *et al.* 2002). Exotic strains of APMV-1 (ND) are notifiable organisms (Tana *et al.* 2011).

6.1.4. Epidemiology

Disease associated with APMV-1 infection varies widely in the type and severity of the syndrome it produces, depending on the viral isolate and strain (Alexander and Senne 2008). Based on the disease produced in chickens under laboratory conditions, five pathotypes have been described (Alexander and Jones 2001):

i. Viscerotropic velogenic ND; a highly virulent form of disease in which haemorrhagic lesions are characteristically present in the intestinal tract.

ii. Neurotropic velogenic ND; an acute, often lethal infection associated with respiratory and nervous signs.

iii. A less pathogenic form of neurotropic ND associated with mesogenic viruses; seen as respiratory and sometimes nervous signs with low mortality restricted to young birds.

iv. Mild or inapparent respiratory infections associated with lentogenic pathotypes.

v. Asymptomatic enteric pathotype; gut infections with lentogenic viruses causing no obvious disease.

Alexander and Senne (2008) concluded that the vast majority of, if not all, birds are susceptible to infection with APMV-1, but the disease seen with any specified strain of virus may vary considerably with host.

It is suggested that spread of infection from one bird to another is primarily via aerosols or large droplets although the evidence to support this is lacking (Alexander and Senne 2008). During infection, large amounts of virus are excreted in the faeces and this is thought to be the main method of spread for avirulent enteric infections which are unable to replicate outside the intestinal tract (Alexander *et al.* 1984).

Brown *et al.* (1999) experimentally infected four-week-old chickens with nine APMV-1 isolates representing all pathotypes. *In situ* hybridisation revealed widespread viral replication in the spleen, caecal tonsil, intestinal epithelium, myocardium, lung, and bursa following challenge with viscerotropic velogenic strains. Neurotropic velogenic strains were associated with viral replication in the myocardium, air sac, and central nervous system. Challenge with mesogenic viral strains was followed by viral replication in the myocardium, air sac and (rarely) in splenic macrophages. Lentogenic isolates resulted in minimal transient viral replication confined to the air sac at 5 days post-exposure and myocardium at 5 and 10 days post-exposure.

Birds slaughtered for meat during disease episodes may represent an important source of virus. Most organs and tissues have been shown to carry infectious virus at some time during infection with virulent NDV (Alexander 1988b). Infected meat has been shown to retain viable virus for over 250 days at -14 to -20°C (Alexander and Senne 2008) and dissemination by frozen meat has been described historically as an extremely common event (Lancaster 1966). Although modern methods of poultry carcase preparation, and legislation on the feeding of untreated swill to poultry, have greatly diminished the risk from poultry products, the possibility of spread in this way nevertheless remains (Alexander 2000).

6.1.5. Hazard identification conclusion

Velogenic isolates replicate in a wide range of body tissues whereas infection with mesogenic and lentogenic isolates is associated with much more limited tissue dissemination. Less virulent APMV-1 strains (i.e. those not falling within the OIE definition of Newcastle disease) are therefore unlikely to be present in the commodity.

Given the biological variability of the ICPI assay, especially when applied to low virulence viruses, claims that New Zealand should be considered free of any strain of APMV-1 with an ICPI>0.2 could be considered not scientifically defensible (Swayne 2010). The OIE definition of Newcastle disease (see 6.1.1 above) incorporates this variability in ICPI results.

All APMV-1 isolates recovered in New Zealand have been shown to have an ICPI<0.7 and a precursor glycoprotein cleavage sequence (residues 113 to 116) containing no more than two basic amino acids. Newcastle disease viruses (as defined by the OIE) are therefore identified as a potential hazard in the commodity.

6.2. RISK ASSESSMENT

6.2.1. Entry assessment

Historically, Lancaster (1966) stated that poultry carcases and offal have been as great a source of NDV as live poultry and have often carried the disease from one country to another.

More recently, MAF (1999) reviewed studies that showed the NDV titer in muscle of infected chickens was about 10^4 EID_{50} (50% egg infectious doses) per gram and the oral infectious dose of NDV in a three-week-old chicken was found to be 10^4 EID_{50} (Alexander 1997), whilst another study demonstrated that tissue pools of muscle, liver, spleen, lung, kidney and bursa collected at 2, 4, 7, and 9 days post-infection were infectious for 3-week-old birds (Lukert 1998). On the basis of these studies, it was concluded that poultry meat is a suitable vehicle for the spread of NDV and that poultry can be infected by the ingestion of uncooked contaminated meat scraps.

The likelihood of entry of NDV is assessed to be non-negligible.

6.2.2. Exposure assessment

Backyard poultry

NDV may be regarded as heat labile and studies have shown that it is likely to be inactivated by domestic cooking (Alexander and Manvell 2004) so there would be a negligible likelihood of backyard poultry being exposed to NDV from scraps of cooked chicken or duck meat. However, NDV can persist in uncooked tissues for prolonged periods and Lancaster (1966) cited a study which demonstrated that the virus remained viable in buried poultry carcases for 121 days.

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

It is assessed that there is a non-negligible likelihood of backyard poultry exposure to NDV from the feeding of raw scraps generated during the domestic preparation of imported chicken or duck meat or from feeding uncooked waste food collected from retail and catering outlets.

Wild birds

Kaleta and Baldauf (1988) concluded that the wealth of reports on ND in free-living birds suggested that virtually all avian species are susceptible to infection although, of the 8,000 known avian species, only 236 (2.5%) had a record of NDV isolation. Since that publication, there has been an increase in the number of species from which NDV has been recovered which led Alexander and Senne (2008) to conclude that the vast majority of, if not all, birds are susceptible to NDV infection.

The likelihood of free-living avian species being infected with NDV, either following exposure to an infected backyard flock or through consumption of uncooked chicken or duck meat in kitchen waste disposed of at sites accessible to susceptible wild avian species is assessed to be non-negligible.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked waste food from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between New Zealand feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (Wintle 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

Recommended minimum biosecurity standards for domestic producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures reduce the likelihood of commercial poultry being exposed to free-living avian species. However, wild birds have been historically implicated in the

introduction and spread of NDV on many occasions (Lancaster 1966) and, more recently, Alexander *et al.* (1998) suggested migratory birds were responsible for the introduction of NDV into British poultry flocks in 1997.

Standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007). Surveys of commercial poultry farms have shown a generally high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents, especially in broiler farms (Rawdon *et al.* 2007; Rawdon *et al.* 2008). However, outbreaks of ND in poultry flocks in the United States in 1975, 1978, and 2002-2003 were associated with backyard game fowl (fighting cocks), with farm employees and proximity to infected backyard game fowl identified as the highest risk factors for commercial flocks (Alexander and Senne 2008). Similarly, trade in backyard flocks and other birds kept for recreational purposes (hobby birds) have been implicated in the introduction and spread of NDV in Europe between 1991 and 1994 (Alexander 2000).

Exposure assessment conclusion

In conclusion, the likelihood of exposure of backyard poultry, wild birds, and commercial poultry to NDV is assessed to be non-negligible.

6.2.3. Consequence assessment

The introduction of NDV would have serious consequences for the poultry industry and could result in substantial mortalities in wild and/or caged birds.

There are reports indicating that both velogenic and vaccine strains of APMV-1 from poultry can cause disease in humans (Yakhno *et al.* 1990; Capua and Alexander 2004; Alexander and Senne 2008). APMV-1 infections in humans have most commonly been reported in association with conjunctivitis, but some reports have referred to chills, headaches, and fever. Given the presence of a lentogenic strain of APMV-1 in New Zealand, the mild and transient nature of the disease and the infrequency of such reports, any consequences to human health are likely to be minor.

The consequences of NDV introduction are assessed to be non-negligible.

6.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and NDV is classified as a risk in the commodity. Therefore, risk management measures can be justified.

6.3. RISK MANAGEMENT

6.3.1. Options

Article 10.9.14 of the current OIE *Code* (OIE 2011) recommends that, for importation of fresh meat of poultry from an ND-free country, zone, or compartment, veterinary authorities should require certification that the entire consignment comes from poultry:

1. which have been kept in an ND free country, zone or compartment since they were hatched or for at least the past 21 days;

2. which have been slaughtered in an approved abattoir in an ND free country, zone or compartment and have been subjected to ante-mortem and post-mortem inspections and have been found free of any sign suggestive of ND.

According to the *Code*, a country, zone, or compartment may be considered free from ND when it has been shown that NDV infection has not been present for the past 12 months, based on surveillance in accordance with Articles 10.9.22 to 10.9.26. If infection has occurred in a previously free country, zone, or compartment, ND free status can be regained three months after a stamping-out policy (including disinfection of all affected establishments) is applied, providing that surveillance in accordance with Articles 10.9.22 to 10.9.26 has been carried out during that three-month period.

Live vaccines derived from low virulence (lentogenic) APMV-1 strains and moderately virulent (mesogenic) APMV-1 strains are used to vaccinate poultry against ND. Inactivated vaccines are also used (Alexander 2008). Mesogenic vaccine viruses (used primarily in countries where ND is endemic) all have two pairs of basic amino acids at their F0 cleavage site and ICPI values around 1.4 so these strains are classified as NDV under OIE criteria (Alexander 2008). Vaccination may protect birds exposed to pathogenic virus from clinical disease but it does not prevent infection and subsequent viral excretion (Parede and Young 1990; Alexander *et al.* 1999), and pathogenic virus may still be recovered from the muscle of infected birds (Guittet *et al.* 1993).

Article 10.9.25 of the *Code* makes provisions for the recognition of ND-freedom in countries, zones, or compartments that practise vaccination against NDV. New Zealand could recognise APMV-1 freedom in a country, zone, or compartment practising vaccination using a lentogenic virus strain with an ICPI < 0.7 or an inactivated APMV-1 vaccine. Vaccine strains with an ICPI ≥ 0.7 would be unsuitable for use in flocks destined for New Zealand.

The OIE *Manual* (Alexander 2008) describes virus isolation, molecular techniques, and serological tests for the diagnosis of ND.

Virus isolation can be performed by egg inoculation with cloacal or tracheal swabs taken from live birds (or pooled organs from dead birds), followed by testing of haemagglutinating activity with monospecific antiserum to APMV-1. The pathogenicity of any APMV-1 isolated could then be assessed by determining the ICPI or by using molecular techniques (reverse-transcription polymerase chain reaction and sequencing). However, the OIE *Manual* makes it clear that *in vitro* molecular testing can confirm if an APMV-1 isolate is virulent but should not be relied on to demonstrate lack of virulence. Failure to demonstrate multiple basic amino acids at the F0 cleavage site using molecular techniques does not confirm the absence of virulent virus (Alexander 2008). Virulent NDV isolates have been described where sequencing has shown an absence of multiple basic amino acids at the F0 cleavage site (Tan *et al.* 2008).

Meat derived from flocks where virus isolation has demonstrated freedom from NDV at slaughter could be considered eligible for import.

Wise *et al.* (2004) have described a real-time reverse-transcription polymerase chain reaction test for detection of NDV in oral or cloacal swabs. This test was found to have a sensitivity of

94% when compared to viral isolation at 4 days post-infection and could therefore be used on a flock basis to demonstrate freedom from NDV.

Haemagglutination inhibition (HI) tests for NDV are widely used and there are a number of commercial enzyme-linked immunosorbent assay (ELISA) kits available. Comparative studies have demonstrated that the ELISAs are reproducible and have a high sensitivity and specificity and they have been found to correlate well with the HI test (Adair *et al.* 1989). Following infection, antibodies appear in the serum within 6-10 days, with the peak response seen after 3-4 weeks (Alexander and Senne 2008). Therefore, serological tests alone cannot reliably demonstrate freedom from infection at the point of slaughter although they may be used as a component of a surveillance programme to demonstrate country, zone, or compartment freedom.

Article 10.9.21 of the *Code* describes the following cooking conditions for poultry meat that will achieve a 7-log reduction in NDV:

Table 2. Temperature/time requirements to inactivate ND virus in poultry meat

Core temperature (°C).	Time (seconds)
65.0	39.8
70.0	3.6
74.0	0.5
80.0	0.03

It would be reasonable to conclude that cooking imported chicken or duck carcases under these conditions would effectively manage the risk of introducing APMV-1.

One or a combination of the following options could be considered in order to effectively manage the risk:

Option 1

Imported chicken or duck meat could be derived from birds kept in a country, zone or compartment free from NDV since they were hatched or for at least the past 21 days. Freedom could be based on surveillance in accordance with Articles 10.9.22 to 10.9.26 of the *Code*.

Vaccination in flocks could be permitted using an inactivated APMV-1 vaccine or a live lentogenic virus strain which is shown to have an ICPI < 0.7.

Option 2

Meat derived from flocks where virus isolation has demonstrated freedom from NDV at slaughter could be considered eligible for import. The pathogenicity of any APMV-1 isolated should be assessed by determining the ICPI.

Option 3

Imported chicken or duck meat could be cooked as specified in Article 10.9.21 of the *Code*.

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7. Avian paramyxoviruses 2-9

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Family: *Paramyxoviridae,* Subfamily: *Paramyxovirinae,* Genus: *Avulavirus* (Alexander and Senne 2008). Nine serogroups of avian paramyxoviruses are recognised, APMV-1 to APMV-9. APMV-1 is assessed in Chapter 6 of this risk analysis.

The prototype strains of APMV-2 to APMV-9 were summarised by Alexander and Senne (2008) as shown in Table 3 (below):

Table 3. Prototype viruses, host range of avian paramyxoviruses (from Alexander, Senne 2008).

Prototype virus strain	Usual natural hosts	Other hosts	Disease produced in poultry
APMV-2/chicken/California/Yucaipa/56	Turkeys, passerines	Chickens, psittacines, rails	Mild respiratory disease or egg production problems, severe if exacerbation occurs
APMV-3*/turkey/Wisconsin/68	Turkeys	None	Mild respiratory disease but severe egg production problems worsened by exacerbating organisms or environment
APMV-3*/parakeet/Netherlands/449/75	Psittacines, passerines	None known	None known
APMV-4/duck/Hong Kong/D3/75	Ducks	Geese	None known
APMV-5/budgerigar/Japan/Kunitachi/74	Budgerigars	None known	No infections of poultry reported
APMV-6/duck/Hong Kong/199/77	Ducks	Geese, rails, turkeys	Mild respiratory disease and slightly elevated mortality in turkeys; none in ducks or geese
APMV-7/dove/Tennessee/4/75	Pigeons, doves	Turkeys, ostriches	Mild respiratory disease in turkeys
APMV-8/goose/Delaware/1053/76	Ducks, geese	None known	No infection of poultry reported
APMV-9/domestic duck/New York/22/78	Ducks	None known	Inapparent infection of commercial ducks.

*Serological tests may distinguish between turkey and psittacine isolates.

7.1.2. OIE list

Not listed.

7.1.3. New Zealand status

Stanislawek *et al.* (2002) recovered APMV-4 from live healthy mallard ducks in New Zealand and found serological evidence for APMV-2, -3, -4, -6, -7, -8, and -9. However, because of cross-reactions and non-specific reactions, the authors were only prepared to claim

their serology findings indicated the presence of APMV-6. A study of caged birds, wild birds, and poultry in New Zealand was unable to find any evidence of APMV-2 or APMV-3 in poultry or APMV-3 in wild birds, and the results of this study did not provide conclusive evidence for the presence of APMV-2 in wild birds (Stanislawek *et al.* 2001).

7.1.4. Epidemiology

Alexander (1993) stated that APMV-2 and APMV-4 tend to be associated with domestic chickens, whilst APMV-4, APMV-6, and APMV-9 are recognised in domestic ducks. However, surveys of wildfowl have demonstrated a wider range of APMV serotypes, with a serological survey in Spain of aquatic wildfowl identifying exposure to APMV -1, -2, -3, -4, -6, -7, -8, and -9 (Maldonado *et al.* 1995).

APMV-2 was first described in 1960 after being recovered from 3-week-old chickens with laryngotracheitis in California (Bankowski *et al.* 1960). Further investigation of this virus (then named myxovirus Yucaipa) identified it as a member of the paramyxovirus group (Dinter *et al.* 1964). APMV-2 has been recovered from both chickens and ducks (Lipkind *et al.* 1982; Goodman and Hanson 1988; Shihmanter *et al.* 1997).

APMV-2 viruses have also been reported in Canada, the former Soviet Union, Japan, the United Kingdom, Germany, Senegal, Czech Republic, Italy, and Israel. The presence of APMV-2 in Israel and Italy was suggested to be associated with the importation of turkey products from North America although subclinical infection of migratory passeriformes has also been suggested as a means of international spread (Alexander 1980).

A recent study recorded the presence of APMV-2 isolates in broilers in China (Zhang *et al.* 2006) and a subsequent serological survey demonstrated widespread seropositivity to APMV-2 (Zhang *et al.* 2007).

APMV-3 isolates are largely associated with captive psittacine and passerine birds (Shortridge *et al.* 1991). APMV-3 has been isolated from turkey flocks (Tumova *et al.* 1979; Alexander 1980; Alexander *et al.* 1983; MacPherson *et al.* 1983), although natural infection of chickens or domesticated ducks with APMV-3 has not been described.

APMV-4 has been recovered from ducks in the United States and Hong Kong (Alexander *et al.* 1979) as well as from wild ducks in Japan (Nerome *et al.* 1984).

APMV-5 was isolated from budgerigar flocks in Tokyo in 1974 (Nerome *et al.* 1978).

APMV-6 isolates have been recovered from migrating wild ducks in Japan (Nerome *et al.* 1984), as well as from ducks and chickens in Hong Kong (Shortridge *et al.* 1980).

Other avian paramyxoviruses of poultry are usually identified as incidental findings during surveillance for avian influenza (Shortridge *et al.* 1980; Alexander and Senne 2008).

APMV-2 and APMV-3 infection of poultry leads to shedding from the respiratory and intestinal tracts (Alexander and Senne 2008). However, there is limited information concerning the epidemiology of avian paramyxoviruses other than APMV-1 (Alexander 2000). Given the similarities between APMV-1 and other avian paramyxoviruses in infection and replication, it has been suggested that the same mechanisms of introduction and spread would apply (Alexander 2000).

7.1.5. Hazard identification conclusion

From the reports cited above, APMV-2, and -6 have been associated with chickens, whereas APMV -2, -4, and -6 have been associated with domestic ducks.

APMV-4 and -6 are recognised in New Zealand, and are not identified as a potential hazard in the commodity.

Replication of APMV-2 is limited to the intestinal and respiratory tracts. Although respiratory and intestinal tissues will be removed from chicken and duck carcases, remnants of these tissues may remain following processing. APMV-2 is therefore identified as a potential hazard in imported whole chicken or duck carcases.

As discussed in Section 5.2, surface contamination during slaughter and processing is likely to be limited so APMV-2 is not identified as a potential hazard in the other commodities considered in this risk analysis.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

Infection with APMV-2 may be associated with mild respiratory signs so infected flocks may not be detected during routine ante and post-mortem inspection. Infected tissues would be limited to any remnants of respiratory or intestinal tissues remaining in chicken or duck carcases after processing. The likelihood of entry is therefore assessed to be very low.

7.2.2. Exposure assessment

Backyard poultry

The heat sensitivity of APMV-2 is likely to be similar to that of APMV-1. Assuming that the mechanisms of introduction and spread for APMV-1 are the same for APMV-2 (Alexander 2000), there is considered to be a negligible likelihood of backyard poultry being exposed to APMV-2 from scraps of cooked chicken or duck meat.

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

Any respiratory or intestinal tissue remnants in imported chicken or duck carcases would be unlikely to be removed prior to cooking although, in the absence of any data to support this, it is assumed that some of this may be discarded as raw tissue prior to cooking and therefore become accessible to poultry.

There is therefore assessed to be a non-negligible likelihood of backyard poultry exposure from raw scraps generated during the domestic preparation of imported whole chicken or duck carcases or from feeding uncooked waste collected from retail and catering outlets.

Wild birds

APMV-2 has been isolated from captive or free-ranging passeriformes, hanging parrots, mynahs, *Neophema* sp., lovebirds, and African grey parrots (Ritchie 1995).

The likelihood of free-living avian species being infected with APMV-2, either following exposure to an infected backyard flock or through consumption of uncooked scraps in kitchen waste disposed of at sites accessible to susceptible wild avian species is assessed to be non-negligible.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked waste food from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (Wintle 2010) although this has now been discarded by at least on large feed manufacturer (Mulqueen 2012).

Recommended minimum biosecurity standards for domestic producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures reduce the likelihood of commercial poultry being exposed to free-living avian species.

Standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007). Surveys of commercial poultry farms have shown a generally high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents, especially in broiler farms (Rawdon *et al.* 2007; Rawdon *et al.* 2008). There have been no reports describing the spread of APMV-2 infection from backyard flocks to commercial poultry.

Exposure assessment conclusion

In conclusion, the likelihood of exposure of backyard poultry, wild birds, and commercial poultry to APMV-2 is assessed to be non-negligible.

7.2.3. Consequence assessment

APMV-2 infection of chickens has been associated with mild respiratory signs. Most APMV-2 infections of passeriformes are mild and self-limiting but infection of psittacines can lead to severe clinical signs including pneumonia, mucoid tracheitis, diarrhoea, and high mortality (Ritchie 1995).

Although NDV is recognised to infect humans, there have been no reports of other APMV serotypes infecting humans (Alexander and Senne 2008). The introduction of APMV-2 would have negligible consequences for human health.

The introduction of APMV-2 in the commodity would be associated with non-negligible consequences to the New Zealand poultry industries and wildlife.

7.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and APMV-2 is classified as a risk in imported whole chicken or duck carcases. Therefore, risk management measures can be justified.

7.3. RISK MANAGEMENT

7.3.1. Options

The *Code* contains no recommendations for sanitary measures appropriate to manage the risk of APMV-2 in poultry meat. However, recognition of country, zone, or compartment freedom from NDV could be extended to include freedom from APMV-2.

Although the OIE *Manual* (Alexander 2008) only describes tests for APMV-1, the samples taken and methods involved for the isolation of other avian paramyxoviruses are identical (Alexander and Senne 2008). Virus isolation can be performed by egg inoculation of cloacal or tracheal swabs taken from live birds (or pooled organs from dead birds), followed by testing of haemagglutinating activity with monospecific antiserum to APMV-2.

Article 10.9.21 of the *Code* describes the following cooking conditions for poultry meat that will achieve a 7-log reduction in NDV:

Table 4. Temperature/time requirements to inactivate ND virus in poultry meat

Core temperature (°C).	Time (seconds)
65.0	39.8
70.0	3.6
74.0	0.5
80.0	0.03

It would be reasonable to conclude that cooking imported chicken or duck carcases under these conditions would effectively manage the risk of introducing APMV-2.

One or a combination of the following options could be considered in order to effectively manage the risk:

Option 1

Chicken or duck meat products that do not contain remnants of intestinal or respiratory tissue could be considered eligible for importation.

Option 2

Imported whole chicken or duck carcases could be derived from birds kept in a country, zone or compartment free from APMV-2 since they were hatched or for at least the past 21 days.

Option 3

Whole chicken or duck carcases derived from flocks where virus isolation has demonstrated freedom from APMV-2 at slaughter could be considered eligible for import.

Option 4

Imported whole chicken or duck carcases could be cooked as specified in Article 10.9.21 of the *Code*.

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8. Avian metapneumovirus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Metapneumovirus* (Gough and Jones 2008). Avian metapneumovirus (aMPV) has been further classified into subtypes A, B, C, and D on the basis of virus neutralisation and sequence analysis (Bäyon-Auboyer *et al.* 1999; Cook and Cavanagh 2002).

Type A and B viruses are found in Europe, Asia, Japan, and South and Central America, whereas type C viruses are found in the United States (Seal 1998; Seal *et al.* 2000; Turpin *et al.* 2002). Two atypical aMPV isolates recovered in France in 1985 (Bäyon-Auboyer *et al.* 1999) were later classified as type D viruses on the basis of sequence analysis (Bäyon-Auboyer *et al.* 2000).

The clinical diseases associated with aMPV infection of poultry are termed turkey rhinotracheitis (TRT), swollen head syndrome (SHS), and avian rhinotracheitis (ART).

8.1.2. OIE list

TRT is an OIE listed disease.

8.1.3. New Zealand status

aMPV is listed as a notifiable organism (Tana *et al.* 2011). In New Zealand the clinical syndrome of TRT or SHS has never been reported and a small serological survey in 1988 found no evidence of TRT antibodies in five different turkey flocks (Horner 1993).

8.1.4. Epidemiology

Turkeys and chickens are considered to be the natural hosts of aMPV (Gough and Jones 2008). aMPV infections were initially described in South Africa, then Europe, the Middle East, Brazil, and the USA. Apart from Australasia, all major poultry rearing regions of the world have reported the presence of aMPV (Gough and Jones 2008). Infection has been estimated to cost the turkey industry in Minnesota around US$ 15 million annually (Rautenschlein *et al.* 2002).

Experimentally infected turkeys show signs of swollen sinuses and nasal discharge, with hyperaemia and exudation seen in the turbinates, sinuses, and trachea at necropsy. Histopathological changes (including inflammatory infiltration, hyperaemia, epithelial hyperplasia, and loss of cilia) are seen in the turbinates, sinuses, and upper and lower trachea (Van de Zande *et al.* 1999).

aMPV infection of chickens is less clearly defined than in turkeys (Gough and Jones 2008). Infection of chickens with aMPV is associated with SHS, which presents as a combination of snicking, conjunctivitis, and swelling around the eyes, over the head and into the submandibular region (Tanaka *et al.* 1995). Gross lesions associated with disease include extensive yellowish gelatinous to purulent oedema in subcutaneous tissue and congestion of the mucosa of the head, neck, and wattles (Lu *et al.* 1994).

aMPV was recovered from a flock of commercial Muscovy ducks associated with coughing and subsequent egg-drop with 2% mortality. The pathogenic role of the virus in this syndrome was unclear as *Riemerella anatipestifer* was also recovered from the birds (Toquin *et al.* 1999). aMPV has also been isolated from sentinel ducks caged near a turkey farm experiencing a TRT outbreak (Shin *et al.* 2000b; Shin *et al.* 2002). However, Pekin ducks have been shown to be refractory to infection with aMPV when inoculated by intranasal instillation (Gough *et al.* 1988).

Bacteria play an important role as secondary pathogens in field and experimental cases of TRT and SHS, with clinical signs being exacerbated and prolonged by concurrent infection with *Bordetella avium*, *Escherichia coli*, *Ornithobacterium rhinotracheale*, or *Mycoplasma gallisepticum* (Cook *et al.* 1991; Naylor *et al.* 1992; Jirjis *et al.* 2004; Marien *et al.* 2005). Co-infection with Newcastle disease virus has also been shown to exacerbate clinical signs and increase morbidity in experimental cases of aMPV infection in turkeys (Turpin *et al.* 2002).

Although early surveys found no evidence of aMPV infection in game birds in the United Kingdom (Gough *et al.* 1990), later studies using a competitive ELISA have indicated that infection is now widespread in pheasants (Gough *et al.* 2001). There is serological evidence of aMPV infection in both reared and free-living pheasants in Italy (Catelli *et al.* 2001). Virus has been identified in pheasants using virus isolation and RT-PCR (Gough *et al.* 2001; Welchman *et al.* 2002). There is serological evidence of aMPV infection in a flock of guinea fowl (Litjens *et al.* 1989). A survey of ostrich farms in Zimbabwe found widespread seroconversion to aMPV (Cadman *et al.* 1994).

Using RT-PCR, aMPV was detected in wild Canada geese, blue-winged teal, sparrows, starlings, a snow goose, and a ring-billed gull in the United States (Shin *et al.* 2000b; Bennett *et al.* 2002; Bennett *et al.* 2004).

Sequence analysis has shown a high sequence identity among wild bird isolates and between wild bird and poultry isolates, suggesting that wild birds may act as a reservoir of infection for poultry (Shin *et al.* 2000b; Bennett *et al.* 2004). No clinical disease has been associated with aMPV infection of wild birds.

Infection is transmitted to susceptible poultry through direct contact or, experimentally, using nasal mucus from infected birds inoculated by the intranasal or intratracheal routes (Alexander *et al.* 1986; McDougall and Cook 1986). There is no evidence of vertical transmission (Gough and Jones 2008).

Following disease introduction, spread occurs rapidly and contaminated water, live animal movements, personnel and equipment have been implicated in outbreaks although spread of aPMVs has only been confirmed by direct contact with infected birds (Gough and Jones 2008). Spread of disease within a country is significantly influenced by the density of the poultry industry (Jones 1996).

Following experimental infection of two-week-old broiler chicks, aMPV RNA can be detected in tissues (blood, lungs, trachea, intestine, and turbinates) for up to 15 days post inoculation (Shin *et al.* 2000a).

Histopathological studies have shown that the main sites of virus replication in experimentally infected chickens and poults are the epithelial cells of turbinates and the lung (Majó *et al.* 1995; Majó *et al.* 1996). An earlier study of experimentally-infected 30-week-old turkeys

demonstrated virus localisation in the turbinates and trachea whilst lungs, air sacs, spleen, ovary, liver, kidney, and hypothalamus were all negative for virus (Jones *et al.* 1988).

Catelli *et al.* (1998) were able to recover large amounts of virus from the nasal tissue, sinus tissue, and trachea of experimentally infected chickens and smaller quantities of virus were recovered from the lungs. No virus was recovered from the kidney, liver, duodenum, bursa of Fabricius, or caecal tonsils. Similarly, Pedersen *et al.* (2001) detected aMPV in the turbinates, sinus, trachea, and lung of experimentally infected four-week-old poults and found that turbinate tissues were significantly more productive sources of virus and viral RNA than were lung and tracheal specimens.

Cook (2000) concluded that the short persistence time of aMPVs in both chickens and turkeys and the restricted tissue distribution of the virus help to minimise the risk of transmission through carcases or processed products.

8.1.5. Hazard identification conclusion

Following infection of chickens and turkeys, virus replication is limited to the respiratory tract tissues. There is no evidence of virus in any other tissues. It is assumed that tissue distribution in ducks would be no different to that described in chickens and turkeys. aMPVs are not identified as a potential hazard in chicken or duck meat or meat products.

Although respiratory tract tissues will be removed from chicken or duck carcases, remnants of these tissues may remain although some of the remaining tissue could be removed during manual inspection. aMPVs are therefore identified as a potential hazard in imported whole chicken or duck carcases.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

Following infection virus is found primarily in the upper respiratory tract. These tissues will be removed from birds at slaughter although it has been previously estimated that approximately 10% of processed chicken carcases would contain some lung tissue, and approximately 0.2% of processed chicken carcases would contain remnants of trachea (MAF 1999). In the absence of any evidence to the contrary, it is assumed that a similar figure would apply to duck carcases.

Considering the above, the likelihood of entry in imported chicken or duck carcases is assessed to be non-negligible.

8.2.2. Exposure assessment

Early studies on TRT virus demonstrated that it was inactivated at 56°C after 30 minutes (Collins *et al.* 1986) so there is considered to be a negligible likelihood of backyard poultry being exposed to aMPVs from scraps of chicken or duck carcases following domestic cooking.

Any respiratory tissue remnants in imported carcases would be unlikely to be removed prior to cooking although, in the absence of any data to support this, it is assumed that some of this may be discarded as raw tissue prior to cooking and therefore accessible to backyard poultry or wild birds. However, spread of aMPVs has only been confirmed by direct contact with infected birds (Gough and Jones 2008).

TRT is widespread in Minnesota but has not spread significantly to other turkey producing areas or into commercial chickens. Furthermore, Minnesota lies directly under a major wildfowl flyway from Canada to Central and South America and there is no evidence of southern spread of type C aMPVs from Minnesota or type A and B viruses from Central and South America (Gough and Jones 2008).

As there is no evidence for the spread of aMPVs other than through direct contact with infected birds, the likelihood of exposure is assessed to be negligible.

8.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and aMPVs are not assessed to be a risk in the commodity. Therefore, risk management measures cannot be justified.

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9. Infectious bronchitis virus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family *Coronaviridae*; Genus *Coronavirus*; Species *Avian Infectious Bronchitis Virus*

The *Coronaviridae* family is divided into two genera, *Coronavirus* and *Torovirus*, but only viruses of the *Coronavirus* genus have been reported to infect birds (Cook 2008). The coronaviruses have been assigned to three groups. The coronaviruses which infect mammals, including humans, belong to groups 1 and 2. Group 3 includes the type species infectious bronchitis virus (IBV) as well as turkey coronavirus (TCoV) (see Chapter 16) pheasant coronavirus (PhCoV) and other emerging avian coronaviruses (Cavanagh 2005).

IBV does not constitute a single homogenous antigenic type. The prototype virus is Massachusetts M41 but a plethora of IBV strains exist and new IBV variants continue to be recognised (Dhinakar Raj and Jones 1997; Cook 2008).

9.1.2. OIE list

Avian infectious bronchitis is an OIE-listed disease.

9.1.3. New Zealand status

Serological testing confirms that IBV is widespread in New Zealand (Watts 2010) and 28 strains have been identified using RT-PCR (Ramneek *et al.* 2005).

In many countries, some of the most frequently used IBV vaccines have been developed from strains isolated either in Europe or in the USA. Vaccine is used in New Zealand although the only live vaccines available are derived from local field strains (Ratanasethakul and Cumming 1983; Howell 1992).

9.1.4. Epidemiology

IBVs are ubiquitous in countries where poultry are reared intensively (Wit *et al.* 2010). There is extensive antigenic variation and differences in virulence and tropism between strains from different geographic regions (McMartin 1993; McFarlane and Verma 2008). The IBVs in Australia belong to a distinct lineage with two genotypically distinct groups. Group I comprises nephropathogenic strains of varying pathogenicity and group II contains three mildly pathogenic respiratory strains (Sapats *et al.* 1996; Ignatovic *et al.* 2006). New Zealand IBV strains are closely related to the group I serotypes from Australia and are distinct from the European and North American strains (Ramneek *et al.* 2005; McFarlane and Verma 2008; Wit *et al.* 2010).

Avian IB is predominantly a respiratory infection, however three clinical manifestations of IBV infection are observed in the field; respiratory disease, reproductive disorders and nephritis (Ignjatovic and Sapats 2000).

The majority of IBV strains around the world, with the exception of Australia and New Zealand, produce prominent respiratory disease (Ignjatovic and Sapats 2000). The QX strain of IBV was first detected in China between 1995 and 2004 and subsequently spread through

Russia and Europe (Irvine *et al.* 2010). The Australian group I strains include the vaccinal strain Vic S as well as the highly nephropathogenic N1/62 (synonym 'T') strains responsible for mortalities of up to 96%. The Australian group II strains only replicate in the trachea and do not induce mortalities (Sapats *et al.* 1996; Ignatovic *et al.* 2006). In New Zealand, the presence of virus may lead to impaired productivity although it is generally not linked to clinical disease (Ramneek *et al.* 2005). The respiratory effects of all New Zealand field strains appear to be mild, but nephritis and outbreaks of uraemia due to IBV have been reported (Lohr 1977a; Howell 1992) and infected layers can have a drop in egg production of up to 12% (Findon 1987).

IBV primarily infects chickens but there are reports of other avian species (racing pigeons, guineafowl, partridge, peafowl and teal) being infected with IBV-like viruses (Barr *et al.* 1988; Ito *et al.* 1991; Cavanagh 2005; Liu *et al.* 2005). These cases were not associated with disease but the isolated viruses were able to infect and cause disease when inoculated into chickens. The capacity of IBV to replicate in other avian hosts without causing overt disease raises potential for these birds to carry and transmit the virus although there is no evidence that this can occur under field conditions (Britton 2011).

Avian coronaviruses are not known to pose any human health risk (Cook 2008).

IBV is highly infectious to chickens of all ages, and under natural conditions will spread between houses within 1 to 2 days, and between farms within 3 to 4 days (Ignjatovic and Sapats 2000).

Respiratory signs are the first and most common clinical manifestation in birds of all ages and include tracheal rales, gasping, sneezing and watery nasal discharge (Cook 2008). In uncomplicated cases these signs are short-lived (10–14 days) and mortalities are generally low. Frequently infection is complicated by secondary infections with organisms such as infectious bursal disease virus, *Mycoplasma gallisepticum*, or *Escherichia coli*. IBV interacts synergistically with these organisms and co-infection is considered the main cause of mortality in older birds (Cavanagh 2007; Lopez and McFarlane 2006).

Nephropathogenic IBV (NIBV) strains initially cause some respiratory signs followed by signs due to kidney damage and mortalities up to 30% (Ignjatovic and Sapats 2000; Cook 2008). Infection of the oviduct can lead to permanent damage in immature birds and a drop in egg production (exceeding 50%) and quality in hens. There are also reports of variant strains causing pectoral myopathy (Dhinakar Raj and Jones 1997) and proventriculitis (Benyeda *et al.* 2010). The QX strain of IBV has been associated with a wide range of clinical problems including respiratory disease, wet litter, mortality due to kidney damage, proventriculitis, and decreased flock performance. Infection of layers with the QX strain has been associated with 'blind' or 'false-layer' syndrome due to oviduct lesions (Irvine *et al.* 2010).

The virus is present in considerable titers in tracheal mucus and faeces during the acute and recovery phases of disease, respectively. The virus may be shed for more than 20 weeks after clinical recovery and can persist in the intestinal tract for several months (Alexander and Gough 1977; Cook 2008). Spread occurs horizontally by aerosol or by ingestion of faeces or contaminated feed or water. The most common source of infection is direct chicken-to-chicken contact, but indirect transmission via mechanical spread, sometimes over long distances, also occurs (OIE 2008). The virus can survive for a considerable time in faeces. True egg transmission is believed to be insignificant.

The upper respiratory tract is the initial site of IBV replication, regardless of strain, following which a viraemia occurs, disseminating the virus to other epithelial surfaces, including the kidney, oviduct, testes, bursa of Fabricius, and alimentary tract (Cavanagh 2007; McMartin 1993).

More recently, Almeida *et al.* (2012) reported the detection of variant IBV in the supracoracoid muscle of chickens using RT-PCR. However, earlier studies of pectoral myopathy associated with IBV failed to recover virus from the musculature of infected chickens using virus isolation or PCR (Trevisol *et al.* 2009, cited in Almeida *et al.* 2012). Similarly, another study of broilers with pectoral myopathy detected IBV in tracheal and caecal tonsil samples but not from muscle tissue (Gomes and Brito 2007, cited in Almeida *et al.* 2012).

Latent infection (from both vaccine and field strains) can establish in the kidney and caecal tonsils for up to 163 days or longer, with subsequent erratic shedding of virus via both faeces and aerosol (Cavanagh and Gelb 2008; Ignjatovic and Sapats 2000) and often with long pauses (up to 42 days) between episodes of shedding (Naqi *et al.* 2003). Virus re-excretion is not generally accompanied by clinical signs and may serve as a source of infection to susceptible chickens (Dhinakar Raj and Jones 1997).

Most strains of IBV are inactivated after 15 minutes at 56°C and after 90 minutes at 45°C (Cavanagh and Gelb 2008). At environmental temperatures of 15-20°C, survival is no more than a few days (McMartin 1993). However, outdoors survival up to 12 days in spring and 56 days in winter has been reported (Cavanagh and Gelb 2008). The virus is rapidly inactivated by common disinfectants (Cook 2008).

9.1.5. Hazard identification conclusion

IB viruses are found in tissues of the respiratory, digestive, reproductive and urinary systems. As discussed in Section 5.2, surface contamination during slaughter and processing is likely to be limited, viruses will not multiply on the carcase surface and contamination will be further reduced during washing. Although a recent RT-PCR study has reported the detection of IBV in the musculature of infected chickens, the failure of other studies to detect virus from muscles using both molecular techniques and virus isolation suggests the titre of virus in muscles is likely to be significantly less than found in tissues such as trachea, lungs, bursa of Fabricius or kidney, which are recognised as sources of virus. IBV is not identified as a potential hazard in chicken or duck meat or meat products.

There are no reports of natural or experimental IBV infection in commercial ducks. There is a single report of a teal infected with an IBV-like virus that was able to infect and cause disease in chickens. No disease was reported in the teal at the time of isolation and it is possible that the virus was a "genuine" teal coronavirus, related genetically but not biologically to IBV (Britton 2011; Cavanagh, 2005; Cavanagh 2011). IBV is not identified as a potential hazard in whole duck carcases.

Chickens infected with IBV may not show gross pathological lesions that would prompt removal from the processing line. Additionally prolonged shedding of IBV can occur following recovery from clinical disease and the trachea, lungs, bursa of Fabricius and kidney are recognised as sources of virus (Naqi *et al.* 2003). As fragments of these tissues may be present in poultry carcases after processing (MAF 1999) and exotic strains of IBV are recognised to be more pathogenic than those present in New Zealand, this virus is identified as a potential hazard in imported whole chicken carcases.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Fragments of infective tissues present in poultry carcases after processing may be a source of IBV and chickens of slaughter age are susceptible to infection. The likelihood of entry of IBV in imported whole chicken carcases is assessed to be non-negligible.

9.2.2. Exposure assessment

Backyard poultry

Most strains of IBV are inactivated after 15 minutes at 56°C and after 90 minutes at 45°C (Cavanagh and Gelb 2008). There is considered to be a negligible likelihood of backyard poultry flocks being exposed to IBV from scraps of cooked whole chicken carcases.

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

IBV can be readily transmitted by the oral route and there is a non-negligible likelihood of exposure to IBV from either raw scraps generated during the domestic processing of imported whole chicken carcases or from feeding uncooked waste food collected from retail and catering outlets.

Wild birds

Evidence suggests that IBV is able to infect and replicate in a wide range of avian species without causing overt disease. Additionally it is known that the virus has a long outdoor survival time in cool climates. The oral dose of IBV sufficient to initiate infection in wild birds is not known. However, the likelihood of free-living avian species being infected with IBV, either following exposure to an infected backyard chicken flock, or through consumption of kitchen waste disposed of at sites accessible to susceptible wild avian species, is assessed to be non-negligible.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked waste food from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (MAF 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

IBV can persist in birds and faeces for several months. Poultry faeces and personnel movements from an infected backyard flock are considered to be likely sources of exposure for commercial poultry farms. However, standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from

keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand, 2007). Surveys of commercial poultry farms have shown a generally high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents, especially in broiler farms (Rawdon *et al.* 2007; Rawdon *et al.* 2008).

Recommended minimum biosecurity standards for domestic producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures reduce the likelihood of commercial poultry being exposed to free-living avian species. Infection of wild birds with IBV, with subsequent spread to poultry, has never been reported and the only evidence that wild birds are able to transmit IBV infection to chickens has been experimental. There are no reports implicating wild birds in the epidemiology of IBV and as described above, the likelihood that IBV would infect a wild bird consuming contaminated meat scraps is extremely low. It is therefore concluded that there is a negligible likelihood of commercial poultry being exposed to IBV through infected wild birds.

Exposure assessment conclusion

In conclusion, the likelihood of exposure of backyard poultry, wild birds, and commercial poultry to exotic strains of IBV is assessed to be non-negligible.

9.2.3. Consequence assessment

In its virulent respiratory forms, IBV is considered the most rapidly spreading virus known in birds (McMartin 1993). In the United Kingdom and the United States of America IBV is the most significant source of economic loss to the broiler industry despite the extensive use of vaccines (Ignjatovic and Sapats 2000; Cavanagh 2007). IBV is highly contagious and is able to spread very rapidly in non-protected birds (Wit *et al.* 2010). The consequence of IBV infection depends on many factors, including the strain of virus, age and breed of chicken, nutrition, environment and intercurrent infections. Most exotic strains result in an acute, highly infectious respiratory disease of chickens, affecting egg production and quality in laying hens, production performance in broilers, and mortalities associated with secondary infections. Infection of commercial poultry flocks would be associated with non-negligible consequences.

Although IBV potentially has a wide host range, disease has only been documented in the chicken and the consequences in other bird species are considered negligible. Non-avian species are not susceptible to infection with IBV.

The consequences of exotic strains of IBV associated with whole chicken carcases are assessed to be non-negligible.

9.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and exotic strains of IBV are classified as a risk in the commodity. Therefore, risk management measures can be justified.

9.3. RISK MANAGEMENT

9.3.1. Options

Circumstantial evidence indicates that unrestricted trade in poultry products has contributed to the spread of some IBV serotypes (Ignjatovic and Sapats 2000). IBV is prevalent worldwide and freedom from IB in conventional, commercial breeding flocks is considered impracticable (McMartin 1993).

IBV is not considered to be a potential hazard in chicken or duck meat or meat products or whole duck carcases and these products could be imported without further sanitary measures.

Most strains of infectious bronchitis virus are inactivated after 15 minutes at 56°C and after 90 minutes at 45°C (Cavanagh and Gelb 2008). Therefore, it is reasonable to assume that cooking whole chicken carcasses would be sufficient to manage the risk of IBV.

Option 1

Whole chicken carcases that have been heated to reach a core temperature of at least 56°C for no less than 15 minutes or at least 45°C for no less than 90 minutes could be considered eligible for import.

Option 2

Whole chicken carcases could be imported from countries where highly pathogenic respiratory IBV strains have not been recognised.

Option 3

Whole chicken carcases from flocks which have not been vaccinated with live vaccines and which showed no clinical signs of IB prior to slaughter could be considered eligible for import.

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10. Infectious laryngotracheitis virus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Family *Herpesviridae*; Subfamily *Alphaherpesvirinae*; Genus *Iltovirus*; Species *Gallid herpesvirus 1* (infectious laryngotracheitis virus, ILTV) (King *et al.* 2012). Clinical disease associated with ILTV infection has been known as laryngotracheitis, infectious laryngotracheitis, or avian diphtheria (Guy and Garcia 2008).

10.1.2. OIE list

Avian infectious laryngotracheitis is an OIE-listed disease.

10.1.3. New Zealand status

Strains of ILTV associated with a mild form of disease have been recognised in New Zealand since 1957 (Webster 1959). Recent reports of ILTV have been associated with respiratory disease in broilers with high morbidity (up to 80 per cent) and low mortality (between one percent and four per cent) (van Andel *et al.* 2010; Varney 2010; Anonymous 2011; Rawdon 2012a; Rawdon 2012b; van Andel 2012).

10.1.4. Epidemiology

Infectious laryngotracheitis (ILT) was first described in 1925 (Beach 1926). Strains of ILTV vary in their virulence from highly virulent strains with high morbidity and mortality to low virulence strains which may be associated with no clinical disease (Cover and Benton 1958; Pulsford and Stokes 1953; Jordan 1966; Sellers *et al.* 2004). ILTV is found throughout the world and disease in commercial poultry is usually well-controlled through the use of modified live virus vaccines (Guy and Garcia 2008).

Infection with ILTV leads to acute respiratory signs, characterised by gasping, coughing, and sneezing (Kernohan 1931). Mild presentations of ILT are common in modern commercial poultry production systems throughout the world (Webster 1959; Linares *et al.* 1994; Sellers *et al.* 2004). These mild forms of disease are associated with slight wheezing, conjunctivitis, swelling of the infraorbital sinuses, and persistent nasal discharge, with low morbidity and very low mortality (no greater than two per cent) (Pulsford and Stokes 1953; Webster 1959; Linares *et al.* 1994; Sellers *et al.* 2004; Guy and Garcia 2008).

Numerous outbreaks of virulent ILTV have been described in Australia since 2007, and studies of recent naturally-infected flocks in Australia have demonstrated a cumulative mortality of 7.5 ± 4.8 per cent (Devlin *et al.* 2011). The emergence of these virulent strains of ILTV co-incided with a shortage of live attenuated vaccine based on SA2 and A20 ILT viruses and the introduction of a live attenuated vaccine using Serva virus. RFLP pattern analysis has identified 9 classes of ILTV circulating in Australia, with A20 and SA2 in class 1, Serva in class 7, and the emerging virulent strains in classes 8 and 9. Preliminary studies of these recent outbreaks hypothesised that class 8 and 9 ILTV may have been subpopulations within the Serva vaccine or arose from *in vivo* passage of the Serva vaccine (Blacker *et al.* 2011).

However, whole-genome sequencing has shown that much of the class 8 and 9 virus genome sequence is identical to the Serva virus with single nucleotide polymorphisms in distinct

genomic regions which are identical or almost identical to sequences in SA2 or A20 viruses. This has lead to the suggestion that virulent class 8 and 9 ILTV viruses in Australia have emerged from the recombination of attenuated herpesvirus vaccine strains (Lee *et al.* 2012).

The chicken is the primary natural host of ILTV (Guy and Garcia 2008). Yamada *et al.* (1980) did demonstrate that ILTV could be recovered from the trachea of experimentally infected ducks although no clinical signs or gross lesions were seen in infected birds. However, there are no other reports of natural or experimental infection of ducks with ILTV. The chicken is the only significant primary host species for this virus and no other reservoir species have been recognised (Hidalgo 2003).

Transmission of ILTV only occurs when virus is placed in the eye, nasal cavity, sinus, or respiratory tract (Beaudette 1937). Exposure of chickens to ILTV vaccines in drinking water cannot be relied upon as it requires accidental contamination of the nasal cavity with vaccine during the act of drinking (Robertson and Egerton 1981).

Following infection, ILTV infects the epithelium of the conjunctiva and respiratory tract. Viral replication is limited to these initial sites of exposure with no evidence of viraemia (Hitchner *et al.* 1977; Bagust *et al.* 1985) although there may be latent infection of the trigeminal ganglia (Williams *et al.* 1992) with re-excretion triggered by stress (Hughes *et al.* 1989).

Gross lesions due to ILTV are seen throughout the respiratory tract of infected chickens but are concentrated in the larynx and trachea and may present as haemorrhage or diphtheritic changes. Gross lesions associated with mild forms of the disease may be limited to oedema and congestion of the conjunctiva and infraorbital sinuses, and mucoid tracheitis (Guy and Garcia 2008).

10.1.5. Hazard identification conclusion

Although ILTV is recognised in New Zealand, reports from overseas indicate that exotic strains of this virus in commercial poultry should be considered more virulent than those described here.

There is no evidence of natural infection of ducks with ILTV. Following infection of chickens, virus replication is limited to the conjunctiva and respiratory tract tissues. There is no evidence of viral replication in any other tissues. ILTV is not identified to be a potential hazard in either whole duck carcases, or chicken or duck meat or meat products.

Although respiratory tract tissues will be removed from chicken carcases, remnants of these tissues may remain although some of the remaining tissue could be removed during manual inspection. Exotic strains of ILTV are therefore identified as a potential hazard in imported whole chicken carcases.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

Following infection virus is found primarily in the upper respiratory tract. These tissues will be removed from birds at slaughter although it has been previously estimated that approximately 10% of processed chicken carcases would contain some lung tissue, and approximately 0.2% of processed chicken carcases would contain remnants of trachea (MAF 1999).

Considering the above, the likelihood of entry in imported chicken carcases is assessed to be non-negligible.

10.2.2. Exposure assessment

Any respiratory tissue remnants in imported carcases would be unlikely to be removed prior to cooking although, in the absence of any data to support this, it is assumed that some of this may be discarded as raw tissue prior to cooking and therefore accessible to backyard poultry or wild birds. However, transmission of ILTV via the oral route requires contamination of the nasal cavity and exposure of the respiratory epithelium. Reflecting this, the likelihood of poultry being exposed to ILTV through the ingestion of raw tissue removed from imported chicken carcases is assessed to be negligible.

10.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and ILTV is not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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11. Avian influenza virus

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Family: *Orthomyxoviridae*, Genus: *Influenzavirus A* (Fauquet *et al.* 2005). Many strains of varying virulence are known.

Influenzavirus A is subtyped based on serologic reactions to the haemagglutinin (H) and neuraminidase (N) surface glycoproteins (WHO Expert Committee 1980). Sixteen subtypes of H and nine subtypes of N are recognised. The distribution of virus subtypes varies by year, geographic location, and host species (Swayne and Halvorson 2008).

11.1.2. OIE list

Highly pathogenic avian influenza (HPAI) and low pathogenic avian influenza (LPAI) in poultry are listed as notifiable diseases.

Article 10.4.1 of the current OIE *Code* (OIE 2011) states that, for the purposes of international trade, avian influenza (AI) in its notifiable form (NAI) is defined as an infection of poultry caused by any influenza A virus of the H5 or H7 subtypes or by any AI virus with an intravenous pathogenicity index (IVPI) greater than 1.2 (or as an alternative at least 75% mortality). NAI viruses can be divided into highly pathogenic notifiable avian influenza (HPNAI) and low pathogenicity notifiable avian influenza (LPNAI):

- HPNAI viruses have an IVPI in 6-week-old chickens greater than 1.2 or, as an alternative, cause at least 75% mortality in 4-to 8-week-old chickens infected intravenously. H5 and H7 viruses which do not have an IVPI of greater than 1.2 or cause less than 75% mortality in an intravenous lethality test should be sequenced to determine whether multiple basic amino acids are present at the cleavage site of the haemagglutinin molecule (HA0); if the amino acid motif is similar to that observed for other HPNAI isolates, the isolate being tested should be considered as HPNAI;

- LPNAI are all influenza A viruses of H5 and H7 subtype that are not HPNAI viruses.

11.1.3. New Zealand status

Influenza A virus in birds is listed as a notifiable organism (Tana *et al.* 2011).

A survey of domestic poultry found no evidence of antibodies to H5 or H7 AI subtypes in broiler, caged/barn layer, or pullet-rearer farms. Three free-range layer farms each had one positive reactor to the H5 subtype and follow-up investigations indicated historic exposure on one of these properties with no evidence of ongoing virus circulation (Tana *et al.* 2007). 170 serum samples from 10 turkey farms were tested in 2007; this found no evidence of exposure to H5 or H7 subtypes (Frazer *et al.* 2008).

A survey of 346 mallard ducks recovered two H5N2 and four H4N6 isolates, and the IVPI of the H5 isolates demonstrated low pathogenicity. 32.5% of sampled ducks showed serologic evidence of exposure to AI (Stanislawek *et al.* 2002). Surveillance of wild birds from 2004 to 2006 isolated 35 AI viruses from resident waterfowl. Subtyping of these isolates identified H1, H2, H4, H7, H10, and H11 subtypes. The H7 isolate was determined to be a low

pathogenic strain (Tana *et al.* 2007). In 2007, a further 34 AI isolates were recovered from resident waterfowl, including two of the H5 subtype. These H5 isolates were determined to be low pathogenic H5N1 strains (Frazer *et al.* 2008; MAFBNZ 2008a).

A recent survey of cloacal swabs from commercial duck, pheasant and quail producers in New Zealand found no evidence of avian influenza viruses (Stanislawek 2010).

A 2003 survey of domestic and wild pigeons found no evidence of AI infections (Black 2004).

11.1.4. Epidemiology

AI viruses are most frequently recorded in waterfowl, which are considered to be the biological and genetic reservoirs of all AI viruses and the primordial reservoir of all influenza viruses for avian and mammalian species (Webster *et al.* 1992; Stallknecht 1998; Perdue *et al.* 2000). Wild birds, particularly migratory waterfowl, may play a major role in the initial introduction of AI viruses into commercial poultry (Halvorson *et al.* 1985; Hinshaw *et al.* 1986b) but once established in commercial poultry, wild birds have very little or no role in secondary dissemination (Nettles *et al.* 1985).

Most AI infections in free-living birds are not associated with disease (Swayne and Halvorson 2008). AI infections have been reported in most domesticated Galliformes and Anseriformes, as well as in emus, ostriches, rhea and psittaciformes (Easterday *et al.* 1997). Galliformes, primarily chickens and turkeys, represent an abnormal host for influenza infection (Suarez and Schultz-Cherry 2000). AI is rare in commercial integrated poultry systems in developed countries but, when infection does occur, it can spread rapidly throughout the integrated system, resulting in epidemics of HPAI or LPAI (Swayne and Halvorson 2008).

Although most influenza viruses found in domestic poultry have been of avian-origin, H1N1, H1N2, and H3N2 swine influenza viruses have also been isolated from flocks experiencing a drop in egg production (Mohan *et al.* 1981; Easterday *et al.* 1997; Suarez *et al.* 2002; Tang *et al.* 2005). In these cases, the proximity of infected flocks to swine operations is consistently suggested as the most likely source of virus.

LPAI has been associated with epidemics of respiratory disease in mink (Englund *et al.* 1986), seals (Lang *et al.* 1981; Webster *et al.* 1981; Geraci *et al.* 1982; Callan *et al.* 1995), and whales (Lvov *et al.* 1978; Hinshaw *et al.* 1986a). In a number of these reported cases, exposure to infected sea birds was suggested as the most likely source of virus. HPAI has been associated with sporadic infections in mammals where there is close contact with or consumption of infected birds (FAO 2006).

LPAI infection of domestic poultry can result in mild to severe respiratory signs including coughing, sneezing, rales, rattles, and excessive lacrimation. Generalised clinical signs such as huddling, ruffled feathers, depression, lethargy, and, occasionally, diarrhoea have also been described. Layers may show decreased egg production. High morbidity and low mortality is normal for LPAI infections (Swayne and Halvorson 2008). Intratracheal inoculation of poultry with LPAI can result in localised infection of the upper and lower respiratory tract (tracheitis, bronchitis, airsaccultitis, and pneumonia) with histological lesions and viral antigen distribution restricted to the lungs and trachea although pancreatic necrosis is also reported (Swayne *et al.* 1992; Shalaby *et al.* 1994; Mo *et al.* 1997; Capua *et al.* 2000). Intravenous inoculation of poultry with LPAI results in swollen and mottled kidneys with necrosis of the renal tubules and interstitial nephritis noted on histopathology and high viral

titers in kidney tissues (Slemons and Swayne 1990; Swayne and Slemons 1990; Slemons and Swayne 1992; Swayne and Slemons 1992; Shalaby *et al.* 1994; Swayne and Alexander 1994; Swayne *et al.* 1994; Swayne and Slemons 1995). However, this renal tropism is strain-specific and is most consistently associated with experimental intravenous inoculation studies (Swayne and Halvorson 2008) although Alexander and Gough (1986) did report the recovery of H10N4 LPAI from kidneys taken from hens presenting with nephropathy and visceral gout. Salpingitis associated with a non-pathogenic H7N2 virus was described by Zielger *et al.* (1999).

In contrast, most cases of HPAI infection of domestic poultry are associated with severe disease with some birds being found dead before clinical signs are noticed. Clinical signs such as tremors, torticollis, and opisthotonus may be seen for 3-7 days before death. Precipitous drops in egg production in breeders and layers are reported. Morbidity and mortality are usually very high (Swayne and Halvorson 2008). HPAI of poultry results in necrosis and inflammation of multiple organs including the cloacal bursa, thymus, spleen, heart, pancreas, kidney, brain, trachea, lung, adrenal glands, and skeletal muscle (Mo *et al.* 1997; Swayne 1997; Perkins and Swayne 2001). Histopathological lesions described include diffuse nonsuppurative encephalitis, necrotising pancreatitis, and necrotising myositis of skeletal muscles (Acland *et al.* 1984). Viral infection of the vascular endothelium is suggested as the mechanism for the pathogenesis of HPAI infections in poultry, especially the central nervous system lesions (Kobayashi *et al.* 1996a; Kobayashi *et al.* 1996b). Viral antigen can be detected in multiple organs, most commonly the heart, lung, kidney, brain, and pancreas (Mo *et al.* 1997).

Infection of wild birds with either HPAI or LPAI usually produces no mortality or morbidity (Swayne and Halvorson 2008) although recent H5N1 HPAI viruses have been associated with deaths in a number of wild bird species in Asia (Ellis *et al.* 2004; Chen *et al.* 2005; Sims *et al.* 2005; Webster *et al.* 2005).

AI virus replicates in the respiratory, intestinal, renal, and reproductive organs and virus is excreted from the nares, mouth, conjunctiva, and cloaca of infected birds (Swayne and Halvorson 2008). Virus transmission is believed to occur by direct contact, through aerosol droplet exposure or via fomites (Easterday *et al.* 1997). However, air sampling during the 1983-84 HPAI outbreak in the northeastern United States did not recover virus from samples taken more than 45m downwind of an infected flock, suggesting airborne transmission is likely to be much less significant for transmission between farms than mechanical movement on fomites (Brugh and Johnson 1987).

An early study found that AI virus persisted in refrigerated muscle tissue for 287 days although feeding meat or blood from a viraemic bird to a susceptible bird did not transmit infection (Purchase 1931). Swayne and Beck (2005) demonstrated that LPAI virus could not be found in the blood, bone marrow, breast or thigh meat of experimentally infected poultry and that feeding breast or thigh meat to a susceptible bird did not transmit infection. However, experimental infection of poultry with HPAI resulted in detectable virus in blood, bone marrow, and breast and thigh meat. An H5N2 isolate was found to achieve only low viral titers in muscle tissue ($10^{2.2\text{-}3.2}$ EID_{50} virus/g) and feeding of susceptible birds with this meat did not transmit infection, whereas an H5N1 isolate achieved a much higher titer in muscle tissue ($10^{7.3}$ EID_{50} virus/g) which was sufficient to achieve transmission in a feeding trial. This study also demonstrated that AI virus vaccination prevented HPAI virus replication in muscle tissue. The authors concluded that their data indicated that the potential for LPAI virus appearing in the meat of infected chickens was negligible, while the potential for having

HPAI virus in meat from infected chickens was high although proper usage of vaccines could prevent HPAI from being present in meat.

11.1.5. Hazard identification conclusion

Studies have shown that LPAI cannot be transmitted to susceptible birds by feeding meat derived from an infected bird. Following natural infection, LPAI virus replication is limited mainly to the respiratory tract tissues although some infectivity might be associated with the pancreas, kidneys and reproductive tract. Notwithstanding the likelihood that some respiratory tract tissues may be present in imported chicken or duck carcases, given the wide range of LPAI viruses that have been described in New Zealand, LPAI is not identified as a potential hazard in imported chicken or duck meat.

HPAI viruses replicate in a wide range of tissues and studies have shown that feeding meat from an infected bird can transmit virus to a susceptible bird. New Zealand is free from all strains of HPAI. HPAI is identified as a potential hazard.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

Swayne and Beck (2005) demonstrated that chicken breast meat was capable of transmitting HPAI (H5N1) to a susceptible bird, resulting in infection and death after 2 days. 80% mortality was described in 4-week-old chickens directly fed an average of <3.5g of breast meat from an infected bird and 100% mortality was described when the meat was added to drinking water.

Although no similar studies have been performed using meat from infected ducks, based on these findings it is reasonable to assume that the likelihood of HPAI being present in either chicken or duck meat should be assessed as non-negligible.

11.2.2. Exposure assessment

Backyard poultry

Thomas and Swayne (2007) studied the thermal inactivation of HPAI in meat from chickens infected intranasally with an H5N1 isolate. This study demonstrated that a core temperature of 70°C for 5.5 seconds would be likely to achieve an 11 log reduction in virus titer and the authors concluded that the U.S. Department of Agriculture Food Safety and Inspection Service time-temperature guidelines[7] would inactivate HPAI in a heavily contaminated meat sample with a large margin of safety.

The study of Swayne and Beck (2005) demonstrates that small scraps of poultry breast meat should be considered capable of infecting susceptible birds so raw scraps generated during the domestic processing of imported chicken or duck meat are likely to be able to transmit infection.

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as

[7] See: http://www.fsis.usda.gov/OPPDE/rdad/FSISNotices/RTE_Poultry_Tables.pdf

backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

There is assessed to be a non-negligible likelihood of backyard poultry being exposed to HPAI from the feeding of raw scraps generated during the domestic preparation of imported chicken or duck meat or from feeding uncooked waste food collected from retail and catering outlets.

Wild birds

Although wild birds are the reservoirs of all AI viruses and play a major role in the introduction of AI viruses in domestic poultry (Swayne and Halvorson 2008), surveillance of wildlife during an H5N2 outbreak in poultry in the United States indicated there was limited transmission of virus from domestic poultry to wild birds and that wild birds had a very limited role in disease dissemination during the outbreak (Hinshaw *et al.* 1986b; Nettles *et al.* 1985). However, due to biosecurity measures on commercial poultry farms, it is reasonable to suggest that there is a much greater likelihood of wild birds being exposed to HPAI from a backyard flock than from a commercial property.

In previous HPAI outbreaks affecting multiple countries, the spread of virus has been directly or indirectly attributable to human activity (Webster *et al.* 2005). However, more recently, infection of wild birds from poultry has been implicated in the spread of H5N1 in Asia (Chen *et al.* 2005; Sims *et al.* 2005; Webster *et al.* 2005).

The likelihood of free-living avian species being infected with HPAI, either following exposure to an infected backyard flock or through consumption of uncooked chicken or duck meat in kitchen waste disposed of at sites accessible to susceptible wild avian species is assessed to be non-negligible.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked waste food from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between New Zealand feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (Wintle 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

Recommended minimum biosecurity standards for domestic producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures reduce the likelihood of commercial poultry being exposed to free-living avian species. However, the introduction of AI viruses to commercial poultry by migratory waterfowl has been documented (Halvorson *et al.* 1985) so the likelihood of exposure of commercial poultry from free-living avian species is assessed as non-negligible.

In most outbreaks of AI investigated, faecal shedding creates a high concentration of virus that may persist in the environment for prolonged periods, and secondary spread from an infected flock appears to follow the movement of people and equipment (Brugh and Johnson 1987).

Standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007). Surveys of commercial poultry farms have shown a generally high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents, especially in broiler farms (Rawdon *et al.* 2007; Rawdon *et al.* 2008).

Exposure assessment conclusion

In conclusion, the likelihood of exposure of backyard poultry, wild birds, and commercial poultry is assessed to be non-negligible.

11.2.3. Consequence assessment

The introduction of HPAI in domestic poultry could result in widespread disease with high mortalities leading to disruption of the poultry industries and export trade in poultry products. The direct and indirect economic costs associated with H5N1 HPAI in Asia from late 2003 to mid 2005 have been estimated to exceed US$ 10 billion (Swayne and Halvorson 2008).

Infection of wild birds with HPAI usually produces no mortality or morbidity (Swayne and Halvorson 2008) although recent H5N1 HPAI viruses have been associated with deaths in a number of wild bird species in Asia (Ellis *et al.* 2004; Chen *et al.* 2005; Sims *et al.* 2005; Webster *et al.* 2005). The impact on native bird species in New Zealand cannot be predicted with any degree of confidence.

Sporadic cases of AI infection of humans have been documented although these have been rare compared to the hundreds of millions of human infections by H1N1 and H3N2 human-adapted influenza viruses that occur each year. Human cases typically present with conjunctivitis, respiratory illness, or flu-like symptoms. Recent Asian H5N1 human cases have been closely associated with exposure to infected live or dead poultry in live poultry markets or villages (Swayne and Halvorson 2008). However, serological surveys of humans in four Thai villages (Dejpichai *et al.* 2009) and a Cambodian village (Vong *et al.* 2006) found no evidence of neutralising antibodies to H5N1 despite frequent direct contact with poultry likely to be infected with this virus, suggesting that the transmission potential from poultry to humans is likely to be low (Swayne and Halvorson 2008).

The introduction of HPAI in the commodity would be associated with non-negligible consequences to the New Zealand poultry industries, wildlife and human health.

11.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and HPAI is classified as a risk in the commodity. Therefore, risk management measures can be justified.

11.3. RISK MANAGEMENT

11.3.1. Options

Article 10.4.20 of the current OIE *Code* (OIE 2011) recommends that, for importation of fresh meat of poultry from an HPNAI-free country, zone, or compartment, veterinary authorities should require certification that the entire consignment comes from poultry:

1. which have been kept in an HPNAI free country, zone, or compartment since they were hatched or for at least the past 21 days;

2. which have been slaughtered in an approved abattoir in an HPNAI free country, zone, or compartment and have been subjected to ante-mortem and post-mortem inspections and have been found free of any sign suggestive of NAI.

According to the *Code*, a country, zone, or compartment may be considered free from HPNAI when it has been shown that HPNAI infection has not been present for the past 12 months, although its LPNAI status may be unknown or, when, based on surveillance in accordance with Articles 10.4.27 to 10.4.33, it does not meet the criteria for freedom from NAI but any NAI virus detected has not been identified as HPNAI virus. If an outbreak of HPNAI occurs in a country previously recognised as free from this disease, under the OIE criteria HPNAI-free status can be regained 3 months after a stamping-out policy is applied, providing that surveillance in accordance with Articles 10.4.27 to 10.4.33 has been carried out during that three month period. Restricting imports of chicken or duck meat to countries, zones, or compartments free from HPNAI as described by the OIE *Code* would effectively manage the risk.

The OIE *Manual* (Alexander 2008) describes both virus isolation and serological tests for the diagnosis of HPNAI.

Virus isolation can be performed by egg inoculation of oropharyngeal and cloacal swabs from live birds (or samples of trachea, lungs, air sacs, intestine, spleen, kidney, brain, liver, and heart from dead birds, either separately or pooled), followed by testing for haemagglutination activity. The presence of influenza A virus is then confirmed using either an agar gel immunodiffusion test or an enzyme-linked immunosorbent assay. Further subtyping of isolates can then be carried out using highly specific antisera or polyclonal antisera raised against a battery of intact influenza viruses. Alternatively, RT-PCR techniques are available to detect the presence of AI virus and the presence of H5 or H7 influenza virus can be confirmed using specific primers.

Meat derived from flocks where virus isolation has demonstrated freedom from H5 or H7 avian influenza viruses at slaughter could be considered eligible for import.

Agar gel immunodiffusion, haemagglutination and haemagglutination inhibition tests are described in the OIE *Manual* for serological diagnosis of AI (Alexander 2008) and ELISAs have been developed to detect antibodies to AI viruses (Swayne and Halvorson 2008). However, antibodies are unlikely to be detected until at least 7 days following infection, so serological assays alone cannot reliably demonstrate freedom from infection at the point of slaughter. However, serology may be used as a component of a surveillance programme to demonstrate country, zone, or compartment freedom.

Swayne and Beck (2005) demonstrated that AI vaccination (using either an inactivated H5N9 vaccine or a recombinant H5 vaccine) prevented HPAI viral replication in breast meat and that breast meat from vaccinated birds that were subsequently infected with HPAI was unable to transmit infection when fed to susceptible birds. The authors of this study suggested that vaccination could be used as a tool to prevent HPAI virus replication in skeletal muscles and thus minimise its potential as a vehicle for transmission of HPAI virus. An earlier study (Capua *et al.* 2002) also demonstrated that an inactivated H7N3 vaccine prevented viraemia and viral replication in pectoral and thigh muscles following challenge with an H7N1 virus.

Vaccination of the source flock with inactivated or recombinant H7 and H5 vaccines could be considered to reduce the likelihood of HPAI virus infection being present in imported chicken or duck meat.

Article 10.4.26 of the OIE *Code* describes cooking procedures recognised to achieve the inactivation of AI virus in poultry meat as shown in Table 5 (below):

Table 5. Temperature/time requirements to inactivate AI virus in poultry meat

Temperature (°C).	Time (seconds)
60.0	507
65.0	42
70.0	3.5
73.9	0.51

Cooking imported chicken or duck meat as specified above could therefore be considered to effectively manage the risk of introducing HPAI.

One or a combination of the following options could be considered in order to effectively manage the risk:

Option 1

Imported chicken or duck meat could be derived from birds kept in a country, zone or compartment free from HPNAI since they were hatched or for at least the past 21 days. Freedom could be based on surveillance in accordance with Articles 10.4.27 to 10.4.33 of the *Code*.

Option 2

Meat derived from chicken or duck flocks where virus isolation has demonstrated freedom from H5 and H7 avian influenza viruses at slaughter could be considered eligible for import.

Option 3

Imported chicken or duck meat could be cooked in accordance with Article 10.4.26 of the OIE *Code*.

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12. Infectious bursal disease virus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Family: *Birnaviridae*, Genus: *Avibirnavirus*, infectious bursal disease virus (IBDV) (Eterradossi and Saif 2008). Two serotypes are recognised (IBDV-1 and IBDV-2) (McFerran *et al.* 1980). Variant and very virulent strains of IBDV-1 (vvIBDV) are described (Rosenberger and Cloud 1986; Chettle *et al.* 1989).

12.1.2. OIE list

Listed.

12.1.3. New Zealand status

IBDV is listed as a notifiable organism (Tana *et al.* 2011). Ongoing industry surveillance has detected no cases of IBD in commercial poultry since 1999 (Brooks 2003; Gerber 2008).

12.1.4. Epidemiology

IBD was first decribed (Cosgrove 1962) in Gumboro, Delaware where 10-20% of birds in infected flocks showed signs of diarrhoea, followed by anorexia, depression, trembling, severe prostration, and death. Younger birds (2-4 weeks-old) were typically affected. Gross lesions initially described were characterised by dehydration, haemorrhage in the leg and thigh muscles, renal changes, and enlargement of the bursa of Fabricius.

Infection with IBDV was linked with immunosupression in 1972 (Allan *et al.* 1972) when prior infection of chickens with IBDV was shown to reduce the effectiveness of subsequent vaccination against Newcastle disease.

A second viral serotype, IBDV-2, was identified in turkeys in 1980 (McFerran *et al.* 1980). Variant strains of IBDV-1 were first recognised in 1986 when chickens vaccinated against IBDV developed lesions typical of IBD when placed on commercial broiler farms (Rosenberger and Cloud 1986). Very virulent strains of IBDV-1 (vvIBDV) were described in 1989 (Chettle *et al.* 1989), which are associated with much higher levels of mortality in infected flocks (Van den Berg *et al.* 1991).

IBDV-1 infections are found worldwide and in countries where the virus is present the incidence is high, either due to natural infection or vaccination. In the USA, variant strains predominate, whereas in Europe, Africa, Asia, and South America vvIBDV strains are predominant (Eterradossi and Saif 2008). IBDV-2 seropositivity is considered to be widespread in chicken flocks in the USA (Jackwood and Saif 1983).

Chickens are the only animals known to develop clinical disease and distinct lesions when exposed to IBDV (Eterradossi and Saif 2008). Variant IBDV-1 strains are associated with few clinical signs but marked bursal lesions, classical IBDV-1 strains are associated with 10-50% mortality, and vvIBDV-1 strains may cause 50-100% mortality in infected flocks (Eterradossi 2010). Although Sivanandan *et al.* (1986) reported bursal necrosis and atrophy in specific-pathogen-free (SPF) chickens experimentally infected with an IBDV-2 isolate, Ismail *et al.* (1988) found that five different IBDV-2 isolates (including the isolate that was

claimed to be used by Sivanandan *et al.* 1986) caused no gross or microscopic lesions in SPF chickens and had no significant impact on bursa-to-body-weight ratio when compared to uninfected controls. It has been subsequently suggested that the isolate used by Sivanandan *et al.* (1986) was an incorrectly labelled IBDV-1 virus (Swayne 2010).

Chickens are most susceptible to infection with IBDV between 3 and 6 weeks of age although the reasons for this age-related susceptibility are poorly understood. Infection is followed by a short incubation period with clinical signs seen 2-3 days after exposure (Eterradossi and Saif 2008).

Following infection, the bursa is the primary target organ of the virus, with marked oedema and hyperaemia seen 3 days after infection (Cheville 1967). Microscopic lesions are seen in the lymphoid tissues with the most marked changes (degeneration and necrosis) being described in the cloacal bursa (Cheville 1967; Mandelli *et al.* 1967; Peters 1967) whilst other histopathological changes have been noted in the spleen, thymus, caecal tonsils, kidney, and liver (Helmboldt and Garner 1964), and also in the Harderian gland (Dohms *et al.* 1981).

Following oral infection of chickens with IBDV, virus can be found in the liver after 5 hours and then progresses to other tissues including the bursa. Following this initial bursal infection there is a second massive viraemia although the peak viral titer in non-lymphoid tissues is much lower than in the bursa and is limited to the viraemic period (Eterradossi and Saif 2008). Virus persists in the bursal tissue of experimentally inoculated chickens for up to 3 weeks, although this period may be shorter in infected chicks with maternal antibodies (Abdel-Alim and Saif 2001).

An IBDV-1 isolate (G14) was recovered from the faeces of a clinically healthy Pekin duck (McFerran *et al.* 1980). Following these findings, Yamada *et al.* (1982) experimentally inoculated a total of 121 ducks with two different strains of IBDV using either the oral or nasal route. Seroconversion was seen in experimentally-inoculated individuals although all ducks remained clinically healthy, no gross lesions were seen in the bursa of Fabricius of inoculated ducks, and no microscopic lesions were seen in the bursa, liver, spleen, kidney, or heart. The ratios of bursa weight to body weight of inoculated ducks and control ducks revealed no obvious differences (Yamada *et al.* 1982).

Sequence analysis of an IBDV strain isolated from a duck (IBDV YL997) demonstrated 97.6% homology with the virulent UK661 strain (Brown *et al.* 1994) and 98.1% homology with the HN942 strain, suggesting this duck isolate to be a vvIBDV strain (Zhang *et al.* 2003).

Three-week-old ducks were experimentally inoculated with either IBDV-1 or IBDV-2 viruses. In both cases, the inoculated ducks demonstrated seroconversion but no clinical signs were noted in the experimental groups. The same study also reported a serological survey of ducks at slaughter that demonstrated widespread exposure to IBDV-2 (Eddy 1990).

In a Nigerian study, domestic ducks were inoculated orally and intraocularly with IBDV-1 but showed no clinical signs, gross lesions or microscopic lesions following inoculation, and no serological response to IBDV was seen (Okoye *et al.* 1990).

A single report (Karunakaran *et al.* 1992) describes clinical signs associated with IBD infection of 5 to 16-day-old ducklings. Infected individuals were observed with tremor, incoordinated gait, torticollis, falling on their sides, paddling movements of legs, and death. At post-mortem nephritis, renal and bursal congestion, and haemorrhage in the thigh muscles were observed.

A serological survey of 380 ducks in China reported that 95.5% of birds were seropositive to IBDV (Wang *et al.* 1997). An IBDV-1 isolate was recovered from these ducks and shown to be pathogenic to SPF chickens (Zhou *et al.* 1998). In another study, chickens inoculated with an IBDV isolate recovered from ducks showed no marked clinical signs but their bursae showed signs of atrophy, lymphoid necrosis, lymphoid depletion, and inflammatory cell infiltration (Wang *et al.* 2007). Similarly, an IBDV-1 isolate recovered from ducks was shown to cause 88-100% mortality in 4-week-old SPF chickens and 40-50% mortality in 8-week-old SPF chickens, whereas IBDV-1 isolates from both chickens and ducks caused no clinical disease, mortality, or gross lesions when inoculated into ducks (Tsai *et al.* 1996).

Sampling of black ducks (*Anas superciliosa*) in Perth, Australia revealed 29% were seropositive to IBDV. One individual sampled was considered to be seropositive to both IBDV-1 and IBDV-2 (Wilcox *et al.* 1983). A survey of Eider ducks found 75% of sampled individuals were seropositive for IBDV, with a high seroprevalence noted in both common Eider (*Somateria mollissima*) in the Baltic Sea and in spectacled Eider (*Somateria fischeri*) in a remote area of Alaska (Hollmén *et al.* 2000). A small serosurvey of indigenous ducks in Nigeria demonstrated widespread seropositivity to IBDV with the authors suggesting that the most likely source of infection was domestic chickens with which they were reared in close association (Oluwayelu *et al.* 2007). However, an earlier survey of 227 ducks in Nigeria found no serological evidence of exposure to IBDV (Okoye 1988). Mai *et al.* (2004) undertook a survey of ducks reared under a relatively uncontrolled extensive management system in Nigeria. None of the 165 individuals sampled were found to be seropositive for IBDV. A smaller survey of ducks in Japan found no antibodies to IBD in either the serum or egg yolk of ducks from two commercial farms (Hirose and Hirai 1976). Geetha *et al.* (2008) collected 183 sera collected from ducks and reported that only two of these were seropositive to IBDV with no clinical signs observed in either of these individuals.

12.1.5. Hazard identification conclusion

IBDV is recognised as a common pathogen of chickens in most poultry-producing countries and there is sufficient evidence to indicate that IBDV may also be found in ducks. IBDV is identified as a potential hazard in both chicken and duck meat.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

Homogenates of bursa, muscle, skin, and fat taken from 4-week-old chickens infected with vvIBDV have been shown to infect 3-week-old recipients when given orally (Quality Control Unit 1997a). Following challenge of 3-week-old chickens with vvIBDV, infectious virus was demonstrated in the liver, kidney, faeces, bursa, and blood 24-96 hours after infection and in muscle homogenates at 48, 72, and 96 hours post infection. The viral titer in muscle was found to peak at $10^{1.17}$ median chick infective doses (CID_{50}) per gram, 3 days post-infection (Quality Control Unit 1997b).

Studies commissioned by MAF have demonstrated that pools of muscle, liver, kidney, spleen, lung, and bursa harvested at 4, 7, 9, 11, 16, 18, and 21 days post-infection with IBDV-1 were all infectious to 3-week-old SPF chickens when given orally (Lukert 1998). A follow-up study was unable to detect infectious virus in the breast muscle of infected 3-week-old birds 7 days after viral challenge whereas liver, lung, kidney, spleen, and bursa contained infectious virus at 7 days post-infection and the bursa was found to remain infectious until 28 days after infection (Lukert 1999).

Studies funded by the Australian government have shown that bursae or muscle tissue, taken from either unvaccinated chickens or chickens vaccinated against IBDV that are subsequently challenged with vvIBDV, contain sufficient virus to transmit infection to naïve chicks (CSIRO 2002). Similar studies using subsequent challenge with variant IBDV found infectious virus to be present in the bursae of both vaccinated and unvaccinated birds challenged with virus although infectious virus was not recovered from the breast and leg muscle of birds that were vaccinated prior to challenge (CSIRO 2004).

Previous quantitative modelling of the likelihood of IBDV introduction in chicken meat (MAF 1999) has concluded that even if only 0.1% of New Zealand's annual broiler consumption was imported from countries with endemic IBD, even in the form of boneless cuts, the introduction of virus would be virtually certain.

Whilst the persistence of IBDV in chicken meat has been thoroughly investigated, there is only one report that has examined the distribution of IBDV in ducks following experimental infection. Oladele *et al.* (2009) infected 5-week-old chicks, turkey poults and ducklings with IBDV-1 and quantified the subsequent distribution of viral antigen in the bursa of Fabricius, thymus, spleen, caecal tonsil, proventriculus, liver, and kidney using immunohistochemistry. This study demonstrated that viral antigen was seen in all of the organs studied up to 168 hours post-infection, although the concentration of antigen (determined by number of positive-staining cells per x40 field of view) was much lower in turkeys and ducks than in chickens. Based on these findings, the authors concluded that ducks were susceptible to IBDV infection but that there was no second massive viraemic phase (as described above in chickens). Oladele *et al.* (2009) also concluded that the chicken host has a facilitating inherent "factor" which permitted maximal replication of IBDV whereas ducks have an innate ability to prevent appreciable replication of IBDV after infection. Current evidence suggests that IBDV should be considered infectious but not pathogenic in ducks, and that seroconversion in these species is limited probably due to poor virus replication (Saif 2011) and although ducks might be carriers of IBDV but there is no evidence to indicate significant replication of pathogenic IBDV-1 in ducks (Eterradossi 2011).

Reflecting the above, the entry assessment for IBDV in chicken meat is assessed to be high whereas the entry assessment for IBDV in duck meat is assessed to be negligible.

12.2.2. Exposure assessment

Backyard poultry

Heat inactivation studies have shown that there is a high probability that IBDV would survive at infectious titers in domestically cooked chicken, especially in deep tissues (MAF 1999).

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

The likelihood of backyard flocks being exposed to IBDV in either raw or cooked imported chicken meat is assessed to be non-negligible.

Wild birds

A survey of avian wildlife in Ireland found evidence of seroconversion to IBDV in rooks and wild pheasants (Campbell 2001) and a Spanish study of birds of prey found evidence of seroconversion to IBDV in birds of the family *Accipitridae* (hawks, eagles, kites, harriers, and Old World vultures) (Hofle *et al.* 2001).

Van den Berg *et al.* (2001) experimentally infected pheasants, partridges, quails and guinea fowls with a very high dose ($10^5 EID_{50}$) of vvIBDV. Guinea fowls were shown to be fully refractory to infection, pheasants and partridges seroconverted but did not excrete virus, and quails were subclinically infected and shed virus in their faeces for several days. The authors concluded that IBDV is highly host-specific and is probably not an infectious disease for the majority of avian species other than the chicken and that game/ornamental birds do not represent a major IBD risk to the poultry industry. It was also noted that Weisman and Hitchner (1978) had failed to produce infection of quail using a lower dose of virus.

Kasanga *et al.* (2008) recently described the detection of IBDV genome in a free-living pigeon in Tanzania. From twenty birds sampled in areas where there were no reported outbreaks of IBD, a single bird was found to be positive by RT-PCR and this individual showed no serological response to IBDV when tested by virus neutralisation. Jeon *et al.* (2008) identified vvIBDV using RT-PCR in a black-billed magpie, two geese, and two ducks in Korea. Before this, Ogawa *et al.* (1998) reported finding two IBDV serotype 1 seropositive rock pigeons from a total of 144 birds of this species sampled in Japan over an eight year period (1989-1997).

A birnavirus isolated from penguins in a UK zoological park was suggested to be IBDV-2 on the basis of testing with monospecific antisera and a virus neutralisation test (Gough *et al.* 2002) and subsequent phylogenetic sequence analysis confirmed the penguin IBDV isolate as a serotype 2 strain (Jackwood *et al.* 2005).

The likelihood of free-living avian species being infected with IBDV, either following exposure to an infected backyard flock or through consumption of kitchen waste disposed of at sites accessible to susceptible wild avian species is assessed to be very low.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked waste food from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between New Zealand feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (Wintle 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

Recommended minimum biosecurity standards for domestic producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures reduce the likelihood of commercial poultry being exposed to free-living avian species.

Only one report of productive infection of wild birds with IBDV has been identified (van den Berg *et al.* 2001) which was achieved through an experimental infection using a very high dose of vvIBDV. The authors of that study concluded that game or ornamental birds investigated in their study do not represent a major IBD risk for the poultry industry.

Although other studies have demonstrated seroconversion of wild birds to IBDV, no studies have shown a natural productive infection of wild birds with this virus.

These findings are consistent with the findings of Biosecurity Australia that, while the establishment of IBDV infection has not been reported in wild birds, wild birds have developed antibody following exposure to the virus, presumably due to transient infection and that there was an extremely low likelihood that vvIBDV would infect a wild bird consuming contaminated meat scraps. Infection of wild birds with IBDV, with subsequent spread to poultry, has not been reported, and it was considered an extremely unlikely event (Biosecurity Australia 2008). It is therefore concluded that there is a negligible likelihood of commercial poultry being exposed to IBDV through infected wild birds.

IBDV is highly contagious and the virus is persistent in the poultry house environment (Eterradossi and Saif 2008). Poultry houses remain infective for a prolonged period after depopulation and water, feed, and droppings taken from an infected house remain infectious for several weeks (Benton *et al.* 1967).

Howie and Thorsen (1981) described the recovery of a non-pathogenic strain of IBDV from the mosquito, *Aedes vexans*, and Okoye and Uche (1986) described rats in Nigerian poultry farms that were seropositive to IBDV. However, there have been no other reports suggesting these may act as vectors or reservoirs of virus (Eterradossi and Saif 2008). Similarly, Pagès-Manté *et al.* (2004) reported the detection of IBDV by RT-PCR performed on the faeces of a dog that had been fed the spleen, liver, bursa, and intestines of four chickens that had each been experimentally infected with $5x10^{4.3}$ EID_{50} vvIBDV. Faeces were positive for virus 24 and 48 hours after ingestion of the infected tissues but not after 72 hours.

Poultry faeces and personnel movements from an infected backyard flock are considered to be likely sources of exposure for commercial poultry farms. However, standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007). Surveys of commercial poultry farms have shown a generally high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents, especially in broiler farms (Rawdon *et al.* 2007; Rawdon *et al.* 2008).

Although 34 farms were found to be infected with IBDV following its introduction into New Zealand in 1993 (Christensen 1995), it has been successfully eradicated from commercial poultry farms in New Zealand without any measures being applied to backyard flocks (Ryan *et al.* 2000; Brooks 2002; Brooks 2003). Furthermore, it has been estimated that, between 1993 and 2001, 8 million processed broilers from IBD-positive flocks were sold into the New Zealand market as fresh or frozen broilers with no further controls and the disease did not re-establish in commercial flocks (Christensen 2009).

However, although the eradication of what was claimed to be a vaccinal strain of IBDV was achieved without any measures being applied to backyard flocks, it would be reasonable to assume that the establishment of a more virulent strain of IBDV in the backyard sector would be likely to result in exposure in any commercial flocks with poor biosecurity.

It is therefore concluded that there is a non-negligible likelihood of commercial poultry being exposed to IBDV through infected backyard flocks.

Exposure assessment conclusion

In conclusion, the likelihood of exposure of commercial poultry, backyard poultry and wild birds is assessed to be non-negligible.

12.2.3. Consequence assessment

Chickens are the only animals known to develop clinical disease and distinct lesions when exposed to IBDV (Eterradossi and Saif 2008). Although there is serological evidence that wild birds may be infected with IBDV, the consequences of infection in wild birds are considered to be negligible.

While IBDV-2 isolates are unlikely to have a clinical impact on infected poultry, classical IBDV-1 strains are associated with 10-50% mortality in infected flocks, variant IBDV-1 strains are associated with few clinical signs but marked bursal lesions, and vvIBDV-1 strains may cause 50-100% mortality in infected flocks (Eterradossi 2010).

Currently the New Zealand poultry industry surveillance scheme for IBDV is estimated to cost around $300,000 per year. Domestic industry has expressed concern that the presence of IBDV2 might result in increased costs for this scheme (Mulqueen 2012).

The consequences of introduction are assessed to be non-negligible for backyard or commercial poultry flocks with poor biosecurity.

12.2.4. Risk estimation

Since the entry, exposure and consequence assessments are all assessed to be non-negligible, the risk is estimated to be non-negligible and IBDV is classified as a risk in chicken meat. Risk management measures are justified.

Since the likelihood of entry was assessed to be negligible for duck meat, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and IBDV is not classified as a risk in imported duck meat.

12.3. RISK MANAGEMENT

12.3.1. Options

Up to 1997 the regulation of garbage feeding to poultry was controlled by the *Garbage (Feed for Swine or Poultry) Regulations 1980*. Under these regulations persons feeding garbage to pigs or poultry had to be licensed and obtain a permit from the Director-General of Agriculture. In 1997 MAF carried out a review of these regulations and it was concluded that they were unenforceable given MAF budgets at that time. The consequences of introduction of IBDV would initially be bourne by backyard poultry flocks and commercial poultry flocks with poor biosecurity. The risks associated with IBDV in chicken meat could therefore be managed through improving flock biosecurity, either through industry-led initiatives or through introducing new domestic legislation. However, it is likely that domestic industry would be reluctant to meet the costs of such measures (Mulqueen 2012).

Any form of meat that minimises trimming or cutting during its preparation prior to cooking and consumption can be expected to pose a lower risk of exposure than whole carcases because of the lower likelihood of scrap generation. Consumer-ready preparations of chicken meat would be considered to have a negligible likelihood of generating waste prior to

consumption. However, given that IBDV in chicken meat would be likely to remain viable after domestic cooking, and feeding of waste food is a recognised practise on New Zealand poultry farms (Mulqueen 2012), limiting imports to consumer-ready forms of poultry meat could not be relied upon to effectively manage the risk.

Although IBD is an OIE-listed disease, Chapter 10.8 of the *Code* contains no recommendations regarding the international trade in poultry meat (OIE 2010). Given the thermal stability of IBDV, realistic cooking times cannot be relied on as a safeguard against IBD virus (MAF 1999). MAF has developed a predictive model to estimate the conditions required to achieve thermal inactivation of IBDV. Based on this model, the following time/temperature combinations are required to achieve >4D (i.e. >99.99%) reduction in IBDV (MAF 2008).

Table 6. Temperature/time requirements to achieve >4D reduction in IBDV based on the MAF CS88 predictive model

Temperature (°C)	Time (minutes)
80	1364
85	500
90	184
95	68
100	25
105	10
110	4
115	2
120	1

No major poultry-producing countries claim to be free of IBDV, therefore the only pre-border option to effectively manage the risk of IBDV introduction in imported chicken meat would be to require demonstration of flock freedom from IBDV.

The OIE *Manual* (Eterradossi 2010) describes test protocols for the detection of specific antibodies to IBDV and detection of the virus in tissues of infected individuals. The agar gel immunodiffusion (AGID) test can be used to detect viral antigen in the bursa of Fabricius. Similarly, an antigen-capture ELISA may be used to detect IBDV antigens in bursal homogenates and an RT-PCR can be used to detect viral genomic RNA in the bursa of Fabricius. AGID, VN and ELISA tests can be used to detect antibodies to IBDV in serum samples. Because infection spreads rapidly in a flock, only a small percentage of a flock need be tested to detect a serological response.

These tests could be used to either demonstrate flock freedom at slaughter or be part of a biosecurity plan to establish a disease-free compartment as described in Chapter 4.3 and 4.4

of the *Code*. MPI is currently reviewing an application for an IBDV-free compartment from Australia and the outcome of this process could be used to inform the development of IBDV-free compartments elsewhere.

One or a combination of the following options could be considered in order to effectively manage the risk:

Option 1

Chicken meat derived from flocks in countries, zones, or compartments that are free of IBDV could be imported without sanitary measures.

Option 2

Chicken meat derived from flocks where testing at slaughter has demonstrated flock freedom from IBDV could be considered eligible for import.

Option 3

Imported chicken meat could be cooked in accordance with the conditions decribed in Table 6 (above).

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13. Duck circovirus

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Family: *Circoviridae*; Genus: *Circovirus*. The *Circovirus* genus includes the species *Beak and Feather Disease Virus* (BFDV), *Canary Circovirus* (CaCV), *Goose Circovirus* (GoCV), *Pigeon Circovirus* (PiCV), *Porcine Circovirus – 1* (PCV-1), and *Porcine Circovirus – 2* (PCV-2).

Sequencing and phylogenetic analyses have confirmed *Duck Circovirus* (DuCV) as a novel and distinct circovirus (Fringuelli *et al.* 2005) and a tentative species in this genus (Fauquet *et al.* 2005), along with *Finch Circovirus* (FiCV) and *Gull Circovirus* (GuCV). DuCV is closely related to GoCV (Hattermann *et al.* 2003; Soike *et al.* 2004).

13.1.2. OIE list

Not listed.

13.1.3. New Zealand status

BFDV (Ortiz-Catedral *et al.* 2009), PiCV (MAF 2009), GuCV (Smyth *et al.* 2006), PCV-1 and PCV-2 (Garkavenko *et al.* 2005) are known to be present in New Zealand.

DuCV has not been recorded in the New Zealand duck population. However, it is recognised that many duck diseases may occur in New Zealand despite them not having been detected (Hemsley 1996). Surveillance programmes of asymptomatic waterfowl and commercial duck farms in New Zealand have been limited and no specific testing for DuCV has been performed. DuCV is prevalent in many parts of the world (Banda *et al.* 2007) and, in the absence of any surveillance to demonstrate freedom, it may be reasonable to assume that DuCV is present in New Zealand.

13.1.4. Epidemiology

The worldwide distribution of DuCV is unknown (Banda *et al.* 2007). It is highly prevalent and widespread in Hungary (Fringuelli *et al.* 2005), Taiwan (ChiouLin *et al.* 2006) and China (ShaoHua *et al.* 2010a, 2010b; ShaoNing *et al.* 2009; Min 2009; Liu *et al.* 2009, 2010) and has also been described in Germany (Hattermann *et al.* 2003; Soike *et al.* 2004) and the United States (Banda *et al.* 2007).

DuCV has been recovered from Muscovy, mulard and Pekin ducks (Hattermann *et al.* 2003; Soike *et al.* 2004; Banda *et al.* 2007) and cross-infection of other avian and mammalian species is considered unlikely (McNulty and Todd 2008). This host-specificity is supported by experimental work (Chen *et al.* 2006; Woods and Latimer 2008) and there are no reports of natural infections in atypical hosts (Woods and Latimer 2008). Circoviruses have never been detected in chickens or turkeys (McNulty and Todd 2008) and the public health significance of avian circoviruses is considered minimal (Woods and Latimer 2008).

The pathogenesis of DuCV, and avian circoviruses in general, is not completely understood and current knowledge is based on field experience, since experimental inoculation in ducks has not been performed (Hattermann *et al.* 2003; McNulty and Todd 2008). However avian

circovirus infections share many characteristics (Soike *et al.* 2004) and it is reasonable to extrapolate from studies of other avian circovirus species.

Circoviral infection is typically reported in juvenile birds but has been reported in birds up to 20 years of age without previous clinical signs of disease (Banda *et al.* 2007; Woods and Latimer 2008). In one study, ducks aged 40-60 days demonstrated greater susceptibility to DuCV infection than those at other ages (ShaoHua *et al.* 2010a). Avian circoviruses are frequently detected in cloacal swabs several months following infection and it is known that adults may be carriers of the virus (McNulty and Todd 2008).

Horizontal transmission, via the respiratory or faecal-oral route, is thought to be the most common route of infection (Fauquet *et al.* 2005; McNulty and Todd 2008) but vertical transmission may also occur, as with BFDV and PiCV (Fauquet *et al.* 2005; Todd *et al.* 2006; McNulty and Todd 2008).

DuCV has a low prevalence in the United States and the infection may not represent a significant problem for the duck industry (Banda *et al.* 2007) although in Hungary, Taiwan, and China DuCV is associated with significant losses up to 70% (Soike *et al.* 2004; Chen *et al.* 2006; Woods and Latimer 2008). However, the factors that dictate the course and outcome of the infection are unknown but may include virus strain, route of exposure, virus dose, levels of maternal antibody and presence of other pathogens (Soike *et al.* 2004; Fringuelli *et al.* 2005; McNulty and Todd 2008; Woods and Latimer 2008). DuCV replicates in healthy birds, with little or no apparent signs of infection, and no pathological significance has been proven (Hattermann *et al.* 2003).

Circoviruses are highly dependent on cellular enzymes for replication, which is typically intranuclear and occurs in targeted rapidly dividing cells such as the basal feather follicular epithelium, lymphoreticular tissues and intestinal crypt epithelium (Schmidt *et al.* 2008; Woods and Latimer 2008). An *in situ* hybridisation study of geese showed that GoCV could be found in all tissue types tested (bursa of Fabricius, spleen, thymus, bone marrow, liver, kidney, lung, and heart) with the exception of brain, and was most abundant in the bursa, liver, and small intestine (Smyth *et al.* 2005). PiCV has additionally been found in the brain and semen of pigeons (Duchatel *et al.* 2009). DuCV DNA has been isolated from extracts of liver, spleen, kidney, bursa, thymus, Harderian gland, heart, blood, lung, and pancreas of ducks (Li *et al.* 2009), but the target cells and major sites of virus replication remain to be identified (McNulty and Todd 2008). Intracytoplasmic globular or botryoid inclusions in these tissues are characteristic of other circovirus infection (McNulty and Todd 2008) but are not a common feature of DuCV infection (Fringuelli *et al.* 2005).

Damage to lymphoreticular tissue impairs both humoral and cellular immune functions. This predisposes birds to secondary pathogens (Soike *et al.* 2004; McNulty and Todd 2008) including *Escherichia coli, Riemerella anatipestifer, Pasteurella multocida,* duck hepatitis virus type 1, and reovirus (Liu *et al.* 2010; ShaoHua *et al.* 2010a). DuCV infection is commonly associated with growth retardation, feathering disorders, and increased rearing losses (Soike *et al.* 2004; Chen *et al.* 2006; Woods and Latimer 2008) as well as signs of secondary infection, including mild arthritis and fibrinous pericarditis (Banda *et al.* 2007). Many birds experience mild, subclinical infections (McNulty and Todd 2008; Liu *et al.* 2010) and DuCV has been isolated in a healthy duck (ShaoHua *et al.* 2010b). No particular clinical syndrome can be specifically associated with DuCV infection (Smyth *et al.* 2005) and most mortalities result from secondary infections (Woods and Latimer 2000).

Studies indicate that circovirus antibody is protective against infection, maternal antibody is somewhat protective, and that cross protective immunity does not develop in avian species exposed to non-host circoviruses. There is no effective treatment for DuCV and attempts should be made to diagnose and treat secondary infections (Woods and Latimer 2000; Woods and Latimer 2008).

PCR has proved to be a specific and sensitive method for diagnosing avian circovirus infection (Fringuelli *et al.* 2005; Chen *et al.* 2006) using various tissues including the bursa of Fabricius and cloacal swabs (Todd *et al.* 2006). Liu *et al.* (2010) developed an indirect enzyme-linked immunosorbent assay method for detecting DuCV-specific antibodies in duck sera. Histological examination alone may not be sufficient to detect infection, as high amounts of circovirus DNA may be present in histologically healthy tissues (Smyth *et al.* 2005).

Circoviruses as a group are environmentally stable and relatively resistant to inactivation by many common disinfectants, acidic environments, and high temperatures (BFDV remains active after incubation at 80°C for 30 minutes) (Woods and Latimer 2008).

13.1.5. Hazard identification conclusion

Avian circoviruses closely related to DuCV are known to be present in New Zealand and DuCV is widespread in many parts of the world. DuCV can be present in healthy ducks (ShaoHua *et al.* 2010b) and there is no evidence to support a pathogenic role for DuCV in ducks (Hattermann *et al.* 2003). No particular clinical syndrome can be specifically associated with DuCV infection (Smyth *et al.* 2005) and most mortalities result from secondary infections (Woods and Latimer 2000).

In the absence of surveillance for DuCV in New Zealand there is no evidence to suggest that it should be considered exotic. Circoviruses have never been reported in chickens. DuCV is therefore not identified as a potential hazard.

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14. Group I adenovirus

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Family: *Adenoviridae*

The family *Adenoviridae* contains four genera (Fitzgerald 2008):

Mastadenovirus	Mammalian adenoviruses
Aviadenovirus	Group I avian adenoviruses, considered in this chapter
Siadenovirus	Group II avian adenoviruses, considered in Chapter 14 of this risk analysis
Atadenovirus	Group III avian adenoviruses (Egg drop syndrome virus and related viruses; see Section 5.1)

The *Aviadenovirus* genus contains five species (A-E) with twelve *Fowl adenovirus* serotypes (FAdV) as well as *Goose adenovirus*, and the tentative species *Duck adenovirus B* (DAdV-2), *Pigeon adenovirus*, and *Turkey adenovirus B* (Smyth and McNulty 2008). Most members of this genus do not have a well-defined role as pathogens, with the exceptions of FAdV strains which cause quail bronchitis and hydropericardium syndrome (Adair and Fitzgerald 2008).

Quail bronchitis virus (QB) and Chicken embryo lethal orphan (CELO) virus are considered to be the same agent and are the type strain of group I, serotype 1 avian adenovirus (FAdV-1) (DuBose and Grumbles 1959; Reed and Jack 2008; Smyth and McNulty 2008).

Hydropericardium syndrome (HPS) (also known as Angara disease, hydropericardium-hepatitis syndrome, infectious hydropericardium, and inclusion body hepatitis/hydropericardium syndrome) is associated with group I, serotype 4 avian adenovirus (FAdV-4) (Hess *et al.* 1999; Ganesh and Raghavan 2000; Schonewille *et al.* 2008).

14.1.2. OIE list

Not listed.

14.1.3. New Zealand status

Serological surveys consistently demonstrate adenoviruses are widespread throughout poultry flocks in New Zealand (Howell, 1992; Anonymous 2001; Poland 2002; Poland 2004). There is also serological evidence of widespread avian adenovirus infection of domestic and wild pigeons (Black 2004).

FAdV-1 (CELO), FAdV-8, and FAdV-12 are recognised as present in New Zealand (Saifuddin 1990; Saifuddin *et al.* 1992). FAdV-4 (HPS) has not been recognised in New Zealand.

14.1.4. Epidemiology

Infections with avian adenoviruses are widespread throughout the world and have been reported in a range of avian species including chickens, geese, turkeys, ducks, pheasants, quail, and budgerigars (Monreal 1992; McFerran 1997; Adair and Fitzgerald 2008). Avian species appear to be susceptible to infection with serotypes of other avian species. For example, in addition to infecting chickens, fowl adenovirus serotypes have been isolated from ducks, guinea fowl, pheasants, turkeys, pigeons, quail, and budgerigars (McFerran 1997; Hess 2000; Adair and Fitzgerald 2008; Smyth and McNulty 2008). It is also known that mixed infections with different FAdV serotypes can occur in the same bird (Hess 2000).

Aviadenoviruses are serologically distinct from members of the other adenovirus genera and they only infect birds (Fauquet *et al.* 2005). There have been no reports of group I adenoviruses infecting mammals (Smyth and McNulty 2008) and they have no known public health importance (McFerran 1997).

The pathogenic role of most of the group I adenoviruses is questionable (McFerran 1997; Hess 2000). Different serotypes, and different strains of the same serotype, vary with regard to their ability to cause disease (Cook 1974; Dhillon and Winterfield 1984; McCracken and Adair 1993; El-Attrache and Villegas 2001). The viruses can be isolated from diseased as well as healthy birds (Monreal 1992) and are readily isolated from the intestinal tract of young and older birds (Cowen *et al.* 1978). It has been suggested that many avian adenoviruses cause disease only in the presence of another pathogen (Adair and Fitzgerald 2008) and the serotypes which do produce disease cause lesions only in a narrow range of organs (Russell 2000).

Following infection, avian adenoviruses can be recovered from the tracheal and nasal mucosa, liver and kidneys, although the highest titers are found in the faeces (Burke *et al.* 1959; Scott and McFerran 1972; Cowen *et al.* 1978; Saifuddin *et al.* 1992; Adair and Fitzgerald 2008). From the available evidence it seems that most strains of avian adenovirus follow the same pattern of infection. Replication occurs in the nucleus, producing characteristic inclusions (Smyth and McNulty 2008). Following initial multiplication, viraemia results in virus spread to virtually all organs (Monreal 1992), especially in the respiratory and alimentary system (McFerran and Adair 1977). At necropsy, necrotising tracheitis, bronchitis, and pneumonia are often present (Smyth and McNulty 2008).

FAdVs are part of a multiple aetiology contributing to respiratory diseases, an infectious hepatitis with inclusion bodies, haemorrhages, and aplastic anaemia (Monreal 1992). Common to all FAdVs (non-virulent and virulent) is the infection of liver cells which can result in inclusion body hepatitis (IBH) (Schonewille *et al.* 2008) and virtually every serotype of fowl *Aviadenovirus* has been isolated from naturally occurring cases of IBH (Smyth and McNulty 2008). The severity of the disease may be either related to the virulence of a particular strain of virus, intercurrent disease, the immune status of the flock or other complicating management factors (Saifuddin *et al.* 1992). Domestic avian species of all ages are susceptible to infection (McFerran 1997).

Most of the group-I avian adenoviruses are excreted in faeces, which may lead to infection through oral route. Airborne and vertical transmission have also been reported (Ashraf *et al.* 2000).

DAdV-2 infection has been linked to an outbreak of disease and mortality in Muscovy ducks in France (Bouquet *et al.* 1982), but its role as a primary pathogen remains unclear (Brash *et*

al. 2009). There are no reports of natural or experimental DAdV-2 infections in chicken. The distribution of this virus in duck populations is unknown (Bouquet *et al.* 1982).

QB virus causes an acute, contagious, highly fatal respiratory disease in young bobwhite quail, (*Colinus virginianus*) (DuBose and Grumbles 1959). There are no reports of natural infection of QB in chickens or ducks (Reed and Jack 2008). CELO virus and other isolates of serotype 1 FAdV are non-pathogenic for chickens under normal circumstances (Saifuddin *et al.* 1992). Chickens and ducks may be experimentally infected with isolates from quail, but develop only very mild or no signs of disease (Yamada *et al.* 1979; McFerran and Smyth 2000).

HPS is caused by fowl adenoviruses serotype 4 which can induce disease in the absence of immune suppression (Naeem *et al.* 1995; Hess *et al.* 1999; Kim *et al.* 2008). HPS differs from IBH only in that the mortality rate and the incidence of HPS are much higher (McFerran and Smyth 2000). HPS was first reported in Pakistan in 1987, and has since been recorded in the Middle East, India, Central and South America, Mexico, Russia, Slovakia, Korea and Japan (Balamurugan and Kataria, 2004; Kim *et al.* 2008). The disease principally affects 3-6 week old broilers, although it has occasionally been seen in older broilers, in broiler breeders aged between 2-32 weeks, and in layers (Cowen 1992; Asrani *et al.* 1997; Ashraf *et al.* 2000). Among other species of birds, HPS has only been recorded in pigeons (Naeem and Akram 1995) and quail (Roy *et al.* 2004). An outbreak of HPS from goslings has been reported but the virus that was isolated was identified as a goose adenovirus (GAdV) (Ivanics *et al.* 2010).

Natural outbreaks of HPS are characterised by the sudden onset of the disease with few or no clinical signs other than sudden heavy mortality (Cowen 1992; Chandra *et al.* 2000). Birds become severely anaemic and in the terminal stages may become dull and depressed (Chandra *et al.* 2000). The course of disease is usually 10-15 days, and mortality, which begins at around 3 weeks of age, peaks at 4-5 weeks then declines, serves as the main clinical feature (Kim *et al.* 2008). Rates of mortality vary, with reports of 20-70% in Pakistan, 10-30% in Iraq, 10-80% in India, 3.5-30% in Russia, and 1.3-11.1% in Korea (Balamurugan Kataria 2004; Kim *et al.* 2008). Such differences in mortality rates could be the result of differences in the age and types of chickens infected or differences in the dose of virus and the route of infection (Kim *et al.* 2008). Although mortality in broilers is usually high, mortality in layers or breeders is lower (10-20%) (Ashraf *et al.* 2000; Kim *et al.* 2008).

HPS is marked by hydropericardium, hepatitis, pulmonary oedema and inclusion body hepatitis and the typical gross lesion in over 90% of dead birds (Anjum *et al.* 1989) is the accumulation of clear, straw coloured fluid in the pericardium (Naeem and Akram 1995; Ashraf *et al.* 2000). Other lesions include discoloured and friable liver, pale kidneys, and a flaccid heart (Ashraf *et al.* 2000; McFerran and Smyth 2000; Roy *et al.* 2004). Variable natural and experimental incubation periods (2-18 days) have been reported (Ganesh and Raghavan 2000), yet assuming that the natural infection occurs by the oral route, experiments indicate that the disease has a very short incubation period (Abdul-Aziz and Hasan 1995).

Adenovirus excretion within a broiler flock with HPS will peak between 4 and 6 weeks of age, whilst in layer replacements peak excretion of adenoviruses between 5 and 9 weeks is described (Mazaheri *et al.* 2003). Because persistence of FAdV-4 in broiler breeders has been demonstrated the virus can be continually shed over a period of several months (Kim *et al.* 2008) and the virus can remain latent in SPF flocks for at least one generation (McCracken and Adair 1993), it is unclear if breeder stocks are the major reservoirs of infection (Ashraf *et al.* 2000).

Following vertical transmission, rapid horizontal spread between and within flocks occurs through contaminated faeces and mechanical vectors (Cowen 1992; Akhtar 1994; Ganesh and Raghavan 2000; Mazaheri *et al.* 2003; Balamurugan and Kataria 2004). Bird-to-bird transmission occurs by the oral-faecal route (Balamurugan and Kataria 2004). An experimental study found that one-day-old chicks died after oral application with low doses of FAdV-4 but that higher doses were required, via intramuscular injection, to induce mortality in SPF birds older than one week of age (Mazaheri *et al.* 2003).

Studies have indicated that FAdV-4 may persist following processing at 60°C for 30 minutes or 50°C for 1 hour, although it is likely to be inactivated following processing at 60°C for 1 hour, 80°C for 10 minutes, or 100°C for 5 minutes (Afzal *et al.* 1991). Although adenoviruses may remain viable for some time, they will not multiply in the environment (McFerran and Smyth 2000).

Roy *et al.* (2004) reported that an inactivated vaccine prepared from the liver of affected birds was found to be highly effective in controlling the disease, although field observations suggest that formalin-inactivated, oil-adjuvanted and formaldehyde inactivated liver organ vaccines fail to provide adequate protection from HPS (Kahn *et al.* 2005; Mansoor *et al.* 2011). In fact Kahn *et al.* (2005) observed that most outbreaks of HPS are truly post-vaccination. Current studies of live attenuated vaccines appear to significantly reduce the adverse effects of HPS but field trials in commercial broiler chicks have not yet been performed (Mansoor *et al.* 2011).

By virtue of its acute nature and sudden onset, the clinical diagnosis of HPS is difficult (Kahn *et al.* 2005). The disease can be diagnosed serologically by agar gel immunodiffusion (AGID) and counter immunoelectrophoresis tests (Roy *et al.* 2004).

14.1.5. Hazard identification conclusion

Group I avian adenovirus infections are widespread in poultry in New Zealand. McFerran and Smyth (2000) suggested that there are no trade implications for conventional avian adenovirus infections except for highly virulent viruses associated with hydropericardium syndrome (which has not been described in ducks) or inclusion body hepatitis (which is recognised in New Zealand). There is only limited evidence to support a pathogenic role for group I avian adenoviruses in ducks and there is no evidence that exotic serotypes/strains likely to be present in ducks are more pathogenic than those in New Zealand. Group I avian adenoviruses are not identified as a potential hazard in duck meat, duck meat products or whole duck carcases.

Although chickens are the natural hosts of FAdV-4 and the virus may be found in many visceral organs, the risk of transmission of virus through poultry meat appears to be small (McFerran and Smyth 2000). This is because flocks infected with significant adenoviruses would show evidence of disease and accordingly should not be slaughtered for human consumption (McFerran and Smyth 2000). Additionally, adenoviruses will not multiply in carcase meat (McFerran and Smyth 2000) and it has been shown that high doses are required in order to induce mortality in birds older than 1 week of age (Mazaheri *et al.* 2003).

The Australian Biosecurity IRA team considered the effect of vaccination on disease expression, and concluded that infected flocks which had been vaccinated might escape detection, and therefore would not be rejected from slaughter (Biosecurity Australia 2008). While this cannot be ignored, current reports from vaccinating countries indicate that commercially available vaccines fail to provide adequate protection from HPS and that most

outbreaks of HPS are truly post-vaccination (Kahn *et al.* 2005; Mansoor *et al.* 2011). Live attenuated vaccines promise to be more effective but are only in the trial stages and are not available commercially (Mansoor *et al.* 2011).

Group I avian adenoviruses are therefore not identified as a potential hazard in chicken meat, chicken meat products or chicken carcases.

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15. Avian adenovirus splenomegaly

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Family: *Adenoviridae*. Genus: *Siadenovirus.* This genus comprises three species: *Frog Adenovirus* (FrAdV-1), *Raptor Adenovirus A* (RAdV-1) and *Turkey Adenovirus A* (TAdV-3) (Kovacs and Benko 2009; ICTV 2011) and four unclassified Siadenoviruses: *Budgerigar Adenovirus 1* (BuAdV-1) (Katoh *et al.* 2009), *Great Tit Adenovirus 1* (GTAdV-1) (Kovacs *et al.* 2010), *Plum Headed Parakeet Adenovirus 1* (also known as *Psittacine AdV-2* (Wellehan *et al.* 2009)), and *Sulawesi Tortoise Adenovirus 1* (Rivera *et al.* 2009).

Avian adenovirus splenomegaly virus (AASV) of chickens, turkey haemorrhagic enteritis virus (THEV), and marble spleen disease virus (MSDV) of pheasants are all considered to be strains of TAdV-3, formally known as a group 2 avian adenovirus (Smyth and McNulty 2008). The three strains can be differentiated using restriction endonuclease fingerprinting and monoclonal antibody affinity (Zhang and Nagaraja 1989).

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

Avian adenovirus splenomegaly of chickens has never been reported and AASV is considered exotic. Haemorrhagic enteritis has been described on one occasion in a turkey flock in New Zealand. However, there is no evidence that the virus is established here (MAF 2010).

15.1.4. Epidemiology

Avian adenovirus splenomegaly (AAS) is a disease of chickens characterised by splenomegaly, pulmonary oedema, and congestion (Pierson and Fitzgerald 2008). AAS affects birds of all ages and is primarily recognised in market-age broiler breeders (Pierson and Domermuth 1997).

Serological evidence indicates that AASV is widespread in chickens in many countries, with reports of up to 50% of chickens in up to 46% of flocks affected (Domermuth *et al.* 1978b, 1979, 1980). In spite of this widespread serologic conversion, clinically affected birds demonstrating viral inclusions are rare (Fitzgerald *et al.* 1994) and AASV is thought by some not to be a primary pathogen (Hess 2000).

Published information on the epidemiology of AASV is scarce, however AASV differs from THEV only at the genomic level (Pierson and Fitzgerald 2008) and therefore the information for THEV is assumed to be applicable.

A variety of avian species including guinea fowl, chukars and psittacines are reported to be susceptible to experimental or natural infection with strains of TAdV-3 (Cowan *et al.* 1988; Gómez-Villamandos *et al.* 1995; Pierson and Fitzgerald 2008; Shivaprasad 2008). A survey of backyard poultry and other avian species near a commercial turkey flock revealed the presence of antibodies against THEV despite restricted visitor contact, and infection of wild birds could be ruled out (McBride *et al.* 1991). However, a serologic survey of 42 wild bird

species in a similar region revealed no evidence of TAdV-3 infection indicating that infection of wild birds is not commonplace (Domermuth *et al.* 1977). Mammals are not susceptible to infection with TAdV-3 and AASV poses no threat to human health (Smyth and McNulty 2008).

Disease associated with AASV is not considered a major problem (McFerran and Smyth 2000; Smyth and McNulty 2008). AASV infection is recognised as splenomegaly in broilers at slaughter and as splenomegaly with pulmonary oedema/congestion in adult chickens (McFerran and Smyth 2000). Morbidity is high but clinical signs are not generally severe (Pierson and Fitzgerald 2008). Mortality is unusual although respiratory compromise can lead to peracute or acute death of chickens and 8.9% mortality has been recorded in one outbreak (Domermuth *et al.* 1982).

Horizontal transmission of TAdV-3 occurs via the faecal-oral route. There is no evidence of vertical transmission (Chandra and Kumar 1998; Pierson and Fitzgerald 2008).

The pathogenesis of AASV infection in chickens is poorly understood. Avian siadenoviruses appear to have an affinity for lymphoid tissue and are lymphocytopathic (Veit *et al.* 1981; Pierson and Fitzgerald 2003). The spleen appears to be the major site of viral replication and macrophages and B lymphocytes are considered to be the primary target cells (Veit *et al.* 1981; Fasina and Fabricant 1982; Gómez-Villamandos *et al.* 1994; Pierson and Fitzgerald 2008). Infected cells have also been identified in the intestine, bursa of Fabricius, caecal tonsils, thymus, liver, kidney, peripheral blood leukocytes, lung, and spleen. A short transitory viraemia accompanies the appearance of virus in these tissues (Fasina and Fabricant 1982).

THEV is relatively resistant and can remain viable for several weeks in carcases protected from drying or in wet faecal material or contaminated litter (Pierson and Fitzgerald 2008). Heating at 65°C for 1 hour, wet storage for 4 weeks at 37°C, 6 months at 4°C, or 4 years at -20°C does not alter infectivity. However, THEV is inactivated by heating at 70ºC for 1 hour or by drying at 37ºC or 25ºC for 1 week (Domermuth and Gross 1971).

Vaccines for AASV have not been developed (Pierson and Fitzgerald 2003).

15.1.5. Hazard identification conclusion

AAS has never been reported in ducks and there is no evidence that ducks are susceptible to infection with AASV. AASV is not identified as a potential hazard in duck meat, duck meat products or imported whole duck carcases.

Following infection of chickens, evidence indicates that viral replication is limited to reticuloendothelial tissues and infectivity is concentrated in the spleen. AASV is not identified as a potential hazard in chicken meat or chicken meat products.

Although evisceration will remove the vast majority of infectivity from chicken carcases, visceral remnants may remain following automated processing. Automated eviscerators are said to be 87-94% efficient (MAF 2010). AASV is therefore identified as a potential hazard in imported whole chicken carcases.

15.2. RISK ASSESSMENT

15.2.1. Entry assessment

Consensus among studies is that the spleen is the most commonly and most consistently affected organ and the most infectious tissue (Domermuth *et al.* 1972; Silim and Thorsen 1981; Fasina and Fabricant 1982). Given the anatomical location of the spleen (dorsal to the right lobe of the liver between the proventriculus and ventriculus) it is unlikely that remnants of splenic tissue would remain in chicken carcases following automated evisceration.

Viraemia occurs only at the peak of clinical disease and coincides with marked splenic pathology (splenomegaly and mottling) which is likely to be detected at slaughter. Faecal contamination during slaughter might result in limited contamination of the skin of an infected bird but, unlike bacteria of public health concern viruses will not multiply on the carcase surface. Furthermore, the commodity considered in this risk analysis will originate from slaughter and processing plants which operate effective Good Manufacturing Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes[8] which will significantly reduce the likelihood of cross-contamination.

Considering the above, the likelihood of AASV entry in imported whole chicken carcases is assessed to be negligible.

15.2.2. Risk estimation

Since the likelihood of entry is assessed to be negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and AASV is not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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[8] For example, based on Codex Alimentarius guidelines for the control of *Campylobacter* and *Salmonella* in chicken meat, CAC/GL 78-2011.

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16. Reovirus

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

Family: *Reoviridae*, Genus: *Orthoreovirus*. Five orthoreovirus species are recognised, with avian reoviruses classified in group II (Fauquet *et al.* 2005).

In poultry, reoviruses are principally associated with viral arthritis/tenosynovitis (Olson and Kerr 1966; Rosenberger and Olson 1997).

16.1.2. OIE list

Not listed.

16.1.3. New Zealand status

Avian reoviruses were first recovered from chickens in New Zealand in 1975, although these isolates were not associated with a definitive clinical disease (Green *et al.* 1976). Tenosynovitis in commercial broiler breeder flocks was first described in 1978 (Bains and Tempest 1978).

Saifuddin *et al.* (1989) described the isolation of a reovirus from the livers of broiler chickens in a flock with high early mortality rates, although the pathogenicity of this isolate was unknown.

Serosurveillance of commercial poultry consistently demonstrates a high seroprevalence to reoviruses in broilers and breeder birds in New Zealand (Poland 2002; Poland 2004; Poland 2005; Frazer 2008).

Muscovy duck reovirus (DRV) has not been recognised in New Zealand although there has been no active surveillance for this pathogen.

16.1.4. Epidemiology

Reoviruses are considered ubiquitous in commercial poultry and are generally thought to be harmless (Jones 2008). Reoviruses have been isolated from poultry affected by a number of disease conditions including viral arthritis/tenosynovitis, respiratory disease, malabsorption syndrome, enteric disease, stunting syndrome, and immunosupression (Dees *et al.* 1972; Sterner *et al.* 1989; Rosenberger and Olson 1997; van der Heide 2000).

Reoviruses have been recovered from healthy commercial chicken flocks, flocks with runting and flocks with viral arthritis/tenosynovitis, and the widespread occurrence of reoviruses in both normal and diseased chickens indicates that the isolation of reoviruses from diseased chickens does not necessarily imply an aetiological relationship of reovirus with disease (Robertson *et al.* 1984).

Viral arthritis/tenosynovitis is recognised to be present in New Zealand so is not considered further here (Howell 1992).

Reoviruses have been associated with respiratory disease in chickens (Subramanyam and Pomeroy 1960) although they are generally not regarded as primary respiratory pathogens in poultry (Jones 2008).

Reoviruses have been recovered from chicks with severe cloacal pasting (Dutta and Pomeroy 1967; Deshmuck and Pomeroy 1969) although studies which infected one-day-old specific-pathogen-free (SPF) chicks with reoviruses associated with enteric disease have been inconclusive (Apple *et al.* 1991).

Reovirus infection was shown to potentiate the pathogenic effect of *Cryptosporidium baileyi* in SPF chickens although reovirus infection alone had no clinical or histological affect on the birds (Guy *et al.* 1988). Similarly, reoviruses have been shown to potentiate the clinical effect of coccidial infections in broilers (Ruff and Rosenberger 1985). Reovirus infections have been shown to enhance the pathological lesions caused by IBDV (Moradian *et al.* 1990) and enhance susceptibility to experimental challenge with *Escherichia coli* isolates (Rosenberger *et al.* 1985). Reovirus inoculation of SPF chickens that had been experimentally immunosuppressed was shown to result in a lethal hepatic infection (Kibenge *et al.* 1987).

A number of studies have investigated the role of reoviruses in malabsorption/runting syndrome, characterised by severe mucoid diarrhoea, pale intestinal tracts with watery contents, and stunted growth (see also Chapter 18). Vertommen *et al.* (1980) and Decaesstecker *et al.* (1986) were unable to demonstrate a significant pathogenic role for reoviruses in this syndrome whereas Page *et al.* (1982) were able to reproduce malabsorption syndrome using reoviruses recovered from clinically affected broilers. Reovirus was recovered from broilers in Australia with runting syndrome (Pass *et al.* 1982) although the widespread occurrence of reoviruses in both normal and diseased chickens indicated that the isolation of reoviruses from diseased chickens did not necessarily imply an aetiological relationship of reovirus with disease (Robertson *et al.* 1984). Reoviruses are not currently considered to be the most important pathogen associated with this disease syndrome (Goodwin *et al.* 1993; Jones 2008)

Reoviruses have been recovered from an ornamental duck (Gough *et al.* 1988), mallard ducks (McFerran *et al.* 1976), and commercial ducks (Jones and Guneratne 1984). Reoviruses which have been shown to be serologically distinct from chicken reoviruses have been recovered from Muscovy ducks (Heffels-Redmann *et al.* 1992).

Muscovy duck reovirus (DRV) has been identified as the cause of a disease of Muscovy ducks characterised by necrotic foci in the liver, spleen and kidneys. Muscovy ducks are affected between 10 days and 6 weeks of age and present with malaise and diarrhoea. Up to 20% mortality has been reported and individuals that recover are markedly stunted. Transmission studies have shown that goslings, Pekin ducks and chickens are resistant to infection with this DRV when inoculated subcutaneously (Malkinson *et al.* 1981). Sequence analysis suggests that DRV should be considered a distinct genogroup from other avian reoviruses (Kuntz-Simon *et al.* 2002a; Zhang *et al.* 2006) and recombinant vaccines have been developed which have been shown to protect against DRV (Kuntz-Simon *et al.* 2002b).

Oral transmission of DRV has not been demonstrated and transmission studies with this virus have only used either subcutaneous (Marius-Jestin *et al.* 1988) or intramuscular inoculation (Malkinson *et al.* 1981; Heffels-Redmann *et al.* 1992; Feng Qiang *et al.* 2008; Quan Xi *et al.* 2009; Quan Xi *et al.* 2010).

16.1.5. Hazard identification conclusion

Reoviruses are likely to be involved in the aetiology of viral arthritis/tenosynovitis in chickens although this disease is recognised in New Zealand and there is a high seroprevalence to reoviruses in broilers and breeder birds. Apart from tenosynovitis in chickens, the relationship between reoviruses and disease in chickens remains unclear (Jones 2008). Reoviruses are not identified as a potential hazard in imported chicken meat.

DRV is recognised as a primary pathogen in Muscovy ducks and there is no evidence to suggest that this disease is present in New Zealand. For the purposes of this risk analysis, DRV is identified as a potential hazard in duck meat.

16.2. RISK ASSESSMENT

16.2.1. Entry assessment

Reovirus infections of chickens are known to have a tropism mainly for heart and liver tissues and, to a lesser extent, spleen and intestine (Mustaffa-Babjee *et al.* 1973; Gouvea and Schnitzer 1982). Following infection with DRV, histological lesions have been described in the heart, liver, spleen, pancreas, tendons and kidney (Malkinson *et al.* 1981; Marius-Jestin *et al.* 1988) as well as transient lesions in the bursa of Fabricius and thymus (Marius-Jestin *et al.* 1988). Gross lesions noted following infection include pericarditis, marbled spleen, and an enlarged friable liver with focal necrosis (Marius-Jestin *et al.* 1988). No studies have investigated the presence of DRV in the musculature of infected individuals.

In regions with intensive Muscovy duck production, clinical signs of DRV infection first appear in the second week after hatching (Heffels-Redmann *et al.* 1992) and may persist until 6 weeks of age (Malkinson *et al.* 1981), with disease usually apparent between 2 and 4 weeks (Le Gall-Reculé *et al.* 1999; Kuntz-Simon *et al.* 2002b).

Following experimental inoculation of sixteen-day-old ducks with DRV, virus was no longer detected in cloacal swabs after 5 days post-infection (Heffels-Redmann *et al.* 1992). The duration of persistence of DRV in the tissues of an infected bird has not been investigated. However, Menendez *et al.* (1975b) studied the persistence of avian reovirus in chickens and demonstrated widespread infection 4 days after inoculation, which largely subsided after 14 days and no infection could be detected after 30 days. In a similar study, active infection, as judged by reovirus isolation from cloacal swabs, was over by the seventeenth day post-inoculation (Menendez *et al.* 1975a).

Muscovy ducks and their crossbreeds reach slaughter weight between 10 and 12 weeks old (Wawro *et al.* 2004). Given the age when Muscovy ducks become infected with DRV, and the likely duration of infectivity in an infected individual, it is unlikely that the virus will be present in imported duck meat.

Furthermore, the available studies of DRV and comparative studies of other avian reoviruses suggest that infectivity is concentrated mainly in the liver and heart and also, to a lesser extent, in the spleen and intestine. These tissues will not be present in the imported commodity.

Reflecting the above, the likelihood of entry is assessed to be negligible.

16.2.2. Risk estimation

Since the entry assessment is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and avian reoviruses are not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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17. Turkey coronavirus enteritis

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

Order: *Nidovirales*, Family: *Coronaviridae*. Coronaviruses are divided into three major antigenic groups with *Turkey Coronavirus* (TCV) and *Infectious Bronchitis Virus* (IBV) in group 3 (Guy 2008).

Cavanagh (2005) has questioned whether or not IBV of chickens, TCV, and pheasant coronavirus are three distinct viral species, or one species with different strains causing disease in one host species and not another.

17.1.2. OIE list

Not listed.

17.1.3. New Zealand status

Coronavirus has not been recorded in the New Zealand turkey population. It is not uncommon for commercial poultry in New Zealand to be seropositive to infectious bronchitis and vaccination is not uncommon (Poland 2005). Clinical disease associated with seroconversion to IBV has been described (Howell 1985).

17.1.4. Epidemiology

Turkeys are believed to be the only natural host for TCV (Guy 2008). Oral inoculation of one-day-old and four-day-old SPF chickens with 100 EID_{50} of TCV did not result in any clinical signs or gross lesions, although virus was detected in the gut content of the experimentally infected birds (Ismail *et al.* 2003). The authors of this study suggested that TCV may be able to replicate in the intestines of chickens. There are no reports of natural infections of chickens or ducks with TCV.

Turkey coronavirus was first described in the USA where it has been associated with mortality in young poults and depressed meat and egg production in older birds. The virus has also been recognised in the UK (Cavanagh *et al.* 2001), Brazil, Italy (Cavanagh 2005), Canada (Dea *et al.* 1986), and Australia (Nagaraja and Pomeroy 1997).

Replication of turkey coronavirus occurs primarily in enterocytes in the jejunum and ileum, and in the epithelium of the bursa of Fabricius (Guy 2008).

Viral transmission occurs via faeces. Experimental attempts to infect turkeys with homogenates of liver, heart, spleen, kidney, and pancreas of infected turkeys have been unsuccessful (Guy 2008). Studies in the UK have detected turkey coronavirus by RT-PCR in samples of caecal tonsil, caecal content, and bursa of Fabricius although no virus was detected in samples of spleen, kidney, or thymus (Culver *et al.* 2006). Earlier experimental infection studies demonstrated that the bursa of Fabricius is a more concentrated source of TCV than intestinal content (Naqi *et al.* 1972).

Following infection, TCV can be detected in intestinal content for up to 42 days by virus isolation and 49 days by RT-PCR, suggesting that prolonged shedding of virus occurs after recovery from clinical disease (Breslin *et al.* 2000).

Calibeo-Hayes *et al.* (2003) demonstrated that domestic houseflies (*Musca domestica*) fed on an inoculum containing $5x10^6$ EID_{50} TCV/ml were able to transmit infection to poults, with the housefly acting as a mechanical vector. Watson *et al.* (2000) investigated the role of mealworms (*Alphitobius diaperinus*) in TCV transmission and concluded that they may well be involved in the transmission of disease within a turkey house during an active outbreak but they were less likely to transmit disease from field soils to a turkey house after the land application of litter.

17.1.5. Hazard identification conclusion

There are no reports of natural infection of either chickens or ducks with TCV. Experimental infection of chickens with TCV does not result in either clinical signs of disease or gross lesions. Although there has been speculation that TCV may be able to replicate in the intestines of chickens following experimental infection, there is no evidence that infectivity would be found elsewhere in the carcase of an infected individual.

TCV is not identified as a potential hazard in the commodity.

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18. Astrovirus

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

Family: *Astroviridae*, Genus: *Avastrovirus*. Members of the Avastrovirus genus infect avian species.

Avian Nephritis Virus (ANV) is an astrovirus of chickens (Imada *et al.* 2000) considered to be present in New Zealand and requires no further consideration here (Howell 1992).

Duck Hepatitis Virus type 2 (DVH2) is a member of the Astrovirus genus (Gough *et al.* 1985; Fu *et al.* 2009) and is considered in Chapter 20.

Chicken Astroviruses (CAstV) have been described which are serologically distinct from ANV and DVH2 (Baxendale and Mebatsion 2004).

18.1.2. OIE list

Not listed.

18.1.3. New Zealand status

Duck hepatitis virus is classified as a notifiable organism (Tana *et al.* 2011).

18.1.4. Epidemiology

CAstV infection of one-day-old chicks leads to mild diarrhoea and distension of the small intestine. High titres of virus can be recovered from the intestines of infected chicks but little or no CAstV can be found in tissues other than the intestine. Retrospective serological surveys have demonstrated exposure to CAstV in chicken flocks in the United Kingdom, the Netherlands, Spain, and the United States. No correlation was found between the presence of antibodies to CAstV in a chicken flock and a history of uneven growth (Baxendale and Mebatsion 2004).

18.1.5. Hazard identification conclusion

The pathogenic role of CAstV is unclear although infectivity is associated with the intestine and is therefore unlikely to be present in the imported commodity. Astroviruses are not identified as a potential hazard.

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19. Avian entero-like viruses

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

A group of nine genera within the Family *Picornaviridae, Enterovirus-Like Viruses* (ELVs) (Guy *et al.* 2008).

Duck Hepatitis Virus types 1 and 3 are considered in Chapter 20.

19.1.2. OIE list

Not listed.

19.1.3. New Zealand status

There are no reports of ELVs in New Zealand. However, it is probable that ELVs have a worldwide distribution (Guy *et al.* 2008). Losses due to "runting and stunting" occur periodically in the New Zealand broiler industry (Howell 1992).

19.1.4. Epidemiology

Natural ELV infections have been described in chickens, turkeys, guinea fowl, partridges, pheasants, ostriches, and psittacine species (Guy *et al.* 2008). No reports could be found of ELV infections in ducks.

The pathogenic role of ELVs requires further clarification although diarrhoea, decreased feed efficiency, and uneven growth have been reported in cases of natural infection where ELVs are usually seen as a component of mixed infections (Guy *et al.* 2008). McNulty *et al.* (1984) suggested a causal relationship between ELVs and runting/malabsorption syndrome in broilers characterised by abnormal faeces, reduced weight gain and poor feathering. Bacteria-free suspensions of intestinal content from broilers showing signs of stunting, lameness and sporadic diarrhoea were only partially successful at reproducing disease when inoculated into control birds (Chooi and Chulan 1985). However, Decaesstecker *et al.* (1986) were able to reproduce stunted growth and mucoid diarrhoea in day-old SPF chicks using ELVs purified from the intestines of chickens clinically affected by 'runting syndrome'. A variable degree of growth retardation is seen when day-old chicks are experimentally infected with ELVs (McNulty *et al.* 1990; McNeilly *et al.* 1994).

ELVs replicate within intestinal enterocytes although the location, severity and duration of intestinal lesions associated with this vary between viruses (Decaesstecker *et al.* 1989). Histologically, ELV replication in the intestines is accompanied by infiltration of the lamina propria with lymphoid cells, mesenchymal cells and macrophages (Frazier and Reece 1990). Clinical signs develop 3 days after infection and resolve after a further 3 to 5 days. Gross and histopathological lesions are restricted to the intestines. No gross or histopathological lesions are seen in the liver, kidney, bursa, heart, or spleen. The jejunum and ileum are the major sites of virus localisation and replication, and viral antigen is most abundant in the enterocytes situated just above the crypt opening (Swayne *et al.* 1990; Hayhow and Saif 1993; Hayhow *et al.* 1993).

Horizontal transmission through ingestion of faeces is considered most likely (Guy *et al.* 2008) although Spackman *et al.* (1984) identified enteroviruses in the meconium of dead-in-shell chicks, suggesting the possibility of vertical transmission. Despins *et al.* (1994) were unable to determine the role of darkling beetle larvae (*Alphitobius diaperinus*) as mechanical vectors of ELVs as their study poults were found to be already infected with ELVs when obtained from the hatchery.

19.1.5. Hazard identification conclusion

The pathogenic role of ELVs is not completely understood. However, infectivity is restricted to the intestinal tract and there is no evidence for infectivity elsewhere in the carcase. Reflecting this, ELVs are not identified as a potential hazard.

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20. Duck hepatitis virus

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

Duck hepatitis is caused by any one of three different viruses (Duck hepatitis virus (DHV) types 1, 2, and 3).

DHV-1 has been classified as an *Avihepatovirus*, a new genus in the *Picornaviridae* family (Tseng *et al.* 2007). Three distinct genotypes have been identified, designated *Duck Hepatitis A Virus* (DHAV) types 1, 2, and 3 (Wang *et al.* 2008).

DHV-2 has been classified as an *Avastrovirus* species in the *Astroviridae* family (Gough *et al.* 1985) and has been renamed *Duck Astrovirus* type 1 (DAstV-1).

DHV-3 has also been classified as an *Avastrovirus* species (Kim *et al.* 2008; Todd *et al.* 2009) and has been renamed *Duck Astrovirus* type 2 (DAstV-2). Sequence analysis has shown that DAstV-1 (DHV-2) and DAstV-2 (DHV-3) are distinct isolates and may represent different species (Todd *et al.* 2009).

These duck hepatitis viruses are distinct from *Duck Hepatitis B Virus* (DHBV), an *Avihepadnavirus*, which does not cause significant clinical disease in ducks (Yang *et al.* 2008).

20.1.2. OIE list

Duck virus hepatitis is an OIE-listed disease.

20.1.3. New Zealand status

Duck hepatitis virus is listed as a notifiable organism (Tana *et al.* 2011).

20.1.4. Epidemiology

Duck hepatitis (DH) has been described in most duck-growing areas of the world although the viruses vary in their distributions. DHAV-1 is distributed worldwide and disease is usually endemic where it is found (Gough and McNulty 2008a). DHAV-2 and DHAV-3 have only been identified in Taiwan, South Korea and China (Kim *et al.* 2009). DAstV-1 (DHV-2) has only been reported in the UK prior to 1985, and DAstV-2 (DHV-3) has only been reported in the USA (Woolcock 2008).

Duck hepatitis is an acute, highly fatal, rapidly spreading disease of young ducklings characterised primarily by hepatitis (Woolcock 2008). The epidemiology of DHV-1, -2, and -3 are similar and the viruses are considered as one unless explicitly stated.

DHV is extremely contagious but morbidity and mortality is variable and decrease with age. Broods less than 1 week of age have 100% morbidity and mortalities as high as 95%. However, older ducklings have low or negligible morbidity and mortality although an inadequate diet increases susceptibility (Woolcock 2008). Ducklings begin to develop age resistance at about 3 weeks and disease is rarely seen in ducklings over 4 weeks of age. Age resistance is essentially complete from 5-6 weeks and mature ducks are refractory to infection

(Asplin 1958; Rispens 1969; Farmer *et al.* 1987). Exceptions to the contagiousness of DHV have been observed and Woolcock (2008) describes an outbreak of DHV where in one pen 65% of the ducks died, while in an adjoining pen mortality was negligible. Hwang (1974) also demonstrated the absence of disease (and mortality) in newly hatched Muscovy ducklings following both indirect contact and intramuscular inoculation with DHV-1 yet was able to isolate the virus from the liver 7 days after exposure.

Ducks are the only natural host of the DH viruses. Phylogenetic analyses demonstrate a close genetic relationship between DAstV and turkey astrovirus type 2 (Fu *et al.* 2009; Pantin-Jackwood *et al.* 2011). However, there are no reports of virus transmission between ducks and turkeys. Experimental infection of other species has achieved mixed results. Some studies failed to infect chickens experimentally (Reuss 1959; Schoop 1959; Hwang 1974), others reported that chicks can become infected and transmit infection without developing clinical signs (Asplin 1970), whilst others show high mortalities (more than 50%) in a variety of non-chicken avian species following experimental inoculation (Hwang 1974). Laboratory animals are refractory to infection with DHV (Reuss 1959; Woolcock 2008) but it is noted that one study reportedly demonstrated infection and subsequent excretion of DHV-1 from brown rats (Woolcock 2008).

There are no reports of natural infections in species other than ducks and field observations indicate that chickens are resistant to natural infection (Hwang 1974; Woolcock 2008). While concerns are repeatedly raised in the literature regarding the potential for wild birds to act as reservoirs of infection and incriminating them as vectors in outbreaks of DHV (Gough *et al.* 1985), they are not supported by serological evidence from several large scale studies which failed to demonstrate DHV in any free-flying wildfowl of multiple species (Asplin 1970; Woolcock 2008).

The DH viruses are not known to have any human health significance (Woolcock 2008).

Natural transmission of DHV occurs by the faecal-oral route (Tripathy and Hanson 1986; MingShu *et al.* 1997) and recovered ducks may excrete DHV in their faeces for up to 8 weeks post infection (Woolcock 2008). Spread occurs horizontally by contact with infected ducklings or fomites (Gough and McNulty 2008a; 2008b). Vertical transmission is not thought to occur (Gough and McNulty 2008a).

The DH viruses are highly resistant to physical and chemical conditions and are extremely stable in the environment (Koci and Schultz-Cherry 2002). The viruses are capable of surviving for several months under normal environmental conditions (Gough and McNulty 2008a) and studies report survival times of 60 minutes at 50°C, 21 days at 37°C, 2 years at 4°C, and 9 years at -20°C (Woolcock 2008). DHV was inactivated after 30min at 62°C (Woolcock 2008).

The DH viruses produce similar clinical signs and pathological changes but DHV-1 causes the most severe disease. The onset and spread of disease is very rapid and ducklings develop opisthotonus and rapidly die in a typical “arched-backward” position. Mortalities of 95%, 50% and 30% in naive flocks following infection with DHV-1, -2 and -3 respectively are not uncommon (Rispens 1969; Woolcock 2008). Following natural infection, the main lesions are seen in the liver; which is enlarged and contains petechial and ecchymotic haemorrhages; although the spleen is sometimes enlarged and mottled and fatty kidneys have been described (Farmer *et al.* 1987; Woolcock 2008).

The pathogenesis of DHV infection of ducks is poorly understood. DHV has been detected in multiple organs following experimental inoculation; including liver, lung, heart, spleen, kidney, brain, muscle and pancreas (MingShu *et al.* 1997; Hwang and Dougherty 1974; Guerin *et al.* 2005, 2007) and it has been shown to replicate in liver and kidney cells (Haider and Calnek 1979). DHV has been successfully transmitted to ducklings via parenteral and aerosol inoculation with the above tissues (Hwang and Dougherty 1974) and by oral inoculation with pancreas extracts (Guerin *et al.* 2005). In contrast, Adamiker (1969, 1970) failed to demonstrate the presence of DHV in the spleen and muscle despite histological changes of these tissues.

Preliminary diagnosis can be made from the characteristic clinical and pathological signs but distinguishing between the different DHVs requires methods such as reverse transcriptase-polymerase chain reaction (RT-PCR) (Gough and McNulty 2008a).

Control of the disease is achieved via attenuated live virus vaccines of breeder ducks and day-old ducklings and treatment with hyperimmune serum (Woolcock 1991).

20.1.5. Hazard identification conclusion

There are no reports of natural infection of chickens with DHV and DHV is not identified as a potential hazard in chicken meat, chicken meat products or whole chicken carcases.

The pathogenesis of DHV infection of ducks is poorly understood. There are conflicting reports in the literature on the tissue distribution of DHV. However, the virus is considered to be pantropic in nature (Hwang and Dougherty 1974) and many tissues have been shown to transmit infection by a number of routes. Therefore DHV is identified as a potential hazard in duck meat, duck meat products and whole duck carcases.

20.2. RISK ASSESSMENT

20.2.1. Entry assessment

DHV is an acute disease of young ducklings and birds of slaughter age are generally resistant to infection. Flocks infected with DHV are very likely to show evidence of disease and accordingly should not be slaughtered for human consumption. However, infection of Muscovy ducklings is often subclinical, which would not be detected. Recovered birds may excrete virus in their faeces for up to 8 weeks without clinical signs and these birds may be of slaughter age. Reversion to virulence has been demonstrated with attenuated live virus passages in ducklings (Woolcock and Crighton 1979) and vaccinated birds may be a source of DHV infection.

Considering the above, the likelihood of entry in imported duck meat, duck meat products and whole duck carcases is assessed to be low.

20.2.2. Exposure assessment

Backyard poultry

DHV is inactivated after 30 minutes at 62°C. Therefore, the likelihood of backyard flocks being exposed to DHV from discarded cooked duck meat, duck meat products or whole duck carcases is assessed to be negligible.

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

DHV is highly contagious, extremely stable in the environment, and may survive for several months under usual environmental conditions. Natural transmission of DHV occurs by the faecal-oral route (Tripathy and Hanson 1986; MingShu *et al.* 1997). Therefore the likelihood of ducklings in a backyard flock being infected with DHV through exposure to raw duck meat, duck meat products or whole duck carcases is assessed to be low.

Wild birds

Ducks are the only natural host of DHV. Mallards are by far the predominant wild duck species in New Zealand (Hemsley 1996; Wood and Garden 2010). Female mallards are secretive while rearing their young and ducklings spend most of their time at well-concealed sites, particularly tall, dense vegetation (Shah *et al.* 2008). The average age at which mallard ducklings begin to fly ranges from 54-80 days (Greenwood 1974; Shah *et al.* 2008) which is after the development of age immunity to DHV (35-42 days) (Asplin 1958; Rispens 1969; Farmer *et al.* 1987). The likelihood of free-living ducks being infected with DHV through exposure to an infected backyard duck flock or to raw duck meat, duck meat products and entire duck carcases is assessed to be very low.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked waste food from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (MAF 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

Standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007). However, the biosecurity standards on commercial duck farms in New Zealand would be unlikely to prevent contact with wild ducks (Mulqueen 2012).

Therefore the likelihood of commercial ducks being exposed to DHV is assessed to be low.

Exposure assessment conclusion

In conclusion, exposure assessment for backyard poultry flocks, wild birds, and commercial poultry is assessed to be non-negligible.

20.2.3. Consequence assessment

Duck hepatitis is an acute, highly fatal, rapidly spreading disease of young ducklings characterised by hepatitis. Infection of backyard duck flocks, wild ducks, or commercial duck flocks with DHV would be associated with non-negligible consequences.

Ducks are the only natural host of DHV so there would be negligible consequences for other commercial poultry species, free-living avian species, or human health.

20.2.4. Risk estimation

Since entry, exposure, and consequence assessments for DHV are non-negligible, the risk estimation is non-negligible and DHV is classified as a risk in the commodity. Therefore risk management measures can be justified.

20.3. RISK MANAGEMENT

20.3.1. Options

DHV is pantropic and has been isolated in multiple tissues and also transmitted by aerosol, parenteral and oral inoculation of susceptible ducklings with a variety of tissues.

Serologic tests have not been useful in diagnosing DHV due to its acute presentation and variable strain (Woolcock 2008).

DHV is inactivated after heating at 62°C for 30 minutes, therefore cooking may be appropriate to manage the risk of introducing the virus in the commodity.

Article 10.6.2 of the *Code* describes recommendations to manage the risk of DVH associated with the international trade in live ducks. These measures could be adapted to effectively manage the risk associated with imported duck meat.

Option 1

Imported duck meat, duck meat products and whole duck carcases could be cooked to a core temperature of at least 62°C for no less than 30 minutes.

Option 2

Duck meat, duck meat products and whole duck carcases could be imported from establishments and/or hatcheries where DHV has not been recognised.

Option 3

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that duck meat, duck meat products and whole duck carcases for import have been derived from birds that:

1. showed no clinical sign of DVH on the day of slaughter;

2. come from establishments which are recognised as being free from DVH;

3. have not been vaccinated against DVH; or

4. were vaccinated against DVH (the nature of the vaccine used and the date of vaccination should also be stated in the certificate).

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21. Duck virus enteritis virus

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

Family; *Herpesviridae*. Subfamily; *Alphaherpesvirinae*.

Species: *Anatid Herpesvirus 1* (also known as *Duck Plague Herpesvirus*) (Fauquet *et al.* 2005).

21.1.2. OIE list

Not listed.

21.1.3. New Zealand status

Duck virus enteritis (DVE) is on the list of notifiable organisms (Tana *et al.* 2011).

21.1.4. Epidemiology

Duck virus enteritis is an acute, sometimes chronic, contagious disease of waterfowl (order Anseriformes) usually associated with high morbidity and mortality. DVE has been reported in many countries in Europe, Asia and North America (Gough 2008) and may also be referred to as duck plague, anatid herpes, eendenpest, entenpest and peste du canard (OIE 2008).

Differences in virulence between viral strains have been reported but no molecular basis for these differences has been identified. A recent report from Vietnam suggests the presence of two DEV subtypes in that country (Gough 2008; Sandhu and Metwally 2008).

Naturally occurring outbreaks of DVE have occurred in a wide variety of domestic and wild ducks, geese, and swans of all ages (Wobeser and Docherty 1987; Gough 2008; Sandhu and Metwally 2008). Natural infection has not been reported in other avian species or mammals (OIE 2008) and transmission studies in a variety of species other than waterfowl have failed to cause infection or an antibody response (Gough 2008). There appears to be a varying degree of susceptibility to infection among the different waterfowl species (Montgomery *et al.* 1981). Muscovy ducks and teal appear to be highly susceptible, whereas mallards appear to be quite resistant and may be a possible natural reservoir of infection (Montgomery *et al.* 1981; Wobeser 1987; Campagnolo *et al.* 2001; Converse and Kidd 2001; Sandhu and Metwally 2008).

In susceptible flocks the first signs are often sudden, high and persistent mortality with a significant drop in egg production (Gough 2008; OIE 2008). In chronically infected partially immune flocks only occasional deaths occur (OIE 2008). Affected birds are listless and ataxic, with pasted eyelids, nasal discharge, anorexia, polydypsia and profuse green diarrhoea (Richter and Horzinek 1993; Gough 2008; Chambal *et al.* 2009). Adult male waterfowl may show prolapse of the phallus and young birds may show congested beaks and bloodstained vents (Gough 2008). The clinical signs may last for several weeks. The morbidity and mortality in a flock may range from 5% to 100% depending on the virulence of the virus and the age and immunologic status of the birds (Campagnolo *et al.* 2001; Sandhu and Metwally 2008). DVE is most likely to affect mature birds (Richter and Horzinek 1993).

Infection leads to increased vascular permeability and subsequent extensive haemorrhage and necrosis. Disease presentation differs between age groups and species. In mature ducks, pathology in the digestive and reproductive organs predominates and there is free blood in body cavities. In ducklings, tissue haemorrhages are less pronounced and lymphoid lesions and subcutaneous oedema of the neck are prominent (Richter and Horzinek 1993; Gough 2008; Sandhu and Metwally 2008). Species of teal (of all ages) show minimal gross lesions (Wobeser 1987) as do all birds which die very rapidly (Gough 2008).

Experimentally virus transmission can occur vertically via the egg and horizontally via the oral, intranasal, intravenous, intraperitoneal, intramuscular and cloacal routes (Sandhu and Metwally 2008). Transmission by bloodsucking arthropods is also considered possible (Sandhu and Metwally 2008). It is most likely that natural infection occurs through the oral or cloacal route, either by direct or indirect contact (Richter and Horzinek 1993; Gough 2008; Sandhu and Metwally 2008).

The initial site of virus replication is the mucosa of the digestive tract, particularly the oesophagus and cloaca, and virus then spreads to the bursa of Fabricius, thymus, spleen, liver and then to other organs (Islam and Khan 1995; Shawky and Schat 2002; Yue *et al.* 2007; Gough 2008; Qi *et al.* 2008; Sandhu and Metwally 2008; Shen *et al.* 2010). The epithelial cells and lymphocytes of these organs are the principal sites of virus replication. The trigeminal ganglia and peripheral blood lymphocytes are the major latency sites of the virus (Shawky and Schat 2002; Yue *et al.* 2007). There are no reports that the virus or viral antigen can be isolated from muscle (Cheng *et al.* 2008).

Virus has also been demonstrated in the eggs of persistently infected ducks, and ducklings from these eggs will routinely shed virus in their faeces (Richter and Horzinek 1993). It is difficult to assess the significance of these carriers as a source of virus for horizontal transmission. However, vertical transmission can itself lower fertility and hatchability in domestic and wild waterfowl and potentially perpetuate and disseminate the virus (Burgess and Yuill 1981).

There is evidence that birds that survive infection may become silent carriers and periodically shed the virus both orally and through excreta for the remainder of their lives (Richter and Horzinek 1993; Campagnolo *et al.* 2001; Gough 2008). Many healthy waterfowl have been found to be carriers of DVE (Burgess *et al.* 1979; Ziedler and Hlinak 1992) and isolated cases in wild anatids, including mallards, have been reported (Wobeser and Docherty 1987). Natural virus reservoirs in non-migratory or migratory waterfowl are unknown (Richter and Horzinek 1993; Converse and Kidd 2001; Sandhu and Metwally 2008). Despite this, carrier birds have been frequently blamed for precipitating DVE outbreaks in domestic and migrating waterfowl populations (Campagnolo *et al.* 2001; Sandhu and Metwally 2008).

Serious outbreaks with high mortality in migratory waterfowl have been reported (Converse and Kidd 2001; Sandhu and Metwally 2008). The most devastating outbreak was responsible for the deaths of over 43,000 wild mallard ducks and several hundred Canada geese. Another outbreak resulted in deaths of over 1,400 wild waterfowl of different species (Goldberg *et al.* 1990; Campagnolo *et al.* 2001). Outbreaks in zoos and game farm flocks have also been reported (Sandhu and Metwally 2008). Outbreaks in commercial ducks and geese are usually associated with contact with wild waterfowl (Gough 2008). Water is particularly important for the transmission of DVE and outbreaks in domestic ducks are frequently associated with aquatic environments cohabited by wild waterfowl (Sandhu and Metwally 2008).

The virus is heat-labile and similar to other herpesviruses in its sensitivity to chemicals, extremes of pH and disinfectants (Richter and Horzinek 1993; Fauquet *et al.* 2005; Gough 2008). At room temperature (22°C), the virus can survive for 30 days, but is inactivated after heating for 10 minutes at 56°C or 90-120 minutes at 50°C (Sandhu and Metwally 2008).

Maternal immunity in ducklings declines rapidly (Richter and Horzinek 1993; Sandhu and Metwally 2008). Field observations suggest that recovered birds are immune to re-infection. However, experimental studies showed that superinfection of persistently infected birds resulted in death (Sandhu and Metwally 2008).

A live attenuated virus vaccine is available but it is not known whether live DVE vaccines can induce latency, particularly if transmission to wildfowl occurs, with subsequent reactivation of vaccinal virus. Inactivated vaccines for DVE have been developed but are not available commercially (Gough 2008).

21.1.5. Hazard identification conclusion

Natural infection has not been reported in chickens and DVE is not identified as a potential hazard in chicken meat, chicken meat products or whole chicken carcases.

There are no reports that the virus has been isolated in the muscle and one study failed to find virus or viral antigen in muscle of infected birds (Cheng *et al.* 2008). Duck virus enteritis is not identified as a potential hazard in duck meat, or duck meat products.

The virus may be isolated from the liver, spleen and kidneys (Islam and Khan 1995; OIE 2008). The kidneys are not removed from duck carcases during processing (Veerkamp 2011) and therefore DVE is identified as a potential hazard in whole duck carcases.

21.2. RISK ASSESSMENT

21.2.1. Entry assessment

Flocks infected with DVE are very likely to show evidence of disease and accordingly should not be slaughtered for human consumption. However subclinically infected birds, latent carriers, or birds with few gross lesions may be overlooked at necropsy (Wobeser 1987). In these birds, fragments of infective tissues present in duck carcases after processing may be a source of virus.

Considering the above, the likelihood of entry of DVE in imported whole duck carcases is assessed to be non-negligible.

21.2.2. Exposure assessment

According to Wobeser (1987), “the long term persistence of infection in carrier birds with periodic shedding of virus; transovarial transmission; ease of transmission by a variety of routes; and relative hardiness of the virus in surface water all suggest that infection could become widespread in nature”.

Backyard poultry

DVE is inactivated after 10 minutes at 56°C therefore the likelihood of backyard flocks being exposed to DVE in scraps from duck carcases following domestic cooking is assessed to be negligible.

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

It is most likely that natural infection occurs through the oral or cloacal route, either by direct or indirect contact (Richter and Horzinek 1993; Gough 2008; Sandhu and Metwally 2008). The oral dose of DVE sufficient to initiate infection is not known. However the large numbers of birds affected in outbreaks indicates that the virus is highly infectious. DVE is stable in the environment, and may survive for a month under usual environmental conditions. Therefore the likelihood of ducklings in a backyard flock being infected with DVE through exposure to scraps of raw duck carcases is assessed to be low.

Wild birds

Waterfowl of order Anseriformes are the only natural host of DEV. Wild mallards, like many other wild waterfowl, are recognised to be silent carriers and periodic shedders of the DVE virus (Campagnolo *et al.* 2001). Mallards are by far the predominant wild duck species in New Zealand (Hemsley 1996; Wood and Garden 2010).

The oral dose of DEV sufficient to initiate infection in wild birds is not known (Burgess *et al.* 1979) and an increased incidence of DVE in migratory waterfowl has not been observed following outbreaks in backyard flocks in the US (Montgomery *et al.* 1981). However the virus has a long outdoor survival time and the likelihood of free-living waterfowl being infected with DEV, either following exposure to an infected backyard duck flock, or through consumption of scraps from raw duck carcases, is assessed to be low.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked food waste from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (MAF 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

Standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007). However, the biosecurity standards on commercial duck farms in New Zealand would be unlikely to prevent contact with wild ducks (Mulqueen 2012).

Recovered birds may excrete virus in their faeces for many years without clinical signs. Experience in other countries is that outbreaks in commercial ducks and geese are usually associated with contact with wild waterfowl (Gough 2008).

Therefore the likelihood of commercial ducks being exposed to DVE is assessed to be non-negligible.

Exposure assessment conclusion

In conclusion, the likelihood of exposure of commercial ducks, backyard ducks and wild waterfowl is assessed to be non-negligible.

21.2.3. Consequence assessment

DVE produces significant economic losses due to mortality, condemnations, and decreased egg production (Sandhu and Metwally 2008).

Exposure of wild susceptible birds in open range farming systems could lead to widespread exposure and dissemination of the virus. A single outbreak of DVE has been responsible for the deaths of over 43,000 wild ducks and several hundred geese.

There is also potential for the virus to be transmitted to New Zealand's endemic wild duck species. New Zealand is home to several endangered waterfowl species including the blue duck, the brown teal, and the subantarctic teal. The blue duck is the only member of its genus and has no close relative anywhere in the world (DOC 2011). It is classified by the Department of Conservation as "Nationally Vulnerable" and by the International Union for the Conservation of Nature (IUCN) as "Endangered" (DOC 2011). The brown teal is New Zealand's rarest waterfowl species on the mainland (DOC 2011).

There are no consequences for humans or species other than waterfowl of order Anseriformes (ducks, geese and swans).

21.2.4. Risk estimation

Since entry, exposure, and consequence assessments for DVE are all non-negligible, the risk estimation is non-negligible and DVE is classified as a risk in whole duck carcases. Therefore risk management measures can be justified.

21.3. RISK MANAGEMENT

21.3.1. Options

Duck virus enteritis is assessed not to be a potential hazard in duck meat and duck meat products. One or a combination of the following options could be considered in order to effectively manage the risk in whole duck carcases.

Option 1

DEV is inactivated after heating at 56°C for 10 minutes, therefore cooking may be appropriate to manage the risk of introducing the virus in the commodity. Imported whole duck carcases could be cooked in accordance with the conditions required to manage the risk associated with NDV (see Section 6.3.1).

Option 2

Whole duck carcases could be imported from establishments where DVE has not been recognised. However it is not easy to certify individual birds or populations to be free from DVE (Burgess *et al.* 1979).

Option 3

Previous editions of the OIE *Code* contained recommendations for sanitary measures against DVE for the importation of live ducks. The following measures have been adapted from these earlier OIE recommendations and could be applied to the importation of whole duck carcases:

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that the birds:

1. showed no clinical sign of DVE on the day of (slaughter);

2. come from establishments which are regularly inspected by the Veterinary Authority;

3. come from establishments which are recognised as being free from DVE;

4. have not been vaccinated against DVE; or

5. were vaccinated against DVE (the nature of the vaccine used and the date of vaccination should also be stated in the certificate).

It should be noted that uncertainty exists over the potential for attenuated live DVE vaccine to undergo reversion to virulence. Vaccinated birds may therefore be a source of DVE infection.

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22. Derzsy's disease virus

22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

Family; *Parvoviridae*. Genus; *Dependovirus*. Species: *Goose Parvovirus* (GPV) (also called Derzsy's disease virus (DDV)) and *Duck Parvovirus* (also called Muscovy duck parvovirus (MDPV) or Barbarie duck parvovirus (BDPV)) (Tattersall *et al.* 2005).

Other waterfowl parvoviruses can be categorised into GPV-related or MDPV-related groups based on their nucleotide sequences (Poonia *et al.* 2006).

22.1.2. OIE list

Not listed.

22.1.3. New Zealand status

Waterfowl parvoviruses (GPV and MDPV) have never been recorded in New Zealand.

22.1.4. Epidemiology

Derzsy's disease is a highly contagious disease affecting young goslings and Muscovy ducklings and has been reported from major goose and Muscovy duck farming countries in many parts of the world, including Europe, Asia and the USA (Gough 2008). Derzsy's disease is known by many different names, including goose influenza, goose plague, goose hepatitis, viral enteritis of goslings, infectious myocarditis, and hepatonephritis-ascites (Gough 2008).

Domestic geese, wild geese and Muscovy ducks (including some hybrids) are the only species in which natural disease has been observed (Gough 2008). Other breeds of duck and domestic poultry appear refractory to infection and the disease has not been reported in other avian species or mammals (Gough 2008). The disease has no known human health significance (Irvine and Homes 2010).

GPV and MDPV differ in host ranges, antigenicity, and nucleotide sequences but the clinical signs and pathological lesions caused by both viruses are similar (Chang *et al.* 2000). GPV can cause severe disease is both goslings and Muscovy ducklings, whereas MDPV is not pathogenic for geese (Glavits *et al.* 2005; Gough 2008). Vaccinating Muscovy ducklings with GPV provides protection against both GPV and MDPV (Woolcock *et al.* 2000).

Disease is strictly age dependant, with birds building a progressive resistance to infection with age. 100% mortality may occur in goslings and Muscovy ducklings under 1 week old, with negligible losses in 4- to 5- week old birds (Gough 2008; Irvine and Holmes 2010).

Older birds do not usually show clinical signs but can develop a subclinical infection, and latency may establish (Gough 2008; Irvine *et al.* 2008; Irvine and Holmes 2010). However, some older birds may show nervous, locomotor and enteric signs (ascites and profuse white diarrhoea), abnormal feather development and stunting (Poonia *et al.* 2006; Gough 2008). Survivors may suffer growth retardation, loss of feathering and ascites (Gough 2008) and

infection in older ducks results in degenerative skeletal muscle myopathy (Woolcock *et al.* 2000; Glavits *et al.* 2005; Poonia *et al.* 2006).

Prominent pathological lesions in young birds include severe enteritis, hepatitis, myocarditis and atrophy of the lymphoid organs (bursa of Fabricius, thymus, spleen). In less acute cases, perihepatitis, pericarditis and ascites are frequent findings. In addition to the lesions observed in the geese, degenerative skeletal muscle myopathy, sciatic neuritis and polio-encephalomyelitis are also frequently observed in Muscovy ducks infected with either GPV or MDPV (Kisary 1993; Glavits *et al.* 2005; Poonia *et al.* 2006).

Birds are infected either vertically via the ova or the egg-shell, or horizontally through the faecal-oral route (Schettler 1971). After entering the digestive tract, the virus replicates in the intestinal wall and then enters the blood stream leading to viraemia and dissemination (Kisary 1993; Gough 2008). In the case of egg transmission, embryos either die during incubation or hatch out in an infected state with similar pathogenesis to that previously described (Derzsy 1967). The most serious GPV outbreaks occur following vertical transmission of the virus (Irvine and Holmes 2010).

The virus is distributed widely in the body and has been detected in the small intestine, bursa of Fabricius, heart, liver, pancreas, spleen, bone marrow, thymus, blood, cardiac muscle, skeletal muscle, tongue and brain of Muscovy ducklings from 2 days post-inoculation *per os* (Limn *et al.* 1996; Takehara *et al.* 1998; Yu *et al.* 2002; Xhu *et al.* 2010).

The replication of GPV or MDPV has not been investigated in detail (Gough 2008). However, virus replication requires cells which are actively synthesising DNA (Kisary 1993). The virus grows well in liver and kidney cells and has also been shown to replicate in the cells of spleen and bone marrow (Zhu *et al.* 2010). The liver, thyroids and pancreas are thought to be the main target organs of the virus (Schettler 1973; Yu *et al.* 2002).

Infected birds excrete large amounts of virus in their faeces resulting in a rapid spread of infection by direct and indirect contact (Gough 2008). Infected birds younger than 1 month of age shed the virus continuously, even if they do not develop clinical signs (Kisary 1993). Recovered birds and those which contract the infection after the age of 1 month may become virus carriers. The carrier state lasts virtually lifelong (Kisary 1993) and affected birds can transmit the virus vertically in their eggs and horizontally in their faeces (Gough 2008; Irvine *et al.* 2008; Irvine and Holmes 2010) although they do not shed the virus persistently (Gough 1987).

GPV and MDPV are extremely resistant to chemical and physical conditions and are not inactivated at temperatures of 65°C for 30 minutes or 56°C for 3 hours (Schettler 1973; Gough 2008). This means the potential for inanimate objects to spread infection is high (Kisary 1993; Poonia *et al.* 2006), which has been demonstrated in several field outbreaks (Irvine and Holmes 2010).

Prophylaxis is based on vaccination of neonatal birds or breeding flocks, and elimination of carrier birds. Live and inactivated GPV, MDPV, and bivalent vaccines are available in most countries where the disease is endemic (Gough 2008; Irvine and Holmes 2010).

22.1.5. Hazard identification conclusion

There are no reports of natural infection of chickens with GPV or MDPV and Derzsy's disease is not identified as a potential hazard in chicken meat, chicken meat products or whole chicken carcases.

Only Muscovy ducks and their hybrids are susceptible to infection with GPV or MDPV. Derzsy's disease is not identified as a potential hazard in duck meat, duck meat products or whole duck carcases from other duck species.

GPV and MDPV have been detected in almost every organ and tissue of the body including the bursa of Fabricius, bone marrow and skeletal muscle (Limn *et al.* 1996; Takehara *et al.* 1998; Yu *et al.* 2002; Xhu *et al.* 2010). Derzsy's disease is identified as a potential hazard in Muscovy duck meat, Muscovy duck meat products and whole Muscovy duck carcases.

22.2. RISK ASSESSMENT

22.2.1. Entry assessment

Derzsy's disease is an acute disease of young Muscovy ducklings and birds of slaughter age are generally resistant to infection. Infected flocks are very likely to show evidence of disease and accordingly should not be slaughtered for human consumption. Recovered birds of slaughter age may be latently infected and a source of virus.

Considering the above, the likelihood of entry in imported Muscovy duck meat, duck meat products and whole duck carcases is assessed to be low.

22.2.2. Exposure assessment

Derzsy's disease is highly virulent and contagious and very low doses of virus are able to induce pronounced pathogenic effects (Jestin *et al.* 1991). Infected birds excrete large amounts of virus in their faeces resulting in rapid spread of infection by direct and indirect contact. Birds can be horizontally infected through the faecal-oral route (Schettler 1971). Additionally GPV and MDPV are extremely resistant to environmental conditions and inanimate objects can spread infection widely.

Backyard poultry

Derzsy's disease may withstand domestic cooking temperatures. The likelihood of Muscovy ducklings in a backyard flock being infected with Derzsy's disease through exposure to raw or cooked Muscovy duck meat, duck meat products or whole duck carcases is assessed to be non-negligible.

Wild birds

Muscovy ducks and geese are the only natural hosts of Derzsy's disease. Mallards are by far the predominant wild duck species in New Zealand but there are large numbers of wild geese which would be susceptible to infection. The likelihood of free-living geese being infected with Derzsy's disease through exposure to an infected Muscovy duck in a backyard flock or to raw Muscovy duck meat, duck meat products and whole duck carcases is assessed to be very low.

Commercial poultry

The feeding of waste food (including poultry meat) from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (MAF 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

Standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007).

However, the transmission of infection from infected wild geese to domestic geese or Muscovy ducks is possible and the likelihood of commercial poultry being infected with Derzsy's disease through exposure to wild geese is assessed to be very low.

Exposure assessment conclusion

In conclusion, exposure assessment for backyard poultry flocks, wild birds and commercial poultry is considered to be non-negligible.

22.2.3. Consequence assessment

Derzsy's disease is an acute, highly fatal, rapidly spreading disease of young goslings and Muscovy ducklings. Infection of backyard Muscovy duck flocks, wild geese or commercial poultry would be associated with non-negligible consequences.

Domestic geese, wild geese and Muscovy ducks (including some hybrids) are the only species in which natural clinical disease has been observed (Gough 2008). There would be negligible consequences for other commercial poultry species, free-living avian species, or humans.

22.2.4. Risk estimation

Since entry, exposure, and consequence assessments for Derzsy's disease are non-negligible, the risk estimation is non-negligible and Derzsy's disease is classified as a risk in the meat derived from Muscovy ducks (or their hybrids). Therefore risk management measures can be justified.

22.3. RISK MANAGEMENT

22.3.1. Options

Waterfowl parvoviruses are pantropic and have been isolated from multiple tissues. Derzsy's disease is not inactivated at temperatures of 65°C for 30 min or 56°C for 3 hours so the cooking conditions required to inactivate NDV (see Section 6.3.1) cannot be relied upon to manage the risk of introducing this virus in the commodity.

Studies in other species have shown that parvoviruses have a marked resistance to thermal inactivation. Porcine parvovirus is reported to have survived exposure to 60°C for 15 hours (Sofer *et al.* 2003), whilst exposure to 80°C for 72 hours was shown to only bring about a 1.3D reduction in bovine parvovirus (Roberts and Hart 2000). Thermal processing of duck meat is therefore unlikely to provide an effective risk management option.

One or a combination of the following options could be considered in order to effectively manage the risk:

Option 1

Muscovy duck meat, duck meat products and whole duck carcases could be imported from establishments where Derzsy's disease has not been recognised.

Option 2

Muscovy duck meat, duck meat products and whole duck carcases could be imported according to the following recommendations:

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that the birds:

1. showed no clinical sign of Derzsy's disease on the day of slaughter;

2. come from establishments which are recognised as being free from Derzsy's disease;

3. have not been vaccinated against Derzsy's disease; or

4. were vaccinated against Derzsy's disease (the nature of the vaccine used and the date of vaccination should also be stated in the certificate).

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23. Arboviruses

23.1. HAZARD IDENTIFICATION

23.1.1. Aetiological agent

Arboviruses replicate in bloodsucking arthropods and are transmitted by bite to a vertebrate host. Over 100 arboviruses have been isolated from avian species or ornithophilic vectors but only five arboviruses are associated with disease in domestic poultry (Guy and Malkinson 2008):

- *Eastern Equine Encephalitis Virus* (EEEV)
- *Western Equine Encephalitis Virus* (WEEV)
- *Highlands J Virus* (HJV)
- *Israel Turkey Meningoencephalitis Virus* (ITV)
- *West Nile Virus* (WNV)

23.1.2. OIE list

West Nile fever and western equine encephalomyelitis (Western) are listed as notifiable to the OIE.

23.1.3. New Zealand status

EEEV, WEEV, and WNV are listed as notifiable organisms (Tana *et al.* 2011).

23.1.4. Epidemiology

The principal vector of EEEV is regarded as *Culiseta melanura* (Chamberlain 1958; Howard and Wallis 1974) although the virus has been identified in other mosquito hosts, mites, lice, simuliid flies, and *Culicoides* spp. (Guy and Malkinson 2008). Wild birds, especially small Passeriformes are the principal vertebrate host of EEEV (Kissling 1958; Williams *et al.* 1971). Natural infection of ducks with EEEV has been described (Dougherty and Price 1960) and, although chickens are susceptible to experimental infection (Tyzzer *et al.* 1938; Tyzzer and Sellards 1941), there are no reports of natural infection in this species (Guy and Malkinson 2008).

Disease is found mainly in eastern parts of North and South America, throughout Central America, and in the Caribbean. Outbreaks occur during late summer and early autumn when mosquito vectors increase (Guy and Malkinson 2008). Infection of ducks is associated with sudden onset posterior paresis and paralysis, with 2-60% mortality and histological lesions in the spinal cord and meninges. Experimental infection of chickens is associated with high rates of mortality, and neurological signs are only occasionally observed with death being associated with myocarditis (Tyzzer and Sellards 1941; Dougherty and Price 1960; Guy and Malkinson 2008).

The global distribution of WEEV is limited to Central America, South America, and western parts of the USA and Canada (Guy and Malkinson 2008). The mosquito vector of WEEV is

Culiseta tarsalis (Chamberlain 1958). WEEV is rarely associated with disease in avian species (Guy and Malkinson 2008) and there are no reports of natural infection of either ducks or chickens.

Natural HJV infection of either chicken or duck flocks has not been described.

ITV infection was first described in turkeys in Israel (Komarov and Kalmar 1960) and subsequently in South Africa (Barnard *et al.* 1980). The disease has not been described elsewhere (Guy and Malkinson 2008). ITV has only been reported in turkeys (Guy and Malkinson 2008) and chickens and ducks are refractory to infection (Komarov and Kalmar 1960).

Geese are the primary poultry species affected by WNV although experimental infection of chickens and ducks is described (Senne *et al.* 2000; Guy and Malkinson 2008). Chickens (and, to a much lesser extent, ducks) are used as sentinel birds to detect WNV as they are susceptible to infection but do not develop disease (Komar 2001; Komar *et al.* 2001; Buckley *et al.* 2006). Although experimentally-infected chickens develop viraemia between 5 and 7 days post infection, no clinical signs or mortality are seen in infected individuals (Senne *et al.* 2000).

23.1.5. Hazard identification conclusion

EEEV has been associated with disease in ducks. Natural infection of chickens and ducks with WNV are recognised although these are not associated with disease. There is no evidence of natural infection of chickens or ducks with other arboviruses.

Article 8.16.2 of the *Code* states that OIE members should not impose trade restrictions for WNV on fresh meat and meat products of poultry, regardless of the WNV status of the exporting country.

Furthermore, arboviruses replicate in bloodsucking arthropods and are transmitted by bite to a vertebrate host. The only way arthropod vectors can be infected is by sucking blood as they do not feed on meat and cannot be infected from meat. Therefore, arboviruses are not identified as a potential hazard in the commodity.

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24. Avian hepatitis E virus

24.1. HAZARD IDENTIFICATION

24.1.1. Aetiological agent

The primary causative agent of hepatitis-splenomegaly (HS) syndrome is a strain of hepatitis E virus (HEV), avian HEV (Payne *et al.* 1999; Haqshenas *et al.* 2001).

24.1.2. OIE list

Not listed.

24.1.3. New Zealand status

The agent of big liver and spleen disease (a synonym for HS syndrome) is listed as an unwanted exotic organism (MAF 2011).

24.1.4. Epidemiology

Chickens are the only known host for avian HEV (Meng *et al.* 2008). HS syndrome has been described in layer and broiler flocks in Canada (Ritchie and Riddell 1991; Tablante *et al.* 1994; Agunos *et al.* 2006), the United States (Huang *et al.* 2002), Australia (Clarke *et al.* 1990; Payne *et al.* 1993) and the United Kingdom (Todd *et al.* 1993).

Infected flocks show elevated mortality, reduced feed consumption, reduced weight gain and a drop in egg production (Ritchie and Riddell 1991; Huang *et al.* 2002; Agunos *et al.* 2006). At post-mortem, the liver may be enlarged and friable with multiple foci of necrosis and haemorrhage, whilst the spleen may be up to three times larger than normal with diffuse to multifocal white areas on both the capsular and cut surfaces (Ritchie and Riddell 1991; Agunos *et al.* 2006).

The natural route of HEV transmission is thought to be through the faecal-oral route (Meng *et al.* 2008) although transmission via the oronasal route in specific-pathogen-free (SPF) chickens has also been demonstrated (Billam *et al.* 2005). Following infection, large amounts of virus can be found in the faeces (Billam *et al.* 2005).

Following experimental infection of SPF chickens with HEV, gross lesions are limited to the liver (hepatomegaly and subcapsular haemorrhage). Histological lesions are also primarily associated with the liver (lymphocytic periphlebitis and hepatocellular necrotic foci) although lesions are also described in the spleen (mild lymphoid hyperplasia), thymus (mild cortical hypoplasia), kidneys (occasional mild lymphocytic interstitial nephritis), and lung (mild lymphocytic and heterophilic parabronchial interstitial inflammation) (Billam *et al.* 2005). Immunofluorescent studies have demonstrated that avian HEV antigen is primarily in the liver and spleen of experimentally-inoculated adult chickens (Clarke *et al.* 1990).

The gross lesions characteristic of HS syndrome (enlargement of the liver and spleen) are not consistently reproduced when SPF chickens are infected with avian HEV. It has therefore been suggested that avian HEV infection is an important factor, but not the sole factor, in the development of HS syndrome (Billam *et al.* 2005).

Studies that have examined the pathogenesis of HEV infections in swine have identified the liver, small intestine and lymph nodes as the main sites of viral replication, with much less pronounced replication in the large intestine, tonsil, spleen and kidney. No evidence of viral replication was detected in muscle tissue using a reverse transcriptase polymerase chain reaction methodology (Williams *et al.* 2001; Choi and Chae 2003)

24.1.5. Hazard identification conclusion

HS syndrome is only described in chickens, therefore HEV is not identified as a potential hazard in imported duck meat and duck meat products.

In chickens, HEV infectivity is located primarily in the liver and spleen which are removed from all commodities considered in this risk analysis. There is no evidence of HEV replication in muscle tissue. HEV is not identified as a potential hazard in imported chicken meat and chicken meat products.

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25. Marek's disease virus

25.1. HAZARD IDENTIFICATION

25.1.1. Aetiological agent

Subfamily: *Alphaherpesvirinae*, Genus: *Mardivirus*, Marek's disease virus (MDV). Three serotypes described, *Gallid herpesvirus 2* (serotype1), *Gallid herpesvirus 3* (serotype 2) and *Meleagrid herpesvirus 1* (serotype 3).

Serotype 1 strains are further divided into pathotypes referred to as mild (m)MDV, virulent (v)MDV, very virulent (vv)MDV, and very virulent plus (vv+)MDV (Witter 1997; Witter *et al.* 2005).

Additional non-oncogenic herpesviruses that have been recovered from turkeys (Kawamura *et al.* 1969; Witter *et al.* 1970) and chickens (Cho and Kenzy 1972) are also regarded as members of the MDV group, although these viruses are not considered to be pathogenic (Schat and Venugopal 2008).

25.1.2. OIE list

Not listed.

25.1.3. New Zealand status

Marek's disease is common in New Zealand poultry (McCausland 1972; Horner and James 1975).

No work has been done to assess the virulence of New Zealand isolates of MDV (Howell 1992). However, based on the clinical signs and pathology seen in association with Marek's disease, New Zealand is considered likely to be free from the more virulent strains of this virus (Stanislawek 2009).

Exotic strains of MDV are listed on the unwanted organisms register (MAF 2011).

25.1.4. Epidemiology

The disease was first described in 1907 and subsequent reports have described a gradual increase in the severity of clinical signs. mMDV is regarded as the classic form of the disease, and presents clinically as paralysis. vMDV became predominant in the 1960s and the vvMDV pathotype was described in late 1970s (Eidson *et al.* 1978). vvMDV and vv+MDV are now considered the dominant types (Schat and Venugopal 2008).

Fully infectious virus replicates in the epithelial cells in feather follicles (Calnek *et al.* 1970a) and virus associated with feathers and dander is infectious (Calnek and Hitchner 1969; Beasley *et al.* 1970; Calnek *et al.* 1970b). Naïve poultry are infected through exposure to infectious dust or dander directly or via aerosols, fomites, or personnel (Schat and Venugopal 2008). Following infection viral shedding begins after 2 to 3 weeks (Kenzy and Biggs 1967) and can continue indefinitely (Witter *et al.* 1971).

Gross lesions observed following infection include enlargement of peripheral nerves, which may show a loss of cross-striations and an oedematous appearance, and lymphomas in the

gonad, lung, heart, mesentery, kidney, liver, spleen, bursa, thymus, adrenal gland, pancreas, proventriculus, intestine, iris, skeletal muscle, and skin (Schat and Venugopal 2008).

Peripheral nerve dysfunction produces clinical signs of asymmetric progressive paresis, which may lead to complete spastic paralysis. Involvement of the vagus nerve can lead to crop dilation and/or gasping and ocular involvement can lead to blindness (Ficken *et al.* 1991; Schat and Venugopal 2008). High mortality is associated with vMDV strains (Witter *et al.* 1980).

Baxendale (1969) reported that ducks showed a very weak serological response following experimental challenge with MDV although inoculated ducks did not develop clinical disease. Powell and Rennie (1984) were unable to reisolate MDV from ducks inoculated with the virus, and these individuals also showed no clinical signs of infection. Chickens are by far the most important natural host for MDV and ducks are probably refractory to infection (Schat and Venugopal 2008).

25.1.5. Hazard identification conclusion

MDV is cell-associated in tumours and in all body organs except in the feather follicle where enveloped infectious virus is excreted and spread by direct contact or by the airbourne route (Purchase 1976). Virus could persist in the skin (in feather follicles) of imported chicken carcases but is unlikely to be present in meat (Payne and Venugopal 2000).

New Zealand is considered likely to be free from the more virulent strains of MDV, which are therefore assessed as a potential hazard in imported chicken meat.

The available evidence suggests that ducks are refractory to infection. MDV is not identified as a potential hazard in imported duck meat.

25.2. RISK ASSESSMENT

25.2.1. Entry assessment

Experimentally, transmission of MDV is most consistently effected by inoculation of blood, tumour suspensions or cell free virus into day old chicks. Chicken meat is considered not to be a likely source of transmission of the disease to susceptible birds (DAFF 2001).

As described above, cell-free infectious MDV is only associated with the epithelial cells of feather follicles so virus may be present on the skin of infected chickens at slaughter. However, as the commodity considered in this risk analysis will originate from slaughter and processing plants which operate effective Good Manufacturing Process (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes, this will remove dust and dander present on the skin surface.

Furthermore, MDV is inactivated when stored at 4°C for 2 weeks (Calnek and Adldinger 1971). Chilling of chicken meat during transportation to New Zealand will further reduce the amount of virus present on the skin surface.

The likelihood of entry is assessed to be negligible.

25.2.2. Risk estimation

Since the entry assessment is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and MDV is not classified as a risk in imported chicken meat. Therefore, risk management measures cannot be justified.

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26. Multicentric histiocytosis

26.1. HAZARD IDENTIFICATION

26.1.1. Aetiological agent

The definitive aetiologic agent of multicentric histiocytosis (MH) has not been identified although MH has been associated with subgroup J avian leukosis virus (ALV) (Hafner and Goodwin 2008).

26.1.2. OIE list

Not listed.

26.1.3. New Zealand status

No record could be found of MH in New Zealand poultry.

26.1.4. Epidemiology

Multicentric histiocytosis has been described by a number of different synonyms including *big spleen Marek's disease*, *reticuloendotheliosis-like syndrome*, *histiocytic sarcomatosis*, and *spindle-cell proliferative disease* (Hafner and Goodwin 2008).

The disease is seen commonly in broiler chickens at slaughter and presents with splenomegaly (enlarged two to four times the normal size) and hepatomegaly (twice the normal size). Miliary pale nodules are seen throughout the spleen, liver, and kidney. Histological lesions are described in the spleen, liver, kidney, pancreas, proventriculus, lung, enteric mucosa, and bone marrow. Clinically affected birds are often described as pale, anaemic, and underweight (Goodwin *et al.* 1999).

Disease was reproduced when homogenates prepared from the liver, spleen and kidney of chickens diagnosed with MH were injected into three-day-old specific-pathogen-free (SPF) chicks although virus isolation and PCR were unable to determine the aetiological agent responsible (Goodwin *et al.* 1999).

Similarly, spleen homogenates from field cases were able to transmit disease to one-day-old SPF chicks and immunohistochemical studies suggested a role for ALV (most likely the tumorigenic subgroup J ALV) in the development of lesions (Takami *et al.* 2005). PCR testing of field cases of this disease detected specific genes of subgroup J ALV in four out of five birds examined (Takami *et al.* 2004).

Broiler chicks inoculated at hatch with subgroup J ALV became persistently viraemic and developed clinical disease whereas immunotolerised chickens inoculated *in ovo* did not. It has therefore been suggested that the lesions seen in MH metastasise from a primary splenic tumour which may be caused by a reactive process in the spleen against a persistent viral load (Pandiri *et al.* 2009).

ALV transmission occurs either vertically or horizontally, through close contact with infected chickens (Venugopal 1999). Infection does not spread indirectly between birds because of the short half life of the virus. ALVs are rapidly inactivated by temperature and even at -15°C the viral half life is less than 1 week (Fadly and Venugopal 2008).

26.1.5. Hazard identification conclusion

The aetiological agent of MH is not known although the available evidence suggests that it is likely to be due to subgroup J ALV. Experimental studies have shown that infectivity for MH is concentrated in the spleen which will be removed from the commodities considered here. Carcases at slaughter presenting with hepatosplenomegaly and multiple white plaques or nodules are likely to be condemned (Takami *et al.* 2005).

Moreover, transmission of infection requires close contact with an infected bird due to the fragility of the virus. MH is therefore not identified as a potential hazard in the commodities considered here.

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27. *Salmonella* Gallinarum-Pullorum

27.1. HAZARD IDENTIFICATION

27.1.1. Aetiological agent

Salmonella Pullorum, the causal agent of pullorum disease, and *Salmonella* Gallinarum, the causal agent of fowl typhoid. These two bacteria are currently placed in a single species, *Salmonella enterica* subsp. *enterica* serovar Gallinarum-Pullorum, hereafter referred to as *S.* Gallinarum-Pullorum (Shivaprasad and Barrow 2008).

27.1.2. OIE list

Pullorum disease and fowl typhoid are both OIE listed diseases.

27.1.3. New Zealand status

S. Pullorum and *S.* Gallinarum are considered to be exotic to New Zealand (Black 1997). Ongoing serological surveillance of commercial chicken breeder flocks has demonstrated freedom from *S.* Pullorum (Anonymous 2000, 2001; Poland 2002, 2004, 2005; Tana 2007; Frazer 2008). A small serological survey of old English game fowl in 2005 found no evidence of exposure to *S.* Pullorum (Christensen 2006).

Exotic serovars and phage types of salmonellae are listed as notifiable organisms (Tana *et al.* 2011).

27.1.4. Epidemiology

Chickens are the natural host for *S.* Gallinarum-Pullorum (Shivaprasad and Barrow 2008). *S.* Gallinarum-Pullorum in chickens is considered to have a worldwide distribution, including Europe (Christensen *et al.* 1994; Hoop and Albicker-Rippinger 1997; Cobb *et al.* 2005), Africa (Bouzoubaa and Nagaraja 1984; Sato *et al.* 1997; Mdegela *et al.* 2000), North America (Salem *et al.* 1992), Central and South America (de Silva 1984; Lucio *et al.* 1984), and Asia (Majid *et al.* 1991; Nabbut 1993; Mayahi *et al.* 1995; Hoque *et al.* 1997; Kwon *et al.* 2000). Pullorum disease and fowl typhoid are rare in modern commercial poultry companies although epizootics do still occur (Johnson *et al.* 1992; Salem *et al.* 1992).

Mortality from pullorum disease usually occurs in the first 2-3 weeks of life although a proportion of individuals become chronic carriers (Berchieri *et al.* 2001). Fowl typhoid tends to cause disease in older chickens although high mortality in young chicks as a result of fowl typhoid has been described in older literature (Beaudette 1925; Beach and Davis 1927; Martinaglia 1929; Komarov 1932).

Both horizontal and vertical transmission are considered to be important in the spread of *S.* Gallinarum-Pullorum. Transmission by contact with infected chicks in the hatchery can disseminate infection and cannibalism can contribute to spread. Contaminated feed, water, and litter may introduce *S.* Gallinarum-Pullorum into a flock and personnel movements, wild birds, mammals, and flies have been implicated in spread of these diseases (Shivaprasad and Barrow 2008).

Chickens hatched from infected eggs may be found moribund or dead in the incubator or shortly after hatching. Mortality usually peaks after 2 to 3 weeks, and is accompanied by

signs of huddling, laboured breathing, poor development, blindness, and synovitis (Johnson *et al.* 1992; Salem *et al.* 1992; Mayahi *et al.* 1995; Shivaprasad and Barrow 2008). Infection of older chickens may not be detected but can result in acute disease outbreaks characterised by egg drop, diarrhoea, pyrexia, depression, dehydration, and death which are followed by intermittent recurrence and less severe losses. Losses due to pullorum disease are reported to vary from 0 to 100% whilst fowl typhoid is associated with losses from 10 to 93% (Cobb *et al.* 2005; Shivaprasad and Barrow 2008).

Pullorum disease and fowl typhoid are systemic infections and *S.* Gallinarum-Pullorum can be recovered from most internal organs of infected chickens, including the liver, spleen, caeca, lungs, heart, ventriculus, pancreas, yolk sac, synovial fluid, and reproductive organs (Shivaprasad and Barrow 2008). Recovery of the organism from muscle tissue following natural infection has not been documented and chicken meat contamination with *S.* Gallinarum-Pullorum has only been described in environments with poor hygiene practices (Maharjan *et al.* 2006).

There are very few reports of natural infections of ducks with *S.* Gallinarum-Pullorum. Anderson *et al.* (2006) did report recovery from a duck in a petting zoo that had been exposed to the faeces of an infected chicken. Prior to this, the last report of *S.* Gallinarum-Pullorum being recovered from ducks was that of Chute and Gershman (1963) who recovered the organism from a single Muscovy duck in a flock of show birds. Serosurveillance of ducks in Tripura, India detected serological evidence of exposure to *S.* Gallinarum-Pullorum in Khakicampbell ducks but the organism was not recovered from any of the seropositive individuals (Ghosh and Nanda 1989).

In experimental studies, ducks have been shown to be resistant to infection. Buchholz and Fairbrother (1992) attempted to determine an LD_{50} for *S.* Gallinarum-Pullorum in mallard ducks using oral inoculation doses of up to $0.4x10^{10}$CFU. However, no ducks showed any clinical signs of illness during this study and the authors concluded that they underwent only a short subclinical infection that was resolved without any lasting tissue damage. Similarly, Barrow *et al.* (1999) concluded that *S.* Gallinarum-Pullorum was totally avirulent for commercial ducks when administered by the oral route.

No recent reports of natural infection of commercial turkeys with *S.* Gallinarum-Pullorum have been found. Brant (1998) commented that pullorum disease was a major problem as the young turkey industry grew but subsequent measures in a number of countries have virtually eliminated the disease. Correspondence with a number of international experts has been unable to uncover any further examples of commercial turkey flocks being infected with this species (Davies 2009; Gast 2009; Rhorer 2009; Shivaprasad 2009).

There is a difference of opinion among investigators concerning the susceptibility of other avian species to *S.* Gallinarum-Pullorum (Buchholz and Fairbrother 1992). Pullorum disease has been described in pheasants (Pennycott and Duncan 1999) and (experimentally) in bobwhite quail (Buchholz and Fairbrother 1992).

There is a single reported outbreak of gastroenteritis affecting 423 people that was suggested to have been caused by *S.* Gallinarum-Pullorum contamination of eggs used in a rice pudding (Mitchell *et al.* 1946) and a subsequent experimental study found that feeding humans with this organism could induce illness (nausea, vomiting, and diarrhoea) although this was only achieved with very high dosages ranging from $1.3x10^9$ to $10x10^9$ organisms (McCullough and Eisele 1951). *S.* Gallinarum-Pullorum was recovered for up to 121 days after infection from

the faeces of rats orally infected with $5x10^8$ organisms, although no clinical disease was noted (Badi *et al.* 1992).

27.1.5. Hazard identification conclusion

Chickens are recognised as the natural host of *S.* Gallinarum-Pullorum. *S.* Gallinarum-Pullorum is identified as a potential hazard in chicken meat and meat products.

Ducks have been shown to be resistant to infection with *S.* Gallinarum-Pullorum. *S.* Gallinarum-Pullorum is not identified as a potential hazard in duck meat or meat products.

27.2. RISK ASSESSMENT

27.2.1. Entry assessment

The OIE *Code* contains measures against *S.* Gallinarum-Pullorum applicable to trade in live poultry and hatching eggs but not for chicken meat.

Wigley *et al.* (2001) infected one-week old chickens orally with 10^9 organisms and reported that they were able to recover *S.* Gallinarum-Pullorum from breast meat only by enrichment culture during the first week after infection but not subsequently, although the organism was found to persist in bone marrow for at least 5 weeks. Furthermore, Georgiev *et al.* (1978) reported that *S.* Gallinarum-Pullorum could persist on frozen poultry for at least 6 months at -18°C.

In contrast to paratyphoid *Salmonella* spp. which colonise the alimentary tract and are frequently described as contaminants of chicken meat, contamination with *S.* Gallinarum-Pullorum has only been described in environments with poor hygiene practices. Maharjan *et al.* (2006) described the recovery of *S.* Gallinarum-Pullorum from 9% of poultry meat samples taken from a local meat market in Kathmandu and it was noted that Maharjan and Sharma (2000) also found that 85.6% of water sources in Nepal were positive for faecal contamination and 10.8% of these were found to contain *Salmonella* spp. Similarly, Soomro *et al.* (2010) recovered *S.* Gallinarum-Pullorum from poultry meat samples collected at Hyderabad market in Pakistan and noted that a lack of disease control programmes associated with poor handling of raw material from production to marketing was a major problem in that country.

Studies of poultry meat in a number of more developed countries including Korea (Chung *et al.* 2003), Poland (Mikoajczyk and Radkowski 2002), Thailand (Padungtod and Kaneene 2006), Northern Ireland (Wilson 2002), Mexico (Zaidi *et al.* 2006), Belgium (Ghafir *et al.* 2005), and Spain (Capita *et al.* 2003) have consistently failed to identify *S.* Gallinarum-Pullorum as a contaminant of chicken meat.

The commodity considered in this import risk analysis will have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate effective Good Manufacturing Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes. Chicken meat derived from such birds is considered unlikely to act as a vehicle for the spread of *S.* Gallinarum-Pullorum (Cobb 2011). The likelihood of entry is therefore assessed to be negligible.

27.2.2. Risk estimation

Since the entry assessment is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and *S. Gallinarum-Pullorum* is not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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28. Paratyphoid salmonellae

28.1. HAZARD IDENTIFICATION

28.1.1. Aetiological agent

This Chapter considers motile non-host-adapted *Salmonella* serotypes referred to collectively as paratyphoid salmonellae. Over 2,500 serotypes of paratyphoid salmonellae are recognised although only 10% of these have been isolated from poultry (Gast 2008).

28.1.2. OIE list

The *Code* contains sections concerned with the prevention, detection, and control of *Salmonella* in poultry. However, paratyphoid infections of poultry are not OIE listed diseases.

28.1.3. New Zealand status

Salmonella isolates recovered from human and non-human sources in New Zealand are submitted to the Enteric Reference Laboratory of the Institute of Environmental Science and Research Ltd (ESR) for serotyping. Details of the serotypes identified are published regularly (ESR 2003a-2008b) and show that a wide variety of *Salmonella* serotypes are present in this country. It has been estimated that officially recorded data probably represents less than 10% of the real incidence of foodborne disease occurring in the community in countries with developed surveillance systems (Clark *et al.* 2000).

S. Enteritidis PT4, *S.* Typhimurium PT44 and PT104, and *Salmonella* spp. (exotic affecting animals) are listed as unwanted exotic organisms (MAF 2011). However, *S.* Enteritidis PT4 and *S.* Typhimurium PT104 have been recovered in New Zealand on several occasions (ESR 2003a-2008b), and there are no reports of *S.* Typhimurium PT44 in birds (MAF 2009).

Exotic serovars and phage types of salmonellae are listed as notifiable organisms (Tana *et al.* 2011).

28.1.4. Epidemiology

Serotypes of paratyphoid salmonellae have a wide variety of pathological effects in poultry (Okamura *et al.* 2001; Roy *et al.* 2001). Pathogenicity may also vary between strains of a single *Salmonella* serotype (Barrow *et al.* 1987). The pathogenicity of an individual *Salmonella* isolate is thought to be determined by the virulence genes which influence a number of characteristics including heat and acid tolerance, haemagglutination, the ability to invade and survive inside cells, and the expression of lipopolysaccharide (Nolan *et al.* 1991; Petter 1993; Humphrey *et al.* 1996).

A survey of Turkish chickens found 18.6% of caecae obtained at slaughter were contaminated with *Salmonella* spp., with *S.* Enteritidis, *S.* Agona, *S.* Thompson, and *S.* Sarajane recovered from broiler birds, and only *S.* Enteritidis found in layers (Carli *et al.* 2001). Subsequent studies have confirmed that *S.* Enteritidis is the predominant serotype found in Turkish breeding flocks (Eyigor *et al.* 2005). Surveys in Poland (Mikolajczyk and Radkowski 2002) and Spain (Capita *et al.* 2003) have similarly found that *S.* Enteritidis is the predominant serotype found contaminating chicken carcases with all serotypes (except *S.* Sarajane) identified in these studies being recorded as present in New Zealand (ESR 2003a-2008b).

5.5% of 8911 Danish broiler flocks were *Salmonella* positive and a total of 27 different serotypes were recovered (Wedderkopp *et al.* 2001). 1.2% of the isolates recovered from this study were from serotypes that have not been reported in New Zealand (ESR 2003a-2008b).

Salmonella Heidelberg, *S.* Kentucky, *S.* Typhimurium, *S.* Braenderup, and *S.* Enteritidis were identified as the five most common *Salmonella* serotypes identified in chickens in the United States (Ferris *et al.* 2003), all of which are recognised in New Zealand (ESR 2003a-2008b). A survey of Salmonella strains from chickens in the Netherlands between 1984 and 2001 identified the ten most common serotypes recovered as *S.* Enteritidis, *S.* Paratyphi B var Java, *S.* Infantis, *S.* Heidelberg, *S.* Mbandaka, *S.* Typhimurium, *S.* Virchow, *S.* Indiana, *S.* Agona, and *S.* Hadar (van Duijkeren *et al.* 2002). Again, all these serotypes have been described in New Zealand (ESR 2003a-2008b).

The most recent surveillance of *Salmonella* recovered from ducks in the United Kingdom (Veterinary Laboratories Agency 2010) describes 13 different serotypes, all of which have been recognised in New Zealand (ESR 2003a-2008b; Salisbury 1958).

Surveys of poultry meat have shown different rates of contamination in different countries, influenced by national control programmes (Wilson 2002). For example, low rates (6%) of *Salmonella* contamination of poultry meat at retail are reported in Wales (Meldrum *et al.* 2005) compared to high rates in both Mexico (40%) (Zaidi *et al.* 2006) and Thailand (57%) (Padungtod and Kaneene 2006).

Young chicks are highly susceptible to paratyphoid salmonellae and infection is associated with illness and high rates of mortality, whereas older birds can tolerate intestinal colonisation or systemic dissemination without significant morbidity or mortality. The development of resistance with age has been linked to the acquisition of gut microflora that either competes for intestinal receptor sites or inhibits *Salmonella* growth (Stavric *et al.* 1987; Gast 2008).

Oral infection of young chicks leads to colonisation of the intestine and may result in persistent faecal shedding. Infection may then spread within macrophages to the liver and spleen (Barrow *et al.* 1987) before disseminating to other tissues, which may be followed by a bacteraemia associated with high mortality which usually peaks when birds are 3 to 7 days old (Morris *et al.* 1969).

Infection of adult birds with large doses of paratyphoid salmonellae may cause no signs of illness (Humphrey *et al.* 1989). Infection of adult chickens with *S.* Enteritidis may lead to bacteraemia and systemic dissemination with clinical signs usually limited to mild transient diarrhoea (Timoney *et al.* 1989), although mortality associated with the inoculation of adult birds with *S.* Enteritidis PT4 has also been described (Humphrey *et al.* 1991). Faecal shedding of salmonellae occurs for the first 2-3 weeks after infection of adult birds then steadily declines although *S.* Enteritidis has been found in the intestinal tract of chickens for several months after oral inoculation (Gast and Beard 1990a; Gast and Beard 1990b; Shivaprasad *et al.* 1990). Intestinal colonisation of adult birds is usually followed by dissemination to a wide range of internal organs (Gast and Beard 1990b). Highly invasive strains of paratyphoid salmonellae may also be found in the eggs laid by infected birds (mainly *S.* Enteritidis (Henzler *et al.* 1998), and possibly also *S.* Heidelberg (Gast *et al.* 2004) and *S.* Typhimurium DT104 (Williams *et al.* 1998)).

Salmonellae may be introduced into a flock by feed (Cox *et al.* 1983; Rose *et al.* 1999), invertebrate vectors (Kopanic *et al.* 1994; Olsen and Hammack 2000; Davies and Breslin 2003; Skov *et al.* 2004), rodents (Henzler and Opitz 1992), wild birds (Refsum *et al.* 2002), or

even human sewage (Kinde *et al.* 1996). Contamination of the environment is then likely to introduce infection into subsequent flocks (Kumar *et al.* 1971).

Vertical transmission of *Salmonella* can either occur through dissemination of highly invasive strains into eggs before oviposition (Gast and Beard 1990a; Keller *et al.* 1995) or through penetration into or through the shell and shell membranes (Gast 2008). *Salmonella* contamination in or on eggs is also recognised to result in extensive spread in hatcheries (Bailey *et al.* 2002).

28.1.5. Hazard identification conclusion

A limited pool of paratyphoid *Salmonella* spp. have been described in surveys of chickens and ducks overseas, the vast majority of which are recognised to be present in this country. There is no evidence to suggest that the exotic *Salmonella* serotypes found associated with chickens and ducks overseas should be considered any more pathogenic than the serotypes recognised as present in New Zealand.

Paratyphoid salmonellae are not identified as a potential hazard in the commodity.

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29. *Salmonella arizonae*

29.1. HAZARD IDENTIFICATION

29.1.1. Aetiological agent

Salmonella enterica subsp. *arizonae* (*S. arizonae*).

S. arizonae represents a diverse group of bacteria with over 300 serotypes identified. Historically these organisms have been classified in the genus *Arizona* and referred to as either the arizona group, arizonas, or paracolons (Shivaprasad 2008).

29.1.2. OIE list

Not listed.

29.1.3. New Zealand status

S. arizonae has never been reported in animals or birds in New Zealand (MAF 1999). Exotic serovars and phage types of salmonellae are listed as notifiable organisms (Tana *et al.* 2011).

29.1.4. Epidemiology

Avian arizonosis occurs throughout the world and has been associated with considerable losses in commercial turkey operations (Mayeda *et al.* 1978; Crespo *et al.* 2004).

S. arizonae is most frequently seen in turkeys although infections of chickens have also been described (Edwards *et al.* 1947; Silva *et al.* 1980). In a survey of 1308 *S. arizonae* isolates that had been serotyped, 826 cultures were associated with turkeys and 87 cultures obtained from chickens whereas only 3 cultures were reported to have been recovered from ducks (Edwards *et al.* 1956). No other reports associating *S. arizonae* with ducks have been found.

A large survey of chicken and turkey livers at slaughter found no evidence of *S. arizonae* in either of these species (Sadler *et al.* 1965). However, Izat *et al.* (1991) recovered *S. arizonae* from frozen poultry carcases at retail in the United States. Similarly, Jiménez *et al.* (2003) reported the recovery of *S. arizonae* from chicken carcases immediately following slaughter. A survey of chickens in Brazil recovered *S. arizonae* from 3% of healthy birds and 10% of clinically sick birds (de Avila and Moeira 1976). On the basis of available information and expert opinion, a Canadian study classified *S. arizonae* as a hazard likely to be associated with processed poultry (Bisaillon *et al.* 2001).

S. arizonae has also been recovered from a variety of other birds including turkey vultures (Winsor *et al.* 1981), sandhill cranes (Windingstad *et al.* 1977), a sulphur-crested cockatoo (Orós *et al.* 1998), canaries, a parrot, and a macaw (Edwards *et al.* 1956; Edwards *et al.* 1959). In addition, *S. arizonae* has been isolated from reptiles (Sharma *et al.* 1970; Cambre *et al.* 1980; Orós *et al.* 1998) and a variety of mammals (Edwards *et al.* 1956; Edwards *et al.* 1959; Sharma *et al.* 1970).

S. arizonae is also recognised as an opportunistic human pathogen in immunocompromised individuals (Guckian *et al.* 1967; Johnson *et al.* 1976; Weiss *et al.* 1986; Waterman *et al.* 1990; Kelly *et al.* 1995).

Wild birds, rodents, and reptiles have been suggested to be the most common sources of infection for poultry flocks (Hinshaw and McNeil 1947; McClure *et al.* 1957; Goetz 1962). *S. arizonae* is then spread in the faeces of infected birds (Shivaprasad 2008).

Naturally infected chickens show neurological signs including ataxia, torticollis, and opisthotonus. Ocular and brain lesions have been described at post-mortem (Silva *et al.* 1980). Youssef and Geissler (1979) described clinical signs including vent pasting, lens opacity, tremors, opisthotonus, and leg paresis following experimental infection of day-old chicks with *S. arizonae*. Post-mortem examination of infected individuals revealed septicaemia, enlargement of the gall bladder, engorgement of the uterus with urates, and unabsorbed yolk sac. *S. arizonae* were recovered from the eye, brain, heart, liver, yolk sac, and intestines of infected birds.

29.1.5. Hazard identification conclusion

S. arizonae primarily localises to the intestinal tract of adult birds although widespread dissemination of the organism has also been described (MAF 2010). This organism has been recognised in commercial chickens. Given the wide host range described for this organism, the limited evidence of *S. arizonae* in commercial ducks is considered sufficient to consider this organism is also likely to be associated with this species. *S. arizonae* is exotic to New Zealand and is therefore identified as a potential hazard in chicken and duck meat.

29.2. RISK ASSESSMENT

29.2.1. Entry assessment

In an infected flock, shedding of *S. arizonae* can be expected to stop by the time birds reach their slaughter weight. However, long-term carriers of infection are described and surveys of poultry meat have identified extremely low rates of *S. arizonae* contamination in frozen chicken carcases at retail (Izat *et al.* 1991). The likelihood of entry is assessed as low.

29.2.2. Exposure assessment

Backyard poultry

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of the Animal Products Act 1999. Such commercial producers should not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and could be considered likely to feed food scraps to their birds (Wintle 2010). The feeding of uncooked waste food (including poultry meat) collected from retail and catering outlets to commercial and non-commercial poultry in New Zealand has been described (Mulqueen 2012).

Except for a few distinctively thermoresistant strains, salmonellae are generally susceptible to destruction by heat (Gast 2008). There is a negligible likelihood that backyard poultry will be exposed to *S. arizonae* in scraps of imported chicken or duck meat after it has been cooked.

S. arizonae may be present on scraps of raw chicken or duck meat generated during domestic processing so there is a non-negligible likelihood that backyard poultry could be exposed to this organism if fed raw meat scraps.

Wild birds

S. arizonae has been recovered from a variety of avian species, and it is reasonable to assume that free-living avian species in New Zealand could be exposed to this organism either from an infected backyard flock or through consumption of uncooked chicken or duck meat in kitchen waste disposed of at accessible sites.

Commercial poultry

As described above, although commercial producers should not feed food scraps to their birds, the feeding of uncooked waste food from retail and catering outlets is recognised on New Zealand poultry farms (Mulqueen 2012). A voluntary agreement was in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand (Wintle 2010) although this has now been discarded by at least one large feed manufacturer (Mulqueen 2012).

Recommended minimum biosecurity standards for domestic producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures reduce the likelihood of commercial poultry being exposed to free-living avian species. However, wild birds have been suggested as a common source for infection of poultry flocks so the likelihood of exposure of commercial poultry from free-living avian species is assessed to be non-negligible.

Standard biosecurity practices on commercial poultry farms include the prohibition of staff in regular contact with poultry livestock from keeping avian species at their homes, regularly contacting owners of cage birds or racing pigeons, and regularly contacting any operation that uses poultry manure in bulk (Poultry Industry Association of New Zealand 2007). Surveys of commercial poultry farms have shown a generally high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents, especially in broiler farms (Rawdon *et al.* 2007; Rawdon *et al.* 2008).

Although on-farm biosecurity measures would reduce the likelihood of commercial poultry being exposed to *S. arizonae* from an infected backyard flock, rodents and reptiles have been suggested as common sources for the introduction of this organism into commercial flocks. Therefore, the likelihood of commercial poultry being exposed to *S. arizonae* from rodents and reptiles that have been in contact with an infected backyard flock is assessed to be non-negligible.

Exposure assessment conclusion

In conclusion, the likelihood of exposure of backyard poultry, wild birds, and commercial poultry to *S. arizonae* is assessed to be non-negligible.

29.2.3. Consequence assessment

S. arizonae is known to infect chickens and turkeys. Infection of chickens is not considered to economically important although infection has been associated with considerable losses in commercial turkey operations (Shivaprasad 2008).

S. arizonae has been recovered from a variety of free-living avian species with no associated clinical disease (Windingstad *et al.* 1977; Winsor *et al.* 1981). However, *S. arizonae* has been described as the cause of a fatal hepatitis in a captive psittacine (Orós *et al.* 1998).

Reptiles are commonly associated with *S. arizonae* and it is considered to be part of their normal intestinal microflora in many species (Cambre *et al.* 1980). However, *S. arizonae* may act as an opportunistic pathogen in individuals with a depressed immune response (Orós *et al.* 1998).

If *S. arizonae* were to become established in New Zealand, infection of humans could occur following exposure to reptiles, wild birds, pet birds, or poultry. *S. arizonae* has been associated with a variety of disease syndromes in immunocompromised humans, including gastroenteritis, septicaemia, and localised infections (Guckian *et al.* 1967).

The introduction of *S. arizonae* in the commodity would be associated with non-negligible consequences for the New Zealand poultry industries, wildlife, and human health. The consequence assessment is therefore assessed to be non-negligible.

29.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and *S. arizonae* is classified as a risk in imported chicken or duck meat. Therefore, risk management measures can be justified.

29.3. RISK MANAGEMENT

29.3.1. Options

To effectively manage the risk of imported chicken or duck meat being contaminated with *S. arizonae*, measures could require birds to be free from infection at slaughter or meat could be treated to ensure the destruction of this organism.

Meat derived from birds originating from flocks in a country, zone, or compartment recognised to be free from *S. arizonae* could be considered suitable for importation without further sanitary measures.

Standard methods for the culture and identification of *S. arizonae* from poultry and their environment have been described (Timms 1971; Shivaprasad 2008). Culturing of litter has also been recommended to identify infected flocks (Snoeyenbos and Smyser 1969; Greenfield and Bigland 1971).

It is assumed that the difficulties in the detection of arizonosis in a turkey flock described by Timms (1971) would also be applicable to chicken or duck flocks:

i. The number of poults in an infected flock showing clinical signs may be as low as 5%

ii. Adult carriers may be free from clinical signs and repeatedly yield negative cloacal swabs

iii. Adult carriers may be serologically negative 12-14 weeks after exposure

iv. There is little correlation between positive cloacal swabs and antibodies in poults and adults

v. *S. arizonae* may be present in very small numbers in the tissues of infected birds

Chapter 6.5 of the *Code* describes measures for the prevention, detection and control of all *Salmonella* in poultry although the emphasis of this Chapter is on *S.* Enteritidis and *S.* Typhimurium. Meat derived from breeding flocks, hatcheries, and rearing farms that comply with the guidelines in Chapter 6.5 of the *Code* and have been shown to be free from *S. arizonae* in accordance with these could be considered suitable for importation.

Schnepf and Barbeau (1989) found that heating to a core temperature of 79°C was sufficient to eliminate viable *Salmonella* from whole roasting chickens that had been bathed in a 500ml solution containing $5x10^7$ *Salmonella* Typhimurium/ml.

Juneja *et al.* (2001) estimated (based on extrapolation from studies performed over a temperature range of 58°C to 65°C) that exposure of turkey meat to 74°C for around 9 seconds would be required to ensure a 7 log reduction in *Salmonella*. The non-linear survival curves generated by the modelling work done by this group (which form the basis of the USDA FSIS standard) are considered by MPI to be appropriate to specify the time/temperature requirements to manage the risk of *Salmonella arizonae* in turkey meat. Based on these results, the following conditions (which will achieve a 7 log reduction in *Salmonella* in turkey meat with 12% fat) are included in MPI's import health standards for turkey meat:

- 60°C for 2030 seconds
- 62°C for 1073 seconds
- 65°C for 370 seconds
- 70°C for 41 seconds
- 72°C for 19 seconds
- 74°C for 9 seconds
- 76°C for 4 seconds
- 79°C for 1 second

One or a combination of the following options could be considered in order to effectively manage the risk:

Option 1

Imported chicken or duck meat could be derived from birds in a country, zone, or compartment free from *S. arizonae*.

Option 2

Imported chicken or duck meat could be derived from breeding flocks, hatcheries, and rearing farms that have been shown to be free from *S. arizonae* in accordance with the guidelines in Chapter 6.5 of the *Code*.

Option 3

Imported chicken or duck meat could be cooked as currently required by MPI's import health standards for turkey meat (described above).

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30. *Campylobacter* spp.

30.1. HAZARD IDENTIFICATION

30.1.1. Aetiological agent

Infection with *Campylobacter* spp., principally *C. jejuni* and *C. coli* (Zhang 2008).

30.1.2. OIE list

Not listed.

30.1.3. New Zealand status

50-60% of raw chicken meat in New Zealand is contaminated with *Campylobacter* spp. (Wong *et al.* 2006). A recent study of 193 *Campylobacter* spp. recovered from New Zealand poultry found no isolates with resistance to quinolone antibiotics although one isolate was identified that was considered to be resistant to erythromycin (French 2009).

Although most public health laboratories in New Zealand do not routinely test the antimicrobial susceptibility of *Campylobacter* isolates, a low level of antimicrobial resistance was seen between 2001 and 2005, with <2% of human isolates showing resistance to erythromycin and 3-4% of isolates resistant to fluoroquinolones (Heffernan *et al.* 2006).

30.1.4. Epidemiology

C. jejuni and *C. coli* are widespread in commercial poultry (Sahin *et al.* 2002) and may be introduced into a flock from litter, drinking water, other animals (farmed, pets or wildlife), insects, or fomites (Zhang 2008). Following introduction, the majority of birds in a flock quickly become colonised with *Campylobacter* (Berndtson *et al.* 1996; Gregory *et al.* 1997). The role of vertical transmission in the epidemiology of *Campylobacter* introduction into poultry flocks remains unresolved (MAF Biosecurity New Zealand 2009).

C. jejuni and *C. coli* are well adapted to avian hosts and produce little or no clinical disease in poultry (Newell and Fearnley 2003; Lee and Newell 2006). Experimental studies have shown that inoculation of chicks up to 3 days old with *Campylobacter* can cause diarrhoea (Ruiz-Palacios *et al.* 1981; Sanyal *et al.* 1984; Welkos 1984). However, other reports have recorded no clinical disease in poultry experimentally infected with *Campylobacter* (Beery *et al.* 1988; Shanker *et al.* 1988; Stern *et al.* 1988; Sahin *et al.* 2003; Knudsen *et al.* 2006). The ostrich is the only avian species where natural infection with *Campylobacter* has been associated with clinical disease (Verwoerd 2000).

Campylobacter are considered to be a leading bacterial cause of human foodborne gastroenteritis (Mead *et al.* 1999). The high prevalence of *Campylobacter* in the intestinal tract of poultry is considered to be the source of carcase contamination in retail poultry (Jeffrey *et al.* 2001) and surveys of chicken carcases consistently show the majority to be contaminated (Willis and Murray 1997; Zhao *et al.* 2001; Jørgensen *et al.* 2002). Zhao *et al.* (2001) also noted that although 70.7% of chicken carcases in their study were found to be contaminated with *Campylobacter*, only 14% of turkey carcases were similarly contaminated. *Campylobacter* isolates recovered from poultry are recognised to have developed resistance to a number of clinically important antimicrobials, including the fluoroquinolones (Avrain *et al.* 2003; Zhang *et al.* 2003; Gupta *et al.* 2004; Luangtongkum *et al.* 2006).

30.1.5. Hazard identification conclusion

Poultry-associated *Campylobacter* spp. are recognised to be prevalent in New Zealand and are considered to be non-pathogenic commensal organisms in farmed avian species. There is no evidence that strains of *Campylobacter* associated with poultry overseas are more virulent than those found in this country.

For the above reasons, *Campylobacter* spp. are not identified as a potential hazard in the commodity.

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31. *Escherichia coli*

31.1. HAZARD IDENTIFICATION

31.1.1. Aetiological agent

Localised or systemic infection caused by avian pathogenic *Escherichia coli* (APEC) (Barnes *et al.* 2008).

E. coli are classified according to the Kauffmann scheme on the basis of their somatic (O), flagellar (H), and capsular (K) antigens. More than 180 O, 60 H, and 80 K antigens are currently described (Stenutz *et al.* 2006).

31.1.2. OIE list

Not listed.

31.1.3. New Zealand status

Colibacillosis has been described in New Zealand poultry (Ross 1984; Orr 1994; Orr 1995; Orr 1998) and has been associated with various disease manifestations including omphalitis, peritonitis, salpingitis, airsacculitis, colisepticaemia, coligranuloma, synovitis, and opthalmitis (Black 1997).

31.1.4. Epidemiology

E.coli is a common inhabitant of the intestinal tract of most mammals and birds. Potentially pathogenic strains of *E.coli* may be found in the intestines of around 13% of healthy chickens (Harry and Hemsley 1965).

Colibacillosis is responsible for significant economic losses in poultry flocks throughout the world. A survey of a poultry processing plant in the United Kingdom found 43% of broiler carcase rejections were due to colisepticaemia (Yogaratnam 1995) and *E.coli* was also found to be responsible for the majority of infections resulting in the condemnation of broiler carcases in Switzerland (Jakob *et al.* 1998). Post-mortem examination of poultry from 503 farms in Belgium demonstrated disease due to APEC in 153 farms (Vandemaele *et al.* 2002), a survey of 100 broiler farms in Jordan found 88% of airsaccultitis cases were due to *E. coli* (El-Sukhon *et al.* 2002), and colibacillosis was found to be one of the most common diseases affecting Californian turkey flocks (Christiansen *et al.* 1996).

APEC isolates are generally considered to act as opportunistic pathogens and avian colibacillosis is thought to be a secondary disease. However, clones of APEC exist that are well adapted as pathogens and may not always require the presence of a primary predisposing infection (Barnes *et al.* 2008). APEC can be distinguished from commensal *E.coli* strains based on the ability to cause mortality in embryos or chicks, and this is regarded as the best single test for discriminating APEC from commensal *E. coli* strains (Gibbs *et al.* 2003; Gibbs and Wooley 2003; Gibbs *et al.* 2004). However, virulence assays do not account for predisposing host or environmental factors which may enable a less virulent isolate to cause disease under natural conditions (Nolan *et al.* 2002).

Surveys to determine which *E.coli* serotypes are present in poultry show that the predominant serotypes vary with geographic region (Sharada *et al.* 2001; Rosario *et al.* 2004) although

Barnes *et al.* (2008) have described the most common serotypes identified as O1, O2, O35, O36, and O78.

Most APEC isolates are only pathogenic to chickens although *E.coli* O157 has been identified in both chickens (Pilipčinec *et al.* 1999) and ducks (Samadpour *et al.* 2002; Leclercq and Mahillon 2003). Chicken meat has also been recognised as a source of *E.coli* with virulence and antimicrobial resistance factors (Doyle and Schoeni 1987; Griffin and Tauxe 1991; Johnson *et al.* 2003).

All ages of poultry are susceptible to colibacillosis although disease is reported more often and with more severe clinical signs in developing embryos and chicks (Harry 1957; Goren 1978; Montgomery *et al.* 1999; Johnson *et al.* 2001). Infections can be predisposed by other infectious agents such as infectious bronchitis virus (Williams Smith *et al.* 1985; Nakamura *et al.* 1996) or haemorrhagic enteritis virus (Newberry *et al.* 1993; van den Hurk *et al.* 1994), or by environmental factors such as dust or high levels of ammonia (Oyetunde *et al.* 1978; Nagaraja *et al.* 1984). The incidence of colibacillosis has been shown to be related to the number of primary infections birds are exposed to before being challenged with *E. coli* (Pierson *et al.* 1996).

New strains of *E.coli* can be introduced into a flock through contact with other animals or their faeces (Barnes *et al.* 2008). Avian-adapted strains may be acquired from free-living waterfowl (Fallacara *et al.* 2001; Fallacara *et al.* 2004; Cole *et al.* 2005) or passerine species (Morishita *et al.* 1999). Houseflies (*Musca domestica*) have also been associated with the transmission of *E.coli* (Rochon *et al.* 2004; Rochon *et al.* 2005).

The most frequent pathology in poultry associated with *E. coli* is systemic infection (Stordeur *et al.* 2002). Clinical signs of avian colibacillosis are highly variable, including localised infections (omphalitis, cellulitis, diarrhoea, vaginitis, salpingitis, and orchitis) and systemic diseases (colisepticaemia, airsaccultitis, meningitis, synovitis, and polyarthritis) (Barnes *et al.* 2008). Although primary enteritis is a common manifestation of *E.coli* infections in mammals, it is considered rare in poultry (Barnes 2008). Morbidity and mortality are highly variable depending on the type of disease associated with infection (Barnes *et al.* 2008).

31.1.5. Hazard identification conclusion

Colibacillosis is recognised in New Zealand poultry and has been associated with a variety of disease presentations. The clinical manifestation of colibacillosis is likely to be determined by underlying host, infectious, or environmental factors. There is no evidence to suggest that strains of APEC found overseas are any more virulent than the strains encountered in this country.

APEC is not identified as a potential hazard in the commodity.

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32. *Riemerella anatipestifer*

32.1. HAZARD IDENTIFICATION

32.1.1. Aetiological agent

Riemerella anatipestifer is a Gram-negative, nonmotile, nonspore-forming rod (Sandhu 2008). The organism was originally named *Pfeifferella anatipestifer* (Hendrickson and Hilbert 1932), then *Moraxella anatipestifer* (Bruner and Fabricant 1954) and *Pasteurella anatipestifer*. Subsequent molecular investigation of this organism has placed it in the genus *Riemerella* (Segers *et al.* 1993).

32.1.2. OIE list

Not listed.

32.1.3. New Zealand status

Anatipestifer syndrome of ducklings due to an organism tentatively classified as *Pasteurella anatipestifer* has been described (Anonymous 1974; Hemsley 1996). A histopathological diagnosis was recorded in 1990 when paralysis of ducks was found to be accompanied by a spectacular meningoencephalitis typical of this organism (Orr 1990).

32.1.4. Epidemiology

R. anatipestifer has a worldwide distribution although the severity of disease varies widely depending on the strain of the organism, the infectious dose, the age of the host, and the route of exposure (Sarver *et al.* 2005; Sandhu 2008).

21 serotypes of *R. anatipestifer* have been described with different serotypes being predominant in different geographical locations (Sandhu and Leister 1991; Sandhu 2008). Harry (1969) identified eight different serotypes (designated A to H) from 171 cultures of *R. anatipestifer* recovered from 73 flocks with anatipestifer septicaemia in Norfolk and Lincolnshire over a 3 year period. Only strains identified as serotype A (designated serotype 1 under current nomenclature) were capable of reproducing disease when inoculated subcutaneously into ducks and were associated with higher flock mortality than the other strains identified. Between 1976 and 1979, the majority of disease outbreaks in ducks in Denmark were also associated with *R. anatipestifer* serotype 1 although in 1980 serotype 3 (which had previously only been recovered from swans and geese) became the predominant isolate associated with disease outbreaks (Bisgaard 1982). Serotypes 1, 2, 3, 5, and 15 have been found to be most prevalent in severe outbreaks of anatipestifer septicaemia (Crasta *et al.* 2002).

The reasons for variation in strain virulence are not fully understood although Crasta *et al.* (2002) linked *R. anatipestifer* expression of the CAMP cohaemolysin with virulence and demonstrated expression of this cohaemolysin in strains from serotypes 1, 2, 3, 5, 6, and 19. The pCFC1 plasmid (found in 60% of isolates studied) has also been suggested as the origin of virulence determinants in *R. anatipestifer* isolates (Chang *et al.* 1998). The divergence of the 21 recognised *R. anatipestifer* serotypes contributes to low cross-protection against different strains and variations in virulence factors, resulting in mixed infections of more than one serotype of *R. anatipestifer* in the same individual and frequent changes of serotypes in the same farm (Yu *et al.* 2008).

Infection with *R. anatipestifer* is considered to be primarily a disease of ducks and geese although disease outbreaks have been reported in chickens (Rosenfeld 1973). *R. anatipestifer* has also been recovered from turkeys (Zehr and Ostendorf 1970; Helfer and Helmboldt 1977; Smith *et al.* 1987; Frommer *et al.* 1990), pheasants (Bruner *et al.* 1970), guinea fowl and quail (Sandhu 2008), partridges (Wyffels and Hommez 1990), and other waterfowl including whistling swans (Wobeser and Ward 1974), black swans (Munday *et al.* 1970), blue and snow geese, mandarin ducks, a white-fronted goose, a black duck, and a wood duck (Karstad *et al.* 1970). Hinz *et al.* (1998) reported the recovery of *R. anatipestifer* from a number of additional species, including guillemot, a herring gull, a black-headed gull, a budgerigar, and pigs.

Transmission is considered to occur via the respiratory route or through skin wounds although an arthropod vector (*Culex* mosquitoes) has also been suggested (Cooper 1989).

Infection is followed by an incubation period of 2-5 days before clinical signs are seen, which include listlessness, ocular and nasal discharge, coughing, sneezing, diarrhoea, ataxia, coma, and death, with a mortality rate of between 5 to 75% (Sandhu 2008).

Post-mortem findings are typically those of acute or chronic septicaemia, characterised by fibrinous pericarditis, perihepatitis, airsaccultitis, and meningitis (Helfer and Helmboldt 1977; Smith *et al.* 1987). In addition, infection can lead to cellulitis with thickening of the skin on the ventral abdomen accompanied by tracks of caseous pus between the dermis and underlying musculature which may be barely noticeable on gross examination (Gooderham 2002).

32.1.5. Hazard identification conclusion

Infection with *R. anatipestifer* may be accompanied by marked clinical signs in live birds and significant post-mortem pathology. Imported chicken and duck meat will be derived from birds that have passed ante-mortem and post-mortem inspection. Although inspection is likely to detect clinically affected individuals, birds infected 2-5 days before slaughter or those exhibiting less marked clinical signs could go undetected.

The history outlined in 31.1.3 above suggests that *R. anatipestifer* should be considered likely to be present in New Zealand. However, given that no further isolates of this organism have been recorded since 1974 and the divergence of the 21 different serotypes recognised globally, it is reasonable to assume that only less virulent serotypes may be present in this country.

Exotic serotypes of *R. anatipestifer* are therefore identified as a potential hazard in the commodity.

32.2. RISK ASSESSMENT

32.2.1. Entry assessment

As described above, infection with *R. anatipestifer* may be associated with lesions barely noticeable on gross examination including caseous pus between the dermis and underlying musculature.

Broth cultures of *R. anatipestifer* remain viable for 2-3 weeks if stored at 4°C (Bangun *et al.* 1981; Sandhu 2008)

The likelihood of entry is assessed to be non-negligible.

32.2.2. Exposure assessment

It is considered unlikely that *R. anatipestifer* would remain viable after processing meat to a core temperature exceeding 60°C for 30 minutes and reaching 80°C for at least 10 minutes (MAF 2006) and *R. anatipestifer* is inactivated at 60°C after 1 hour (Harry and Deb 1979). Therefore there is a negligible likelihood of *R. anatipestifer* persisting in scraps of chicken and duck meat following domestic cooking.

Hendrickson and Hilbert (1932) found that feeding pure cultures of *R. anatipestifer* to ducklings over a 10 day period did not transmit infection and were only able to reproduce disease using intravenous inoculation. Similarly, Asplin (1956) demonstrated that infection could be readily transmitted through wounds, scratches, fissures, or punctures of the skin but was unable to infect ducks using a culture suspension of *R. anatipestifer* given orally.

Graham *et al.* (1938) were able to transmit disease to young ducks when *R. anatipestifer* was administered intraperitoneally, intravenously, intratracheally, or (occasionally) intra-conjunctivally. However, installation of *R. anatipestifer* into the crop did not result in infection.

Dougherty *et al.* (1955) reported that they were able to successfully transmit disease to ducks using intratracheal and intraperitoneal inoculations of suspensions of ground spleen, liver, and serosal exudates.

Hatfield and Morris (1988) inoculated 16-day-old ducks with 10^9 CFU of *R. anatipestifer* given either intramuscularly, intranasally, or orally. Intramuscular challenge resulted in clinical signs and mortality in all birds within 3 days, intranasal challenge resulted in clinical signs (but no deaths) in 2 of 12 inoculated birds, and no disease signs or deaths were observed in orally challenged ducks.

Sarver *et al.* (2005) inoculated ducks with *R. anatipestifer* using a range of challenge doses ($0.5x10^2$ CFU to $0.5x10^6$ CFU) given via the subcutaneous, intravenous, oral, and nasal routes. Whilst inoculation via the intravenous and subcutaneous routes was associated with significant mortality at all challenge doses, there were no deaths associated with oral inoculation using a dose of either $0.5x10^2$ CFU or $0.5x10^4$ CFU and only one death (n=11) recorded following oral inoculation with a dose of $0.5x10^6$ CFU.

Considering the above evidence, there is a negligible likelihood of *R. anatipestifer* being transmitted to susceptible species through the ingestion of uncooked chicken or duck meat scraps. The likelihood of exposure is assessed to be negligible.

32.2.3. Risk estimation

Since the exposure assessment for *R. anatipestifer* is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and this organism is not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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33. *Ornithobacterium rhinotracheale*

33.1. HAZARD IDENTIFICATION

33.1.1. Aetiological agent

Ornithobacterium rhinotracheale is a Gram-negative, nonmotile, highly pleomorphic, rod-shaped, nonsporulating bacterium. The organism is closely related to *Riemerella anatipestifer* and *Coenonia anatine* and has previously been designated as *Pasteurella*-like and *Kingella*-like (Chin *et al.* 2008).

33.1.2. OIE list

Not listed.

33.1.3. New Zealand status

O. rhinotracheale has not been isolated in New Zealand (Black 1997).

33.1.4. Epidemiology

Van Empel *et al.* (1997) described seven distinct serotypes of *O. rhinotracheale* and currently 18 serotypes of the organism have been identified (designated A to R) with serotype A most common amongst chicken isolates. Different serotypes are associated with different geographical origins and pathogenicity varies between isolates (Chin *et al.* 2008).

O. rhinotracheale has been recovered from both chickens (Charlton *et al.* 1993; Vandamme *et al.* 1994) and ducks (van Empel and Hafez 1999). Outbreaks of disease associated with *O. rhinotracheale* have been reported in chicken flocks throughout the world including Belgium (Devriese *et al.* 1995), Brazil (Canal *et al.* 2003; Canal *et al.* 2005), Egypt (Elgohary and Awaad 1998), France (Leroy-Sétrin *et al.* 1998), Japan (Sakai *et al.* 2000), Jordan (El-Sukhon *et al.* 2002), Mexico (Soriano *et al.* 2002), Pakistan (Naeem *et al.* 2003), Peru (Hung and Alvarado 2001), South Africa (Travers 1996), and the United States (Odor *et al.* 1997; Sprenger *et al.* 2000).

In natural disease outbreaks in commercial poultry, *O. rhinotracheale* is often identified as a co-infection alongside other respiratory pathogens such as *Escherichia coli* (Odor *et al.* 1997; Elgohary and Awaad 1998; Sakai *et al.* 2000; El-Sukhon *et al.* 2002), *Bordetella avium* (El-Sukhon *et al.* 2002), Newcastle disease virus (Travers 1996; Odor *et al.* 1997), infectious bronchitis virus (Odor *et al.* 1997), *Mycoplasma synoviae* (Zorman-Rojs *et al.* 2000), or *Chlamydia psittaci* (van Loock *et al.* 2005).

In experimental studies, infection with *O. rhinotracheale* alone is associated with minimal pathological lesions and the severity of lesions is enhanced by co-infection with other respiratory pathogens (van Empel *et al.* 1996; van Empel *et al.* 1999). However, a number of studies have shown that *O. rhinotracheale* alone is capable of causing respiratory disease in chickens (Travers *et al.* 1996; Sprenger *et al.* 1998; van Veen *et al.* 2000).

Infection with *O. rhinotracheale* is associated with a short incubation period, with seroconversion seen in 4-week-old chickens within 4 days of experimental infection (van Empel *et al.* 1999).

Post-mortem findings in infected chickens include airsacculitis together with a unilateral pneumonia. Subcutaneous cranial oedema with osteitis, osteomyleitis, and encephalitis are also decribed (Odor *et al.* 1997; Chin *et al.* 2008).

The trachea, lungs, and air sacs are considered the best tissues from which to isolate *O. rhinotracheale* from infected birds. Following experimental infection, the organism has also been recovered from blood, liver, joints, brain, ovary, and oviduct, although field trials have been unsuccessful in recovering *O. rhinotracheale* from heart blood and liver (Chin *et al.* 2008).

33.1.5. Hazard identification conclusion

Infection with *O. rhinotracheale* may be accompanied by marked clinical signs in live birds and significant post-mortem pathology, which are likely to be detected during ante-mortem and post-mortem inspection. However, the severity of clinical signs, duration of the disease, and mortality of *O. rhinotracheale* outbreaks are extremely variable (Chin *et al.* 2008).

Following infection, lesions and infectivity are restricted mainly to the respiratory tissues. *O. rhinotracheale* is not identified as a potential hazard in those commodities that exclude respiratory tract material.

Although respiratory tract tissues will be removed from chicken and duck carcases, remnants of these tissues may remain following processing. *O. rhinotracheale* is therefore identified as a potential hazard in imported whole chicken or duck carcases.

33.2. RISK ASSESSMENT

33.2.1. Entry assessment

The clinical signs associated with *O. rhinotracheale* infection are extremely variable (Chin *et al.* 2008) so it is unlikely that infected flocks would be reliably detected during ante-mortem inspection.

Following infection, *O. rhinotracheale* is found primarily in the respiratory tract. These tissues will be removed from birds at slaughter although it has been previously estimated that some upper respiratory tract tissue will remain in around 0.2% of processed chicken carcases (MAF 1999). In the absence of any evidence to the contrary, it is assumed that a similar figure would apply to duck carcases. Furthermore, the commodity considered here may include carcases with the head attached (see Section 4).

Considering the above, the likelihood of *O. rhinotracheale* entry in imported chicken or duck carcases is assessed to be non-negligible.

33.2.2. Exposure assessment

O. rhinotracheale is closely related to *Riemerella anatipestifer* (Chin *et al.* 2008). *R. anatipestifer* is inactivated at 60°C after 1 hour (Harry and Deb 1979) so it is assessed that there is a negligible likelihood of *O. rhinotracheale* persisting in scraps of chicken or duck meat following domestic cooking.

Any respiratory tissue remnants in imported chicken or duck carcases would be unlikely to be removed prior to cooking although, in the absence of any data to support this, it is assumed

that some of this may be discarded as raw tissue prior to cooking and therefore accessible to backyard poultry or wild birds.

Van Empel *et al.* (1996) demonstrated that injection of *O. rhinotracheale* directly into air sacs resulted in a significant decrease in the daily weight gain of turkeys and that aerosol challenge of turkeys resulted in a severe airsacculitis but no growth retardation. Sprenger *et al.* (1998) were able to reproduce clinical disease in turkeys using intratracheal inoculation with a pure culture of the organism and demonstrated that this route was more effective than intravenous inoculation with a pure culture.

As there is no evidence for the spread of *O. rhinotracheale* other than by the respiratory route, ingestion of scraps of chicken or duck meat discarded from imported carcases would not transmit infection so the likelihood of exposure is assessed to be negligible.

33.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and *O. rhinotracheale* is not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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34. *Bordetella avium* (turkey coryza)

34.1. HAZARD IDENTIFICATION

34.1.1. Aetiological agent

Bordetella avium is a Gram-negative, nonfermentative, motile, strictly anaerobic bacillus, previously described as *Alcaligenes faecalis* (Jackwood and Saif 2008).

34.1.2. OIE list

Not listed.

34.1.3. New Zealand status

B. avium has not been isolated in New Zealand (Black 1997).

34.1.4. Epidemiology

Turkeys are considered to be the natural host of *B. avium* although the organism has also been recovered from other avian species (Simmons *et al.* 1981; Hinz *et al.* 1983; Raffel *et al.* 2002). Strains of *B. avium* recovered from turkeys and chickens are similar and cross-infection can occur between these species (Simmons *et al.* 1981).

B. avium has been recovered from ducks and chickens in Germany (Hinz *et al.* 1983). A survey of wild and domesticated birds in the United States reported serological evidence of exposure to *B. avium* in domesticated chickens, mallard ducks, Muscovy ducks, and a wood duck, and *B. avium* was recovered from tracheal swabs of mallard ducks (Raffel *et al.* 2002).

Isolates of *B. avium* show very little antigenic, cultural, or biochemical variation although differences in pathogenicity have been reported for different strains (Saif *et al.* 1980; Rimler and Simmons 1983).

Bordetellosis is recognised in commercial flocks in major turkey-producing regions throughout the world including Germany (Hinz *et al.* 1978) and the United States (Saif *et al.* 1980; Panigrahy *et al.* 1981; Boycott *et al.* 1984; Kelly *et al.* 1986), although co-infection with other bacteria and viruses is thought to be significant in outbreaks of disease (Heller *et al.* 1984; Lister and Alexander 1986).

Disease is usually seen in turkeys from 2 to 6 weeks old (Hinz *et al.* 1978; Panigrahy *et al.* 1981; Boycott *et al.* 1984) although infection of mature birds (39 to 40 weeks old) may also be associated with clinical disease (Kelly *et al.* 1986). Transmission of infection occurs through close contact or exposure to contaminated litter or water and is enhanced by social or physiological stress. Aerosol transmission is considered unlikely (Simmons and Gray 1979). Following infection with *B. avium*, the incubation period is 4 to 10 days, which leads to inflammation of the respiratory mucosa with accompanying clinical signs of sneezing, mouth breathing, stunting, oculonasal discharge, submandibular oedema, dyspnoea, tracheal collapse and a predisposition to other infectious diseases. Signs of disease subside after 2 to 4 weeks (Saif *et al.* 1980; Panigrahy *et al.* 1981; Gray *et al.* 1983; van Alstine and Arp 1988; Jackwood and Saif 2008).

Outbreaks are usually associated with a high morbidity and low mortality (Saif *et al.* 1980; Kelly *et al.* 1986) although higher mortality rates and more severe clinical signs may be seen in the presence of concomitant infections (Saif *et al.* 1980; Boycott *et al.* 1984; Cook *et al.* 1991).

Gross lesions (nasal and tracheal exudates, distortion of tracheal cartilage, and hyperaemia of the nasal and tracheal mucosae) are confined to the upper respiratory tract (Arp and Cheville 1984). Microscopically, *B. avium* adheres to ciliated epithelium of the nasal mucosa, progressing down the trachea and into the primary bronchi. Bacteria have not been found attached to any other cell types in infected birds (Arp and Fagerland 1987). *B. avium* can be recovered from the trachea and primary bronchi of infected birds but not from lung parenchyma (van Alstine and Arp 1988).

B. avium infection of chickens results in respiratory disease with similar but less severe clinical signs and lower mortality compared to the disease described in turkeys. Simmons *et al.* (1981) described disease in 2 to 6-week-old broilers which presented as severe conjunctivitis, râles and nasal exudation. At post-mortem examination, affected chickens were found to have a small to moderate accumulation of clear mucus in the nasal turbinates and trachea with no other changes observed in uncomplicated cases. As has been described with turkeys, secondary bacterial infections were also seen and were associated with perihepatitis, pericarditis, and airsacculitis. Coryza-like disease in ducks has been produced experimentally using isolates of *B. avium* recovered from turkeys (Hinz *et al.* 1983).

34.1.5. Hazard identification conclusion

Following infection, *B. avium* attaches to and causes lesions in the upper respiratory tract tissues. There is no evidence of this agent in any other tissues. *B. avium* is not identified as a potential hazard in those commodities that exclude upper respiratory tract material, namely chicken or duck meat or meat products.

Although respiratory tract tissues will be removed from carcases, remnants of these tissues may remain following processing. *B. avium* is therefore identified as a potential hazard in imported whole chicken or duck carcases.

34.2. RISK ASSESSMENT

34.2.1. Entry assessment

Infection with *B. avium* may be associated with mild clinical signs unless concomitant infections are present so it is unlikely that infected flocks would be reliably detected during ante-mortem inspection.

Following infection, *B.avium* is only found in upper respiratory tract tissues that will be removed from birds at slaughter. However, it has been previously estimated that some upper respiratory tract tissue will remain in around 0.2% of processed chicken carcases (MAF 1999).

Considering the above, the likelihood of *B. avium* entry in imported chicken or duck carcases is assessed to be very low.

34.2.2. Exposure assessment

B. avium can be considered susceptible to heat as cultures of the organism are killed following exposure to 45°C (Arp and McDonald 1985) so it is assessed that there is a negligible likelihood of *B. avium* persisting in scraps of meat following domestic cooking.

Any respiratory tissue remnants in imported chicken or duck carcases would be unlikely to be removed prior to cooking although, in the absence of any data to support this, it is assumed that some of this may be discarded as raw tissue prior to cooking and therefore accessible to backyard poultry or wild birds.

Simmons and Gray (1979) demonstrated that disease could be transmitted to poults through direct contact with an infected bird or via litter or water contaminated by an infected bird. However, disease was not transmitted when nasal mucus, faeces or a suspension of triturated nasal turbinates from clinically ill poults were inoculated into susceptible poults by the nasal or oral routes. Given this, it is reasonable to conclude that ingestion of raw scraps of meat discarded from imported chicken or duck carcases would not transmit infection.

The likelihood of exposure is assessed to be negligible.

34.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and *B. avium* is not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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35. *Mycoplasma* spp.

35.1. HAZARD IDENTIFICATION

35.1.1. Aetiological agent

Class; *Mollicutes* (also called mycoplasmas). The avian *Mollicutes* are divided into two orders: *Mycoplasmatales* (containing the genera *Mycoplasma* and *Ureaplasma*) and *Acholeplasmatales* (containing the genus *Acholeplasma)* (Nicolet 1996; Stipkovits and Kempf 1996).

To date, *M. gallinaceum, M. gallinarum, M. gallisepticum, M. glycophilum, M. iners, M. iowae, M. imitans, M. lipofaciens, M. pullorum, M. synoviae* and *U. gallorale* have been isolated from chickens (Stipkovits and Kempf 1996; Bradbury and Morrow 2008) and *M. anatis, M. cloacale, M. gallisepticum, M. glycophilum, M. imitans, M. lipofaciens, A. axanthum,* and *A. laidlawii* have been isolated from ducks (Goldberg *et al.* 1995; Stipkovits and Kempf 1996; Bradbury and Morrow 2008).

There have also been sporadic reports of "non-avian" mycoplasmas infecting chickens with or without causing clinical signs. *M. bovis* was isolated on one occasion from broiler chickens on a cattle farm (Ongor *et al.* 2008) and *M. meleagridis* was isolated from layer hens housed adjacent to a turkey-breeding operation (Khiari *et al.* 2011). These are isolated cases and the associated mycoplasma species are not considered to be hazardous to chickens (Adler 1958; Yamamoto and Bigland 1964; Yamamoto *et al.* 1965; Bradbury and Morrow 2008; Chin *et al.* 2008). No such reports of unusual transmission to ducks have been reported.

35.1.2. OIE list

Avian mycoplasmosis (*M. gallisepticum* and *M. synoviae*) is an OIE-listed disease.

35.1.3. New Zealand status

The avian mycoplasmas are not listed as notifiable organisms in New Zealand (Tana *et al.* 2011).

M. gallinaceum, M. gallinarum, M. gallisepticum, M. iners, M. synoviae and *A. laidlawii* are present in New Zealand (Black 1997; Christensen 1997; Bingham 2010; Bingham 2011). Disease surveillance in New Zealand poultry indicates that seropositivity to *M. gallisepticum* is not unusual, although clinical disease associated with this organism is rarely described, suggesting that exotic strains of *M. gallisepticum* may be more virulent than those currently present (Anonymous 1994a -1999).

M. anatis is frequently reported in healthy wild and domestic ducks throughout the world (Samuel *et al.* 1996). Wild mallards are commonly exposed to *M. anatis* without any evidence of disease and there is believed to be a high transmission rate among wild birds (Goldberg *et al.* 1995). *M. anatis* may be pathogenic to domestic ducks, causing reduced growth rates, respiratory and reproductive disorders (Tian and Gou 1991; Samuel *et al.* 1995; Goldberg *et al.* 1996). However, there are few reports of naturally-occurring disease in the field. A mycoplasma of unknown species has been isolated from a Pekin duck in New Zealand (Hemsley 1996) and given that *M. anatis* is the most commonly isolated mycoplasma species in ducks (Bencina *et al.* 1988) it is reasonable to assume that it is present in New Zealand (Manktelow *et al.* 1970; Hemsley 1996).

No records could be found of the recovery of *M. cloacale, M. glycophilum, M. imitans, M. iowae, M. lipofaciens, M. pullorum*, or *Ureaplasma* spp. from New Zealand poultry although there have been no surveys to look for these organisms.

35.1.4. Epidemiology

M. cloacale is recovered commonly in wild and domestic duck species in many countries without any associated signs of disease (Goldberg *et al.* 1995). Natural infections of chickens have not been described.

M. gallisepticum has a world-wide distribution (Levisohn and Kleven 2000) and occurs primarily in domestic and free-ranging gallinaceous birds, especially chickens and turkeys (Ley 2008). Isolation of *M. gallisepticum* from the respiratory tract of ducks with no apparent clinical signs has been reported (Benčina *et al.* 1988) and experimental infection of specific-pathogen-free (SPF) ducks with *M. gallisepticum* resulted in colonisation but only limited respiratory signs (Levisohn and Kleven 2000). The organism has also occasionally been recovered from other avian species including pheasants, chukar partridge, peafowl, Japanese quail (Reece *et al.* 1986; Cookson and Shivaprasad 1994; Murakami *et al.* 2002; Benčina *et al.* 2003), geese (Buntz *et al.* 1986), a yellow-naped Amazon parrot (Bozeman *et al.* 1984), greater flamingos and white pelicans (El-Shater 1996). No human health or zoonotic issues are associated with *M. gallisepticum* (Levisohn and Kleven 2000).

Variability occurs both within and among strains of *M. gallisepticum* (Benčina *et al.* 1994; Garcia *et al.* 1994) and chicken embryo mortality was found to be affected by strain differences (Levisohn *et al.* 1985). However, these differences did not correlate with the pathogenicity of respiratory infection *in vivo* (Levisohn *et al.* 1986).

M. gallisepticum strains may also vary in tissue tropism (Bradbury and Morrow 2008). Epithelial surfaces are the main targets, especially the trachea (Bradbury and Morrow 2008). However, transient systemic infections have been described which may result in infection at other sites (Thomas *et al.* 1966; Chin *et al.* 1991). Variant strains of *M. gallisepticum* have shown predeliction for other organs, including the cloaca (MacOwan *et al.* 1983) and the eye (Power and Jordan 1976).

Birds are susceptible to *M. gallisepticum* at any age, although clinical signs are more common in young birds (Bradbury and Morrow 2008). *M. gallisepticum* can have a range of clinical manifestations, but generally infection without complicating factors is mild or sub-clinical in chickens (Yagihashi and Tajima 1986; Levisohn and Kleven 2000). Respiratory signs are most common and other clinical presentations, including swelling of the hock and lameness, are rare (Bradbury and Morrow 2008). Subclinical infection may cause marked reduction in feed consumption efficiency and loss of production in laying birds (Clisson and Kleven 1984).

More severe disease is associated with concurrent infections or environmental stressors (Kleven 1998) and *M. gallisepticum* is frequently present in a multi-factorial disease complex (Jordan 1972; Kleven 1998) causing chronic respiratory disease in chickens (Bradbury and Morrow 2008).

The incubation period varies from 6 to 21 days depending on strain virulence (Ley 2008). Transmission of *M. gallisepticum* infection occurs vertically (*in ovo*) (Lin and Kleven 1982; Glisson and Kleven 1985; Ortiz *et al.* 1995), and horizontally by direct or indirect contact via the upper respiratory tract or conjunctiva following exposure to aerosols or droplets (Levisohn

and Kleven 2000). Spread within a flock occurs through close contact but fomites may play a role in spread between flocks (Bradbury and Morrow 2008).

M. gallisepticum can be isolated from a variety of organs, usually from the respiratory or reproductive tract (Domermuth *et al.* 1967; Amin and Jordan 1978; MacOwan *et al.* 1983; Nunoya *et al.* 1997). Experimentally it has been shown that more virulent strains can be recovered from a wide range of tissues, including the bursa of Fabricius, spleen, liver and kidney (Varley and Jordan 1978).

M. gallisepticum remains viable in chicken faeces and on cloth for up to 3 days at 20°C, in egg yolk for 18 weeks at 37°C or 6 weeks at 20°C, and in distilled water for 24 hours at both 4°C and 22°C (Ley 2008).

Although vaccines are commercially available, maintenance of freedom from infection in commercial breeding stock is the basis of most control and eradication programmes (Levisohn and Kleven 2000).

M. glycophilum is rarely reported in the literature and it has not been described as a cause of disease in chickens or ducks. It is not known whether *M. glycophilum* is pathogenic (Bradbury *et al.* 2001). However, unpublished pathogenicity studies have indicated that it may cause caecal enlargement and possible stunting of chickens (Forrest and Bradbury 1984; Loria *et al.* 2008). Natural infections of chickens resulting in disease have not been described.

M. imitans has been isolated from chickens, ducks, geese, and partridges (Ganapathy and Bradbury 1999) but its clinical significance has not yet been established (Levisohn and Kleven 2000). Natural infection of chicken or ducks has not been described and it is known that *M. imitans* alone does not produce any clinical signs or gross lesions in these species (Abdul-Wahab *et al.* 1996; Ganapathy and Bradbury 1999). Experimental infection of SPF ducks with *M. imitans* resulted in colonisation of the respiratory tract, but no pathogenic effects were reported (Levisohn and Kleven 2000). *M. imitans* has been shown to share many phenotypic and genotypic properties with *M. gallisepticum* and like *M. gallisepticum* it has the potential to act synergistically with respiratory viruses to exacerbate disease in chickens (Abdul-Wahab *et al.* 1996).

M. iowae is almost worldwide in distribution, although due to eradication efforts it is now encountered rarely in commercial poultry (Bradbury and Morrow 2008). However, recent reports have recorded cases of disease in commercial turkeys in the USA and Italy (Catania *et al.* 2012).

The natural host of *M. iowae* is the turkey (Bradbury and Kleven 2008) and it has also been found in chickens (Yoder and Hofstad 1962; Benčina *et al.* 1987a), ducks (Lo *et al.* 1994), parrots (Bozeman *et al.* 1984), geese, and wild birds including starling, cormorants, heron, wood pigeons, and a European eider (Al-Ankari and Bradbury 1996). There are many different strains of *M. iowae*, and an unusually large degree of antigenic variation among strains (Al-Ankari and Bradbury 1996).

Naturally-occurring disease appears to be restricted to turkeys with occasional reports of disease in chickens (Trampel and Goll 1994; Al-Ankari and Bradbury 1996). Experimentally *M. iowae* can cause mortality in chicken embryos, as well as stunting, poor feathering, leg abnormalities, and airsacculitis in chickens (Bradbury and Kleven 2008). Few reports describe these clinical signs in chickens under field circumstances (Bradbury and Kleven

2008). Although clinical disease may not occur in wild birds, it is thought that they may act as mechanical vectors, shedding the bacteria through their faeces (Bradbury and Morrow 2008). There are no reports of disease caused by *M. iowae* in free-ranging or commercial ducks.

Transmission of *M. iowae* is predominantly vertical (*in ovo*). Horizontal transmission can also occur although the organism does not spread rapidly (Bradbury and Kleven 2008). Unlike other avian mycoplasmas, *M. iowae* shows a resistance to bile salts and a predilection for the gastrointestinal tract (Bradbury and Kleven 2008). Repeated oral challenge of turkey poults with high dose *M. iowae* failed to induce clinical signs, but persistent shedding of the organism was observed in most birds, and at necropsy *M. iowae* was recovered from tissues of the respiratory tract, gastrointestinal tract, spleen, and kidney (Shah-Majid and Rosendal 1987). In the mature bird the oviduct, semen, cloaca, and faeces are sources of infection (Mirsalimi *et al.* 1989; Bradbury and Kleven 2008) and *M. iowae* has been shown to survive in the gastrointestinal tract for at least 3 weeks (Shah-Majid and Rosendal 1987). Little is known about the natural route of infection in chickens (Al-Ankari and Bradbury 1996). *M. iowae* has only been isolated from the respiratory tract, oviduct, and hock joint of naturally infected chickens (Yoder and Hofstad 1962; Rathore *et al.* 1979; Al-Ankari and Bradbury 1996).

M. iowae has been shown to survive for 5 days or more on feathers and at least 6 days on human hair, cotton, rubber, and straw (Christensen *et al.* 1994). *M. iowae* appears to be slightly hardier in environmental conditions than other mycoplasmas although the organism appears to be inactivated by proper cleaning and disinfection.

M. lipofaciens has been uncommonly isolated from the respiratory tract of healthy chickens, turkeys, and ducks (Priante *et al.* 2011, Bradbury *et al.* 1983, Bencina *et al.* 1987b), as well as a raptor egg and is believed to be a commensal organism. Experimental infection has caused some chicken and turkey embryo mortality (Lierz 2009, Bradbury *et al.* 1983). There are no reports of natural infections causing disease in chickens or ducks.

M. lipofaciens has been isolated from the nares of a veterinarian reporting throat pain, rhinitis and nasal pain (Lierz *et al.* 2008). Cross-reactivity to other *Mycoplasma* spp. cannot be excluded although there remains a possibility that *M. lipofaciens* can be transmitted to humans and may cause clinical symptoms (Lierz *et al.* 2008).

M. pullorum is frequently isolated from healthy birds, including chickens, quail, partridge, pheasants, turkeys, and ducks (Lo *et al.* 1994; Kleven and Ferguson-Noel 2008) and the organism is not considered to be pathogenic (Poveda *et al.* 1990). Naturally-occurring disease in chickens or ducks due to *M. pullorum* has not been described.

Ureaplasma gallorale has been recovered from healthy poultry on a number of occasions (Harasawa *et al.* 1985; Koshimizu *et al.* 1987; Priante *et al.* 2011) and very little is known about the spread of infection and pathogenicity of this organism (Stipkovits and Kempf 1996). Experimental studies have had mixed results. In one study inoculation failed to produce clinical signs or macroscopic lesions (Koshimizu *et al.* 1982) and in another, inoculation produced mild upper respiratory clinical signs with a serofibrinous airsacculitis and peritonitis (Stipkovits *et al.* 1978). There are no reports of naturally-occurring disease caused by *Ureaplasma* spp. in chickens or ducks.

35.1.5. Hazard identification conclusion

M. cloacale, M. glycophilum, M. imitans, M. lipofaciens, M. Pullorum, and *U. gallorale* have been isolated from healthy chickens or ducks and there is little evidence to suggest that they have a pathogenic role in natural infections. Where experimental infections have resulted in clinical disease, the organisms are confined to the upper respiratory tract tissues.

M. gallisepticum is found predominantly in respiratory tissues although highly virulent strains may disseminate more widely. Fresh or frozen poultry meat products produced for human consumption are not ordinarily considered risks for *M. gallisepticum* infection (Levisohn and Kleven 2000).

M. iowae has been isolated from the upper respiratory tract, oviduct, and hock joint of chickens.

Mycoplasma spp. and *Ureaplasma* spp. are identified as a potential hazard in those commodities that exclude upper respiratory tract material, reproductive tract tissues, and abdominal viscera (chicken and duck meat and meat products).

Although upper respiratory tract material, reproductive tract tissues, and abdominal viscera will be removed from chicken and duck carcases, remnants of these tissues may remain following automated processing (Veerkamp 2011). It is reported that over 95% of the upper respiratory tract tissue is removed during automatic evisceration.

M. cloacale, exotic strains of *M. gallisepticum, M. glycophilum, M imitans, M. iowae, M. lipofaciens, M. Pullorum,* and *U. gallorale* are therefore identified as a potential hazard in imported whole chicken and duck carcases.

35.2. RISK ASSESSMENT

35.2.1. Entry assessment

Mycoplasma infections are rarely associated with marked clinical signs unless accompanied by concurrent infections or environmental stressors. Subclinically infected birds are, therefore, unlikely to be detected during ante- and post-mortem inspection.

The lack of a cell wall renders mollicutes fragile in the environment (Bradbury and Morrow 2008). They are readily killed by disinfectants and do not survive for prolonged periods outside the host (Bradbury and Morrow 2008; Ley 2008). In one study, *M. gallisepticum* persisted for 7-28 days at 4°C, 7-14 days at room temperature, and less than 7 days at 30°C and 37°C (Nagatomo *et al.* 2001). It is thought that mycoplasmas may be able to exist for a longer period within animal tissues (Nagatomo *et al.* 2001). Chandiramani *et al.* (1966) recovered *M. gallisepticum* from muscle tissue of intravenously inoculated chickens for up to 49 days at 6°C, 3 days at 20°C, and less than 1 day at 37°C; and from whole carcases for up to 4 weeks when stored under conditions varying between 2°C and 24°C. In contrast, Peters *et al.* (1966) demonstrated the organism in the respiratory tract, brain, kidney, and spleen from 3 to 30 days following intra-tracheal inoculation of turkeys but was unable to isolate the organism from skeletal muscle.

Although experimental infections have been associated with widespread dissemination, *Mycoplasma* spp. and *Ureaplasma* spp. localise principally in respiratory and reproductive tissues following natural infection. As noted above, remnants of these tissues may remain following automated processing.

Considering the above, the likelihood of entry of *M. cloacale,* exotic strains of *M. gallisepticum, M. glycophilum, M imitans, M. iowae, M. lipofaciens, M. Pullorum,* or *U. gallorale* in imported whole chicken or duck carcases is assessed to be very low.

35.2.2. Exposure assessment

The growth range for a number of *Mycoplasma* spp. is described as 24°C to 42°C with rapid inactivation described at temperatures above 53°C (Mitscherlich and Marth 1984). It is therefore assessed that there is a negligible likelihood of *Mycoplasma* spp. or *Ureaplasma* spp. persisting in scraps of chicken or duck carcases following domestic cooking.

Any respiratory or reproductive tissue remnants in imported chicken or duck carcases would be unlikely to be removed prior to cooking, although in the absence of any data to support this, it is assumed that some remnants may be discarded as raw tissue prior to cooking and therefore accessible to backyard poultry or wild birds.

Horizontal transmission of *Mycoplasma* spp. occurs either through aerosol or infectious droplet transmission resulting in localised infection of the upper respiratory tract or conjunctiva, or through venereal transmission (Chin *et al.* 2008; Kleven and Ferguson-Noel 2008; Ley 2008). Additionally, *M. iowae* has been shown to spread via the oral route under experimental conditions (Shah-Majid and Rosendal 1987). The oral dose sufficient to initiate infection is not known and there are no field observations to support this as a natural pathway (Bradbury and Kleven 2008).

Fresh or frozen poultry meat products produced for human consumption are not ordinarily considered risks for *M. gallisepticum* infection (Levisohn and Kleven 2000). Goldberg *et al.* (1995) were unable to isolate any of the mycoplasmas usually associated with clinical problems in domestic poultry from wild birds and infection of wild birds with *M. iowae,* with subsequent spread to poultry, has never been reported.

Considering the above, the likelihood of exposure for *M. cloacale,* exotic strains of *M. gallisepticum, M. glycophilum, M imitans, M. iowae, M. lipofaciens, M. pullorum* and *U. gallorale* is assessed to be negligible.

35.2.3. Risk estimation

Since the exposure assessment for *M. cloacale,* exotic strains of *M. gallisepticum, M. glycophilum, M imitans, M. iowae, M. lipofaciens, M. pullorum* and *U. gallorale* is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and these organisms are not classified as risks in the commodity. Therefore, risk management measures cannot be justified.

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36. *Brachyspira* spp.

36.1. HAZARD IDENTIFICATION

36.1.1. Aetiological agent

Avian intestinal spirochaetosis is associated with colonisation of the large intestine with *Brachyspira* spp. Currently nine species of *Brachyspira* are described, with the four main pathogenic species in birds being *B. intermedia, B. pilosicoli, B. alvinipulli,* and *B. hyodysenteriae* (Hampson and Swayne 2008).

36.1.2. OIE list

Not listed.

36.1.3. New Zealand status

B. pilosicoli and *B. hyodysenteriae* have been isolated in New Zealand. Neither *B. intermedia* nor *B. alvinipulli* have been identified (Midwinter and Fairley 1999).

36.1.4. Epidemiology

Intestinal spirochaetosis has been recognised in chickens in the Netherlands (Davelaar *et al.* 1986; Dwars *et al.* 1989 – 1993), the United Kingdom (Griffiths *et al.* 1987), and elsewhere in Europe (Burch *et al.* 2006; Hampson and Swayne 2008), as well as in the United States (Swayne *et al.* 1992; Trampel *et al.* 1994), and Australia (McLaren *et al.* 1996; Stephens and Hampson 2002; Phillips *et al.* 2005; Stephens *et al.* 2005).

Most outbreaks of intestinal spirochaetosis in chickens are associated with *B. intermedia* with a smaller number due to *B. pilosicoli* (Stephens and Hampson 1999; Stephens *et al.* 2005). *B. alvinipulli* has been reported rarely in chickens and there have been no cases of *B. hyodysenteriae* in this species (Hampson and Swayne 2008).

Avian intestinal spirochaetosis is principally a disease of chickens although an outbreak associated with *B. pilosicoli* has been described in a commercial turkey flock (Shivaprasad and Duhamel 2005). No reports of outbreaks in commercial ducks have been identified.

Infection of poultry may be subclinical with no associated disease. However, clinical signs may develop from 5 days to several weeks after initial exposure (depending on the dose and other environmental factors) including diarrhoea, reduced egg production, and reduced growth rate. More severe disease, including sudden death, has been reported in rheas and geese (Nemes *et al.* 2006; Hampson and Swayne 2008).

Intestinal spirochetes colonise the caeca and rectum, but not the small intestine (Hampson and Swayne 2008). *B. pilosicoli* has been associated with spirochetaemia in humans but this has not been reported in any other species (Hampton and Swayne 2008).

36.1.5. Hazard identification conclusion

Avian intestinal spirochaetosis is principally a disease of chickens. Infectivity is confined to the lower intestinal tract, which is removed from the commodities under consideration here.

Reflecting this, the agents of avian intestinal spirochaetosis are not identified as a potential hazard.

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37. *Aegyptianella* spp.

37.1. HAZARD IDENTIFICATION

37.1.1. Aetiological agent

Aegyptianella spp. are obligate intracellular organisms in the family Anaplasmataceae (Barnes and Nolan 2008).

37.1.2. OIE list

Not listed.

37.1.3. New Zealand status

Aegyptianella spp. have not been described in New Zealand.

37.1.4. Epidemiology

Aegyptianellosis has been described in a variety of birds. *A. pullorum* may be transmitted from infected chickens to ducks, geese, and quails, and *Aegyptianella* spp. may also be transmitted from wild bird species to domestic poultry. Experimental studies have shown that erythrocytes infected with *A. pullorum* taken from chickens were unable to infect domestic turkeys although other studies have described the presence of either *A. pullorum* or *Aegyptianella*-like organisms in turkeys (Gothe 1996).

A. pullorum infections of domestic poultry have been described in countries in Africa, the Mediterranean, and the Middle East, as well as in India and Pakistan. Transmission of infection requires the presence of a tick vector of the genus *Argas* (Gothe 1996).

Following infection, *Aegyptianella* spp. parasitise erythrocytes through endocytosis. The parasite then replicates within erythrocytes through repeated binary fission which culminates in lysis of the host cell. Infection is limited to erythrocytes and no parasites can be seen in the liver, spleen, bone marrow, kidney, brain, heart, or lung by histological examination of infected poultry (Gothe 1996).

Clinical signs associated with infection include severe anaemia, ascites, heart failure due to right ventricular hypertrophy, and death (Huchzermeyer *et al.* 1987).

Aegyptianellosis does not occur in commercial broiler flocks (Huchzermeyer *et al.* 1987). The disease is found mainly in free-range poultry and is transmitted by fowl ticks in the genus *Argas* (Barnes and Nolan 2008). *Argas* spp. ticks are not found in New Zealand (McKenna 1996; Loth 2005)

37.1.5. Hazard identification conclusion

Aegyptianella spp. infections are limited to erythrocytes with no infectivity found in other body tissues and are found principally in free-range poultry and wild birds. Furthermore, transmission of *Aegyptianella* spp. requires the presence of *Argas* spp. ticks, which are not found in New Zealand. Even if these ticks were present, they would have to feed on an infected bird, rather than meat products, before they could transmit disease.

For the above reasons, *Aegyptianella* spp. are not identified as a potential hazard.

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38. *Borrelia* spp.

38.1. HAZARD IDENTIFICATION

38.1.1. Aetiological agent

Borrelia anserina causes borreliosis in a number of avian species including chickens, turkeys, pheasants, geese, and ducks (Barnes and Nolan 2008).

38.1.2. OIE list

Not listed.

38.1.3. New Zealand status

Borrelia anserina has not been described in New Zealand.

38.1.4. Epidemiology

The primary hosts of *B. anserina* are chickens, turkeys, and pheasants, although infections have also been reported in ducks, geese, grouse, and canaries (Cooper and Bickford 1993).

Clinical signs in infected birds include cyanosis, pallor of the comb and wattles, ruffled feathers, dehydration, inactivity, and anorexia, and may progress to paralysis and coma. Infection with strains of low virulence may result in mild or inapparent clinical signs (Cooper and Bickford 1993).

Borreliosis leads to an acute septicaemia characterised by variable morbidity and high mortality (Barnes and Nolan 2008), with typical post-mortem findings including a pronounced splenomegaly, hepatomegaly, renal enlargement, severe diarrhoea, and anaemia (Rivetz *et al.* 1977).

B. anserina is not resistant outside the host (Barnes and Nolan 2008) although infected blood kept at 4°C and -18°C for 4 weeks was able to transmit infection when experimentally inoculated into susceptible chickens (Bok *et al.* 1975).

Natural transmission of infection requires the presence of *Argas* spp. ticks, which act as the disease reservoir and primary vector (Barnes and Nolan 2008). *Argas* spp. ticks are not found in New Zealand (McKenna 1996; Loth 2005)

Historically, when the poultry industry in a number of countries comprised several small enterprises with poor sanitation, borreliosis was considered to be a severe disease affecting the industry. However, the disease is now confined to small flocks kept for subsistence or very limited local sale where the tick vector remains established (Ataliba *et al.* 2007; Lisbôa *et al.* 2009).

38.1.5. Hazard identification conclusion

Borreliosis is no longer considered to be a disease of commercial poultry farming. Furthermore, transmission of *B. anserina* requires the presence of *Argas* spp. ticks, which are not found in New Zealand. Even if these ticks were present, they would have to feed on an infected bird, rather than meat products, before they could transmit disease.

For the above reasons, *B. anserina* is not identified as a potential hazard.

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39. *Coenonia anatina*

39.1. HAZARD IDENTIFICATION

39.1.1. Aetiological agent

The only member of the genus *Coenonia*. A *Riemerella anatipestifer*-like organism (Vandamme *et al.* 1999).

39.1.2. OIE list

Not listed.

39.1.3. New Zealand status

No records describe the recovery of *C. anatina* in New Zealand. However, as noted in Chapter 32, anatipestifer syndrome of ducklings due to an organism tentatively classified as *Pasteurella anatipestifer* has been described (Anonymous 1974; Hemsley 1996). A histopathological diagnosis was recorded in 1990 when paralysis of ducks was found to be accompanied by a spectacular meningoencephalitis typical of this organism (Orr 1990).

39.1.4. Epidemiology

Isolates of *C. anatina* have been recovered from Pekin ducks, Muscovy ducks, and geese (Vandamme *et al.* 1999).

There is very little information describing the epidemiology of *C. anatina* infection although this organism can be considered closely related to both *Ornithobacterium* spp. and *R. anatipestifer* (Vandamme *et al.* 1999). These organisms have been considered previously (Chapters 32 and 33).

Post-mortem findings following infection with *R. anatipestifer* are typically those of acute or chronic septicaemia, characterised by fibrinous pericarditis, perihepatitis, airsaccultitis, and meningitis (Helfer and Helmboldt 1977; Smith *et al.* 1987). In addition, infection can lead to cellulitis with thickening of the skin on the ventral abdomen accompanied by tracks of caseous pus between the dermis and underlying musculature which may be barely noticeable on gross examination (Gooderham 2002).

The trachea, lungs, and air sacs are considered the best tissues from which to isolate *O. rhinotracheale* from infected birds. Following experimental infection, the organism has also been recovered from blood, liver, joints, brain, ovary, and oviduct, although field trials have been unsuccessful in recovering *O. rhinotracheale* from heart blood and liver (Chin *et al.* 2008).

Transmission of *R. anatipestifer* is considered to occur via the respiratory route or through skin wounds although an arthropod vector (*Culex* mosquitoes) has been suggested for turkeys in California (Cooper 1989).

39.1.5. Hazard identification conclusion

It is recognised that infection with either *R. anatipestifer* or *O. rhinotracheale* may be accompanied by marked clinical signs in live birds and significant post mortem pathology.

However, birds exhibiting less marked clinical signs may go undetected during ante-mortem and post-mortem inspection. It is assumed that this also applies to *C. anatina*.

Although *R. anatipestifer* should be considered likely to be present in New Zealand, no isolates of this organism have been recorded since 1974 and there is no record of *C. anatina* being decribed here.

C. anatina is therefore identified as a potential hazard in imported duck meat

39.2. RISK ASSESSMENT

39.2.1. Entry assessment

Broth cultures of *R. anatipestifer* remain viable for 2-3 weeks if stored at 4°C (Bangun *et al.* 1981; Sandhu 2008). It is reasonable to extrapolate from this that the likelihood of entry for *C. anatina* should be assessed as non-negligible.

39.2.2. Exposure assessment

It is considered unlikely that *R. anatipestifer* would remain viable after processing duck meat to a core temperature exceeding 60°C for 30 minutes and reaching 80°C for at least 10 minutes (MAF 2006) and *R. anatipestifer* is inactivated at 60°C after 1 hour (Harry and Deb 1979). Therefore it is assumed that there is a negligible likelihood of *C. anatina* persisting in scraps of duck meat following domestic cooking.

Studies cited in Chapter 31 of this risk analysis (Hendrickson and Hilbert 1932; Graham *et al.* 1938; Asplin 1956; Hatfield and Morris 1988; Sarver *et al.* 2005) have demonstrated that there is a negligible likelihood of *R. anatipestifer* being transmitted to susceptible species through the ingestion of uncooked meat scraps. Similarly, as discussed in Chapter 32, there is no evidence for the spread of *O. rhinotracheale* other than by the respiratory route.

Considering the above evidence, there is considered to be a negligible likelihood of *C. anatina* being transmitted to susceptible species through the ingestion of uncooked duck meat scraps. The likelihood of exposure is assessed to be negligible.

39.2.3. Risk estimation

Since the exposure assessment for *C. anatina* is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimation is negligible and this organism is not classified as a risk in the commodity. Therefore, risk management measures cannot be justified.

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40. Long-segmented filamentous organisms

40.1. HAZARD IDENTIFICATION

40.1.1. Aetiological agent

Long-segmented filamentous organisms (LSFOs) are Gram-positive, anaerobic, spore-forming bacteria found in the ileum and jejunum of poultry (Barnes and Nolan 2008). *Candidatus arthromitus* has been proposed as a name for this group of organisms (Snel at al 1995).

40.1.2. OIE list

Not listed.

40.1.3. New Zealand status

No reports have been found of LSFO infections in New Zealand poultry.

40.1.4. Epidemiology

LSFOs attach to the intestinal epithelium, embed in the apical cytoplasm of enterocytes, replace microvilli, and produce a strong stimulation of the mucosal immune system (Yamauchi and Snel 2000).

A retrospective study associated LSFO infections with a range of clinical signs in chickens including diarrhoea, death, and loss of production, although the authors did not propose a cause-and-effect relationship but rather that LSFOs were either normal flora, commensal organisms that overgrow when certain gastrointestinal events occur, or pathogens. The authors of this study also commented that LSFOs were seen in all segments of the small intestine but were never seen in the caecum or colon, in other portions of the gastrointestinal tract, or in other organs (Goodwin *et al.* 1991)

40.1.5. Hazard identification conclusion

The pathogenic role of LSFOs is unclear. It is likely that these organisms may not be pathogens but overgrowths associated with enteric disease (Goodwin *et al.* 1991; Barnes and Nolan 2008).

Nevertheless, LSFOs have only been identified in the small intestine which is removed from the commodities under consideration here. Therefore, LSFOs are not identified as a potential hazard.

References

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41. *Planococcus* spp.

41.1. HAZARD IDENTIFICATION

41.1.1. Aetiological agent

Planococcus halophilus, a motile Gram-negative coccus, normally associated with the marine environment (Barnes and Nolan 2008).

41.1.2. OIE list

Not listed.

41.1.3. New Zealand status

No reports of *P. halophilus* being recovered in New Zealand have been identified.

41.1.4. Epidemiology

There is one report of *P. halophilus* being associated with an outbreak of necrotic hepatitis in chickens in Pakistan (Abdel Gabbar *et al.* 1995). Pure cultures of *P. halophilus* were recovered from necrotic foci in the livers of affected 43-week-old-layers. The source of the organisms was postulated to be contamination of feed (fish meal and other marine by-products), with the high ambient temperature at the time (46°C) possibly also contributing to this outbreak.

41.1.5. Hazard identification conclusion

There has been only one reported case of *P. halophilus* infection of chickens, associated with extreme environmental conditions and feed contamination. Affected birds were reported to have marked liver lesions at necropsy.

Because there have been no further reports of poultry infections with this organism and the imported commodity will have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate effective Good Manufacturing Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes, *P. halophilus* is not identified as a potential hazard.

References

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42. *Chlamydia psittaci*

42.1. HAZARD IDENTIFICATION

42.1.1. Aetiological agent

Obligate intracellular Gram-negative bacteria, *Chlamydia psittaci* (Andersen and Vanrompay 2008).

42.1.2. OIE list

Listed.

42.1.3. New Zealand status

Psittacosis was first described in 66 imported Australian parrots in 1954, with diarrhoea, listlessness, and death affecting at least 31 birds (Cairney 1954). Laboratory investigations between 1984 and 1985 identified *C. psittaci* isolates from budgerigars, parakeets, pigeons, rosellas, and cockatiels (Bell and Schroeder 1986). Psittacosis is considered to be prevalent in New Zealand wild pigeons, with a prevalence rate of between 9.5% and 25% (Motha *et al.* 1995).

An unpublished survey of faecal samples from captive and wild endangered and threatened avian species was reported by Motha *et al.* (1995) to have detected *C. psittaci* in a number of species, including kakapo, takahe, and kiwi. However, a subsequent survey found no evidence of psittacosis in native psittacines with perhaps the exception of kakas and wekas on Kapiti Island and it was suggested that the earlier unpublished findings were likely to have been false positive results due to the choice of test (Motha *et al.* 1995).

42.1.4. Epidemiology

C. psittaci serovars can be distinguished in specialised laboratories by a panel of serovar-specific monoclonal antibodies (Andersen 1991; Andersen 1997). Restriction fragment length polymorphism analysis and genotyping techniques are also available to distinguish serovars (Vanrompay *et al.* 1997; Geens *et al.* 2005). There are 8 known serovars of *C. psittaci*, with serotypes C and E being associated with ducks.

Chlamydiosis in ducks is usually a severe disease, with morbidity between 10% and 80%, with up to 30% mortality (Andersen and Vanrompay 2008) although some outbreaks may be associated with few clinical signs (Arzey *et al.* 1990; Newman *et al.* 1992; Hinton *et al.* 1993).

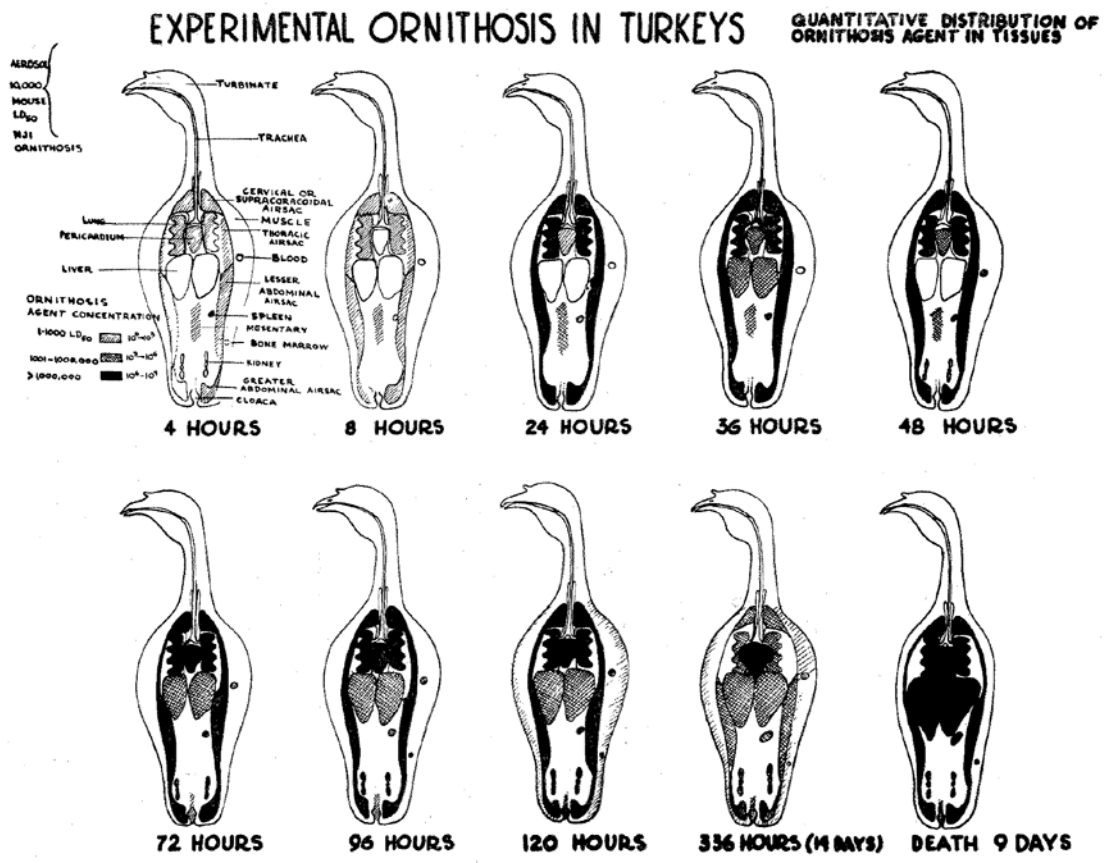
Chlamydiosis in commercial chickens has been described in both broilers (Barr *et al.* 1986) and layers (Arzey and Arzey 1990), although epidemiologic and laboratory evidence indicates that chickens are relatively resistant to infection (Andersen and Vanrompay 2008). Reports describe conjunctivitis, nasal exudation, and tracheitis in infected individuals.

Transmission of *C. psittaci* occurs through inhalation of contaminated material, with large numbers of chlamydiae found in the respiratory tract exudate and faeces of infected birds (Andersen 1996). Page (1959) was unable to transmit infection following oral inoculation of turkeys using a *C. psittaci* dose of 340,000 mouse LD_{50}. Transmission via arthropod vectors has also been suggested (Eddie *et al.* 1962; Page *et al.* 1975) and there is evidence for limited

vertical transmission (Lublin *et al.* 1996). *C. psittaci* is an obligate intracellular organism that has been described as an "energy parasite" as it depends on the host cell for adenosine triphosphate (ATP) and other high-energy metabolites (Moulder 1991).

Following experimental inoculation of turkeys with four strains of chlamydiae, primary replication was found to occur throughout the respiratory tract after 2-7 days, with subsequent replication occurring throughout the intestinal tract, especially in the jejunum, caecum, and colon (Vanrompay *et al.* 1995). An earlier study (Page 1959) quantified the tissue distribution of *C. psittaci* in turkeys following aerosol exposure and found that the organism multiplied primarily in the lungs, air sac system and pericardium, although infectivity was also detected in other tissues (including the kidneys) and in muscle tissue after 120 hours. These findings are illustrated below in Figure 2.

Figure 2: Distribution of *C psittaci* in turkey tissues following aerosol exposure (Page 1959).



For diagnostic purposes, the best tissues to recover the organism are the air sacs, spleen, pericardium, heart, liver, and kidney (Andersen and Vanrompay 2008) and proper handling using a transport medium is necessary to prevent loss of infectivity (Spencer and Johnson 1983).

42.1.5. Hazard identification conclusion

It is not known which serovars of *C. psittaci* are present in New Zealand. Infectivity is concentrated in the respiratory tissues and intestinal tract although some infectivity can be detected in muscle and renal tissues. Infective tissue is therefore likely to remain following evisceration.

Infection with a highly virulent strain would be likely to result in carcase condemnation, although slaughterhouse inspection might be unlikely to detect birds infected with less virulent strains or birds in the early stages of infection.

Reflecting the above, exotic strains of *C. psittaci* are identified as a potential hazard in the commodity.

42.2. RISK ASSESSMENT

42.2.1. Entry assessment

Meat after rigor is usually between pH 5.4 and pH 5.6 because of the conversion of muscle glycogen to lactic acid. The ultimate pH of uncooked poultry meat can be expected to be within the range 5.7 to 6.0 (Fletcher *et al.* 2000). The optimal pH for the survival of rickettsiae is 7.0 (Bovarnick *et al.* 1950), and the pH range for the growth of *C. psittaci* is limited to 6.5 to 7.5 (Mitscherlich and Marth 1984). *C. psittaci* would be unlikely to survive in the normal pH range of poultry meat.

Furthermore, *C. psittaci* is an obligate intracellular organism depends on the host cell for ATP and other high-energy metabolites (Moulder 1991) and the recovery of the organism for diagnostic purposes requires proper handling using a transport medium to prevent loss of infectivity (Spencer and Johnson 1983).

As the disease does not spread by the oral route, infection would not establish even if infected raw tissues were consumed by a susceptible host (MAF 1999). Chicken meat is not considered to pose a risk of transmission of *C. psittaci* to susceptible animals (DAFF 2001).

Reflecting the above, the likelihood of entry of viable *C. psittaci* is assessed to be negligible.

42.2.2. Risk estimation

Since the entry assessment for *C. psittaci* is negligible, under the methodology used in this risk analysis (see Section 5.3) the risk estimate is negligible and *C. psittaci* is not classified as a risk in imported chicken and duck meat. Therefore, risk management measures cannot be justified.

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43. Nematodes, acanthocephalans

43.1. HAZARD IDENTIFICATION

43.1.1. Aetiological agent

The nematodes and acanthocephalans that have been identified in chickens and ducks are summarised below in Table 7.

43.1.2. OIE list

Not listed.

43.1.3. New Zealand status

McKenna (1998) described the nematode species recognised in New Zealand birds. *Ascaridia galli, Capillaria annulata, Capillaria caudinflata, Capillaria obsignata, Heterakis gallinarum, Heterakis vesicularis*, and *Syngamus trachea* were recorded in domestic fowl and *Amidostomum acutum, Capillaria anatis, Contracaecum microcephalum, Echinuria Australis, Echinuria uncinata, Porrocaecum crassum*, and *Trichostrongylus tenuis* were recorded in various duck species. No acanthocephalan parasites were described in domestic poultry or ducks.

43.1.4. Epidemiology

Avian nematodes often have a broad host range and 25 families of nematode have been described from nine orders in avian species (Yazwinski and Tucker 2008).

Poultry nematodes have either a direct or indirect life cycle, with approximately half the known species requiring an invertebrate intermediate host. Acanthocephalans (thorny-headed worms) live in the intestinal tract of vertebrates and all require an intermediate host to complete their life cycle.

Regardless of species, the eggs of all female nematodes and acanthocephalans enter the environment in faeces.

Few parasites are considered important in modern commercial poultry although they remain a concern in small free-range flocks and commercial game-birds (McDougald 2008).

The nematodes and acanthocephalans that have been described in chickens and ducks are summarised below in Table 7.

Table 7. Nematodes, acanthocephalans of chickens and/or ducks (Yazwinski, Tucker 2008).

Parasite	Location	Intermediate host	Pathogenicity	Species
Upper digestive tract				
Capillaria annulata	Mucosa of oesophagus and crop	Earthworms	Inflammation of crop and oesophageal walls with masses of worms found in sloughing tissue	Chicken
Capillaria contorta	Mucosa of oesophagus, crop, and sometimes mouth.	None	Inflammation and thickening of the crop and oesophagus. May invade mouth and upper oesophagus in heavy infestations	Chicken, Duck
Gongylonema ingluvicola	Mucosa of crop, sometimes oesophagus and proventriculus	Beetle and cockroaches	Limited local lesions in crop mucosa	Chicken
Cymea colini	Proventriculus wall at junction with gizzard	Cockroach	Little or no pathology associated with this parasite	Chicken experimentally
Dispharynx nasuta	Proventriculus wall, sometimes in oesophagus, rarely in the small intestine	Pillbug and sowbug	Proventricular ulceration and thickening with parasites concealed beneath the proliferating tissue.	Chicken
Tetrameres americana	Proventriculus wall	Grasshoppers and cockroach	Thickening of proventricular wall	Chicken, Duck
Tetrameres confusa	Proventriculus	Unknown	Not described	Chicken
Tetrameres crami	Proventriculus	Amphipods	Not described	Duck
Tetrameres fissispina	Proventriculus	Amphipods, grasshoppers, earthworms, and cockroaches	Considerable tissue reaction with degeneration, oedema and leukocyte infiltration	Chicken, Duck
Echinura uncinata	Esophagus, gizzard, proventriculus, small intestine	Water flea	Mortality without clinical signs or chronic infection leading to proventricular nodules and emaciation	Duck
Cheilospirura hamulosa	Cardiac and/or pyloric regions of the gizzard	Grasshoppers, beetles, weevils, and sandhoppers	Heavy infestations may be associated with damage to the gizzard wall	Chicken
Amidostomum anseris	Gizzard	None	Heavy infestations may be associated with damage to the gizzard wall	Duck
Amidostomum skrjabini	Gizzard	None	Clinical disease outbreaks in young ducks	Duck (Chicken experimentally)
Epomidiostomum uncinatum	Gizzard	None	Not described	Chicken, Duck
Streptocara crassicauda	Gizzard	Amphipod	Not described	Chicken, Duck

Table 7 (continued).

Parasite	Location	Intermediate host	Pathogenicity	Species
Small intestine				
Ascaridia galli	Small intestine. Also found in the oesophagus, crop, gizzard, body cavity, oviduct and egg due to aberrant migration	None	Weight loss and intestinal blockage in heavy infestations	Chicken, Duck
Capillaria obsignata	Small intestine	None	Catarrhal exudate and thickening of intestinal wall associated with heavy infestations	Chicken
Capillaria caudinflata	Small intestine mucosa	Earthworms	Not described	Chicken, Duck
Capillaria bursata	Small intestine mucosa	None	Not described	Chicken
Capillaria anatis	Small intestine and caecum	Unknown	Not described	Chicken, Duck
Porrocaecum ensicaudatum	Small intestine	Earthworm	Not described	Chicken, Duck
Caecum				
Heterakis dispar	Caecum	None	Relatively nonpathogenic	Duck
Heterakis gallinarum	Caecum	None	Inflammation and thickening of the caecal walls. Carrier of *Histomonas meleagridis*	Chicken, Duck
Heterakis isolonche	Caecum	None	Diarrhoea and weight loss are common. Caecal wall nodules.	Chicken, Duck
Subulura brumpti	Caecum	Beetles or cockroaches	None decribed	Chicken, Duck
Subulura strongylina	Caecum	Beetles or cockroaches	No noticeable lesions	Chicken
Subulura suctoria	Caecum and small intestine	Beetles	No noticeable lesions	Chicken
Strongyloides avium	Caecum and small intestine	None	Marked thickening of caecal wall	Chicken
Trichostrongylus tenuis	Caecum and small intestine	None	Caecal distension and congestion with thickening of caecal wall. Anaemia associated with heavy infestation	Chicken, Duck

Table 7 (continued).

Parasite	Location	Intermediate host	Pathogenicity	Species
Respiratory tract				
Cyathostoma bronchialis	Larynx, trachea, bronchi and abdominal air sacs	Earthworms may act as paratenic hosts but not necessary	Hyperplasia of bronchial epithelium, pneumonitis and respiratory distress	Chicken, Duck
Syngamus trachea	Trachea, bronchi and bronchioles	Earthworms, slugs and snails may act as paratenic hosts but not necessary	Obstruction of tracheal lumen may lead to suffocation	Chicken
Other tissues				
Oxyspirura petrowi	Eye	Unknown	Ophthalmia, possibly progressing to destruction of eyeball.	Chicken
Oxyspirura mansoni	Eye	Cockroach	Ophthalmia, possibly progressing to destruction of eyeball.	Chicken, Duck
Cardiofilaria nilesi	Body cavity	Mosquito	Not described	Chicken

43.1.5. Hazard identification conclusion

A wide range of nematodes and acanthocephalans of chickens and ducks could be considered exotic to New Zealand. However, as the eggs of all these parasites are deposited in the faeces of infested birds and the intestinal tract is removed from all commodities considered here, nematodes and acanthocephalans are not identified as a potential hazard.

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44. Cestodes, trematodes

44.1. HAZARD IDENTIFICATION

44.1.1. Aetiological agent

Tapeworms (Class: *Cestoda*) and flukes (Class: *Trematoda*). The cestode and trematode parasites of chickens identified by Taylor *et al.* (2007) are shown below in Table 8. The cestode and trematode parasites of ducks identified by Taylor *et al.* (2007) are shown below in Table 9.

Table 8. Chicken cestode, trematode parasites (Taylor *et al.* 2007).

Parasite	Location	Intermediate host	Pathogenicity
Cestodes			
Amoebataenia sphenoides	Small intestine	Earthworms	Low pathogenic significance
Choanotaenia infundibulum	Small intestine	Housefly, beetles, and grasshoppers	None described
Cotugnia digonopora	Small intestine	Unknown	None described
Davainea proglottina	Small intestine	Gastropod molluscs	The most pathogenic of the poultry cestodes. Scolex penetrates deeply in the duodenal wall resulting in haemorrhagic enteritis. Heavy infestations may be fatal
Fimbriaria fasciolaris	Small intestine	Copepods (*Cyclops* and *Diaptomus* spp.)	None described
Hymenolepis cantaniana	Small intestine	Beetles	None described
Hymenolepis carioca	Small intestine	Dung and flour beetles, sometimes *Stomoxys* spp.	Considered to be of low pathogenicity
Metroliasthes lucida	Small intestine	Grasshoppers	None described
Raillietina cesticillus	Small intestine	Various genera of beetles	Heavy infestations associated with catarrhal enteritis. Birds may show a reduced growth rate, emaciation, and weakness
Raillietina echinobothrida	Small intestine	Ants of the genera *Pheidole* and *Tetramorium*	Hyperplastic enteritis at the site of attachment may result in caseous nodules in the intestinal wall
Raillietina teragona	Small intestine	Ants of the genera *Pheidole* and *Tetramorium*	Large caseous nodules may form at the site of attachment in the wall of the small intestine
Trematodes			
Brachylaemus commutatus	Caecum	Land snails	Large numbers may be associated with enteritis and diarrhoea

Catatropis verrucosa	Caecum	Snails	Large numbers may be associated with enteritis and diarrhoea
Collyriclum faba	Skin	Snails and dragonfly nymphs	Hard subcutaneous cysts (3-10mm diameter) found around cloaca and may also be present along the thorax, abdomen, beak, and neck in heavy infestations
Echinoparyphium recurvatum	Small intestine	Snails, frogs, freshwater clams, and mussels	Heavy infections may cause weakness, anaemia and emaciation
Hypoderaeum conoideum	Small intestine	Snails, frogs, freshwater clams, and mussels	Large numbers may be associated with enteritis and diarrhoea
Notocotylus attenuatus	Caecum, rectum	Snails	Large numbers may be associated with enteritis and diarrhoea
Plagiorchis arcuatus	Rectum and oviduct	Snails, crustacea, molluscs, and insects	Light infestations affect egg production, heavy infestations can be fatal
Postharmostomum commutatum	Caecum	Snails	Large numbers may be associated with enteritis and diarrhoea
Prosthogonimus macrorchis	Cloacal bursa and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal
Prosthogonimus ovatus	Rectum and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal
Prosthogonimus pellucidus	Large intestine and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal

Table 9. Duck cestode, trematode parasites (Taylor *et al.* 2007).

Parasite	Location	Intermediate host	Pathogenicity
Cestodes			
Fimbriaria fasciolaris	Small intestine	Copepods (*Cyclops* and *Diaptomus* spp.)	None described
Hymenolepis lanceolata	Small intestine	Aquatic copepod crustaceans	Moderate to heavy infections may cause a catarrhal enteritis and necrosis of the mucosa
Trematodes			
Bilharziella polonica	Mesenteric and pelvic veins	Snails	Generally considered non-pathogenic.
Catatropis verrucosa	Caecum	Snails	Large numbers may be associated with enteritis and diarrhoea
Echinoparyphium recurvatum	Small intestine	Snails, frogs, freshwater clams, and mussels	Heavy infections may cause weakness, anaemia and emaciation
Echinostoma revolutum	Caecum and rectum	Snails	Large numbers may be associated with enteritis and diarrhoea

Filicollis anatis	Small intestine	Isopods	Male worms cause localised nodules at the point of attachment. Females may rupture the peritoneum
Hypoderaeum conoideum	Small intestine	Snails, frogs, freshwater clams, and mussels	Large numbers may be associated with enteritis and diarrhoea
Hyptiasmus tumidus	Nasal and orbital sinuses	Not described	Infection causes nasal catarrh
Notocotylus attenuatus	Caecum, rectum	Snails	Large numbers may be associated with enteritis and diarrhoea
Polymorphus boschadis	Small intestine	*Gammarus pulex*, fresh water shrimp, and crayfish	Causes inflammation of the intestinal mucosa with localised haemorrhage. Heavy infection can induce anaemia
Prosthogonimus macrorchis	Cloacal bursa and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal
Prosthogonimus pellucidus	Large intestine and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal
Typhlocoelum cucumerinum	Trachea, air sacs, oesophagus	Snails	Infected birds may show dyspnoea and asphyxia
Typhlocoelum cymbium	Trachea, bronchi	Snails	Tracheal obstruction may lead to asphyxia

44.1.2. OIE list

Not listed.

44.1.3. New Zealand status

From the above lists, McKenna (2010) identified the cestodes *Choanotaenia infundibulum, Davainea proglottina, Echinolepis (Hymenolepis) carioca, Fimbriaria fasciolaris,* and *Raillietina cesticillus* and the trematodes *Catatropis sp., Echinoparyphium recurvatum, Echinostoma revolutum, Hypoderaeum conoideum, Notocotylus attenuatus,* and *Typhlocoelum cucumerinum* in New Zealand.

44.1.4. Epidemiology

Cestode infestation is associated with free-range rearing or backyard flocks and is rare in intensively reared poultry. The usual sites for adult cestode attachment are the duodenum, jejunum and ileum, and gravid proglottids are shed daily from adult worms in the intestinal tract. All cestodes require an intermediate host to complete their life cycle. Various insects, crustaceans, earthworms, slugs, snails, and leeches have all been identified as intermediate hosts for different tapeworm species (McDougald 2008).

Trematode parasites require a mollusc intermediate host to complete their life cycle and some species also require the presence of a second intermediate host. Adult flukes continually shed eggs in the faeces of their hosts and these must develop in intermediate hosts before being ingested by another host (McDougald 2008).

With the exception of *Collyriclum faba*, all cestodes and trematodes associated with chickens and ducks deposit their eggs in the faeces of infected birds.

Immature *Collyriclum faba* migrate to the subcutaneous tissue of infected birds where they form cysts and pass eggs into the environment through an opening in the cyst wall. These then complete their lifecycle by passing through snails then dragonfly larvae. This parasite is only found in birds with access to marshy areas (Taylor *et al.* 2007).

44.1.5. Hazard identification conclusion

The intestinal tract is removed from all commodities considered here. Commercially reared chickens would be unlikely to be raised in wet marshy areas required for the persistence of the trematode *Collyriclum faba*. Cestodes and trematodes are not identified as a potential hazard.

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45. *Eimeria* spp.

45.1. HAZARD IDENTIFICATION

45.1.1. Aetiological agent

Intracellular parasites. Phylum *Apicomplexa*; Genus *Eimeria. Eimeria acervulina, E. brunetti, E. hagani, E. maxima, E. mitis, E. mivati, E. necatrix, E. praecox, and E. tenella* are associated with chickens. *Eimeria* spp., *Wenyonella* spp., and *Tyzzeria* spp. are recognised in ducks (McDougald and Fitz-Coy 2008). Co-infection with two or more species is common (McDougald *et al.* 1986).

45.1.2. OIE list

Not Listed.

45.1.3. New Zealand status

E. acervulina, E. brunetti, E. maxima, E. necatrix, and *E. tenella* are recognised in New Zealand (McKenna 1998).

45.1.4. Epidemiology

Coccidian oocysts are ingested and release sporozoites in the small intestine. These enter either epithelial cells or intraepithelial lymphocytes. The protozoan parasite then multiplies within the intestinal tract, undergoing at least two generations of asexual development before the sexual phase of the life cycle which results in the formation of oocysts which are then shed in the faeces (McDougald and Fitz-Coy 2008).

The location of the parasite within the intestinal tract and the degree of tissue damage varies between species, with the characteristic gross pathology being used to identify individual species. However, biochemical and molecular tools may also be used to distinguish *Eimeria* species (Shirley 1986; Tsuji *et al.* 1997).

The damage caused to the intestinal tract by coccidiosis can allow colonisation by secondary bacteria such as *Clostridium perfringens* (Hembolt and Bryant 1971; McDougald and Hu 2001) or *Salmonella* Typhimurium (Arakawa *et al.* 1981; Baba *et al.* 1982), and may also exacerbate other diseases such as histomoniasis (McDougald and Hu 2001).

Eimeria acervulina is commonly found in commercial poultry throughout the world including New Zealand (McKenna 1998; McDougald and Fitz-Coy 2008). Heavy infections may result in mortality although light to moderate infections may be clinically inapparent. In light infections gross lesions (mucosal thickening with watery mucoid content) are limited to the duodenum although these may extend into the small intestine in heavy infections. Histopathologically, gametocytes can be seen in the mucosal cells lining the villi.

E. brunetti is also recognised to be present in New Zealand (McKenna 1998). Infections usually localise to the lower small intestine but may extend from the gizzard to the cloaca and into the caeca in severe cases. Infection can produce moderate mortality, weight loss and poor feed conversion. Grossly, infection results in thickening of the intestinal mucosa, with severe infections leading to necrosis and erosion over the entire mucosa. Histopathology reveals merozoites in the intestinal epithelium (McDougald and Fitz-Coy 2008).

E. hagani infections localise to the upper small intestine, with parasites found in the villous tips. *E. hagani* is considered to be a moderately pathogenic species, with infection producing haemorrhagic spots, catarrhal inflammation, engorged capillary beds, and watery intestinal content (McDougald and Fitz-Coy 2008).

E. maxima is recognised to be present in New Zealand (McKenna 1998). Infections localise to the mid-small intestine although heavy infections may extend throughout the small intestine. This species is considered to be moderately-highly pathogenic, with infections causing poor weight gain, diarrhoea, and mortality. Asexual reproduction is limited to the epithelial cells of the mucosa although sexual reproduction occurs in deeper tissues, leading to congestion, oedema, cellular infiltration, and thickening of the mucosa. Grossly, the intestine may become flaccid and filled with fluid (McDougald and Fitz-Coy 2008).

E. mitis is found in the lower small intestine where it causes indistinct lesions which are often overlooked. Schizonts and gametocytes are superficial in the intestinal mucosa (McDougald and Fitz-Coy 2008).

Infection with *E. mivati* can cause reduced weight gain and morbidity with lesions seen in the duodenum, midgut, and lower small intestine although heavy infections may extend to the caeca and cloaca. Histopathologically, the parasite can be found in the mucosal cells of the villi of the small intestine (McDougald and Fitz-Coy 2008).

E. necatrix is recognised to be present in New Zealand (McKenna 1998). This species (along with *E. tenella*) is considered to be the most pathogenic found in chickens, causing severe weight loss, morbidity, and mortality. Lesions can extend from the ventriculus-gizzard junction to the ileo-caecal junction. Histopathologically, clusters of schizonts can be seen in the submucosal and lamina propria, with lesions extending through the gut muscle layers to near the serosal membranes (McDougald and Fitz-Coy 2008).

E. praecox is not associated with prominent lesions in infected chickens although heavy infections may cause reduced weight gain, dehydration, and poor feed conversion. Infections are usually confined to the duodenal loop with the parasite present in the epithelial cells on the sides of the villi (McDougald and Fitz-Coy 2008).

E. tenella is recognised to be present in New Zealand (McKenna 1998). This species infects the caeca and causes severe disease with high morbidity and mortality. Schizonts develop in the lamina propria so the mucosa and blood vessels are disrupted when merozoites are released (McDougald and Fitz-Coy 2008).

Coccidiosis is seen sporadically in ducks and may lead to significant morbidity and mortality. Thirteen species of coccidia have been reported in domestic and wild ducks (McDougald *et al.* 1987). Infected birds may exhibit anorexia, weight loss, weakness, distress, and up to 70% mortality. Either bloody or cheesy exudate may be seen at necropsy with sloughing of the intestinal epithelium. The infecting parasite may invade the mucosal and submucosal layers of the gut as deep as the muscular layers (McDougald and Fitz-Coy 2008).

45.1.5. Hazard identification conclusion

A wide range of coccidian parasites of chickens and ducks could be considered exotic to New Zealand. However, as the oocysts of all these parasites are deposited in the faeces of infected birds and the intestinal tract is removed from all commodities considered here, *Eimeria* spp. are not identified as a potential hazard.

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46. *Cochlosoma anatis*

46.1. HAZARD IDENTIFICATION

46.1.1. Aetiological agent

Cochlosoma anatis is a flagellate protozoan initially identified in the European domestic duck (Bermudez 2008).

46.1.2. OIE list

Not listed.

46.1.3. New Zealand status

No record of *C. anatis* infection in New Zealand could be found.

46.1.4. Epidemiology

C. anatis was previously considered non-pathogenic but recent reports suggest it should be considered a significant pathogen of both turkeys and ducks (Cooper *et al.* 1995; Bollinger and Barker 1996; Bermudez 2008). Oral transmission of *C. anatis* to turkeys, chickens, and quail has been demonstrated experimentally (Lindsay *et al.* 1999) and houseflies have also been implicated in transmission of the organism from the environment to a susceptible host (McElroy *et al.* 2005).

Natural infection of domestic ducks has been described in the caecum, colon, and lower ileum (Kimura 1934; Travis 1938; Watkins *et al.* 1989). Following experimental infection of day-old ducklings, *C. anatis* was found in the jejunum and colon (Bollinger and Barker 1996).

Infection has been associated with swelling of the intestinal wall and a catarrhal enteritis, although some studies have described no pathogenic changes in birds following infection (Travis 1938). Bollinger *et al.* (1996) found that *C. anatis* infection increased intestinal villus length and altered mucosal enzyme concentrations in ducklings.

C. anatis can be found throughout the intestinal tract of young poults and localises in the region of the caecal tonsil in adult birds (McNeil and Hinshaw 1942). Histologically, large numbers of *C. anatis* can be found in the lumen and within the intervillous spaces of the duodenum and jejunum. Most of the parasites are free within the gut lumen although some appear to be firmly attached to the intestinal epithelium (Cooper *et al.* 1995).

This parasite is often found during the investigation of cases of hexamitiasis in turkeys (McNeil and Hinshaw 1942; Campbell 1945), although *C. anatis* has been described as the cause of natural outbreaks of diarrhoea and enteritis in turkeys (Cooper *et al.* 1995).

Experimental studies suggest that *C. anatis* alone may have some pathogenic potential although greatest effect is seen when *C. anatis* infections occur alongside other enteric pathogens (Bermudez 2008).

46.1.5. Hazard identification conclusion

The pathogenic role of *C. anatis* is not completely understood. Nevertheless, *C. anatis* is only found in the gut lumen and (to a lesser degree) attached to the intestinal epithelium. As the intestinal tract is removed from the commodities under consideration here, *C. anatis* is not identified as a potential hazard.

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47. *Leucocytozoon* spp.

47.1. HAZARD IDENTIFICATION

47.1.1. Aetiological agent

Phylum *Apicomplexa*; Order *Haemospororina*; Family *Plasmodiidae*; Genus *Leucocytozoon* (Bermudez 2008). At least 67 valid species are recognised, with 66 of these found in birds (Hsu *et al.* 1973).

47.1.2. OIE list

Not listed.

47.1.3. New Zealand status

Leucocytozoon fringillinarum, *L. tawaki*, and *Leucocytozoon* sp. have been described in New Zealand in a chaffinch, a Fiordland crested penguin, and a yellow-eyed penguin respectively (McKenna 2010).

47.1.4. Epidemiology

Leucocytozoon spp. have a two host life cycle. Sporogeny occurs in insects (*Simuliid* flies and *Culicoid* midges), sporozoites then migrate to the salivary glands of the insect. Following infection of the vertebrate host schizongeny takes place in the internal organs (e.g. liver, brain, spleen, and lungs).

Outbreaks of leucocytozoonosis have been recorded in chickens, turkeys, and other domestic poultry species (Adams *et al.* 1987; Dick 1978; Yu *et al.* 2000).

Leucocytozoon simondi (also described as *L. anatis*) is associated with domestic ducks and a range of other wild duck species in the United States (Bennett and Cameron 1974; Bennett *et al.* 1975; Hsu *et al.* 1973). This species has not been associated with chickens. Known vectors of *L. simondi* include *Simulium venustum*, *S. croxtoni*, *S. euradminiculum*, and *S. rugglesi* (Bermudez 2008). *L. simondii* has been associated with heavy mortality in domestic ducks and geese in eastern North America. Infected birds display lethargy, inappetance, diarrhoea, laboured breathing, and death. Infection leads to anaemia and tissue damage due to the widespread secondary megaloschizonts and histopathology of infected individuals reveals hypertrophy, congestion and haemosiderosis in the liver and spleen, together with necrotic foci in the liver (Fallis *et al.* 1974). However, experimental infection of wild ducks has also been shown to cause no mortality or difference in growth rate (Shutler *et al.* 1996; Shutler *et al.* 1999).

Leucocytozoon caulleryi has been described in chickens in southern and eastern Asia. Outbreaks occur frequently in Japan, with summer epizootics causing death in growing chicks and reduced egg production in hens (Miura *et al.* 1973). *L. caulleryi* has also been recognised in the United States (Noblet *et al.* 1976). The vectors of *L. caulleryi* are considered to be *Culicoides arakawa*, *C. circumscriptus*, and *C. odibilis* (Fallis *et al.* 1974). In the vertebrate host, schizonts are found in the lung, spleen and thymus (Hsu *et al.* 1973) and megaloschizonts (which may be visible on gross examination) can be found throughout the carcase, including the liver, spleen, kidneys, pancreas, heart, lungs, proventriculus,

ventriculus, intestines, and brain. Disease outbreaks are characterised by peritoneal, perirenal and subdural haemorrhage (Goto *et al.* 1966).

Leucocytozoon sabrezi is recorded in chickens in Southeast Asia, the insect vector is unknown (Bermudez 2008). *Leucocytozoon schoutedeni* is found in chickens in East Africa (Dick 1978), with *Simulium* flies recognised as the insect vector.

47.1.5. Hazard identification conclusion

The only way arthropod vectors can be infected with *Leucocytozoon* spp. is by sucking blood as they do not feed on meat and cannot be infected from meat. Tissue-based parasites are unlikely to be transmitted in chicken or duck meat because of their complex life cycles requiring specific hosts (Biosecurity Australia 2008). *Leucocytozoon* spp. are not identified as a potential hazard.

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48. *Plasmodium* spp. (avian malaria)

48.1. HAZARD IDENTIFICATION

48.1.1. Aetiological agent

Phylum: Apicomplexa, Genus: *Plasmodium*.

48.1.2. OIE list

Not listed.

48.1.3. New Zealand status

Plasmodium cathemerium, *P. elongatum*, *P. relictum*, and *Plasmodium* sp. have been identified in New Zealand birds (McKenna 2010)

48.1.4. Epidemiology

All species of *Plasmodium* are transmitted by mosquitoes. Around 65 species of *Plasmodium* have been described although the species considered specific for domestic fowl are found mostly in Asia, Africa, and South America. Natural parasites of domestic hens and ducks are *P. gallinaceum*, *P. juxtanucleare*, *P. lophurae*, and *P. fallax.* Experimentally, *P. relictum*, *P. elongatum*, *P. cathemerium*, and *P. circumflexum* have been transmitted to domestic poultry (Bermudez 2008).

Avian plasmodia develop in mosquitoes of the genera *Culex, Aedes* and (rarely) *Anopheles*. Gametocytes develop into infective sporozoites in the mosquito host and are transmitted at feeding. Subsequent development in the avian host can lead to heavy parasitaemias. Infected birds may show no clinical signs or infection can lead to sudden death or severe anaemia with up to 90% mortality (Bermudez 2008).

48.1.5. Hazard identification conclusion

The life cycle of *Plasmodium* spp. depends on host/mosquito interaction and the only way arthropod vectors can be infected is by sucking blood as they do not feed on meat and cannot be infected from meat. Avian malaria is not identified as a potential hazard.

References

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49. *Haemoproteus* spp.

49.1. HAZARD IDENTIFICATION

49.1.1. Aetiological agent

Family: *Plasmodiidae*, Genus: *Haemoproteus*. Over 128 species of *Haemoproteus* have been identified in birds, with the overwhelming majority of *Haemoproteus* spp. associated with wild bird species (Levine and Campbell 1971; Bermudez 2008).

49.1.2. OIE list

Not listed.

49.1.3. New Zealand status

Haemoproteus danilewsky and *Haemoproteus* sp. have been identified in New Zealand (McKenna 2010). Neither of these isolates were associated with commercial poultry.

49.1.4. Epidemiology

Transmission of *Haemoproteus* spp. occurs by biting dipteras of the families *Hippoboscidae* and *Ceratopogonidae*. Sporogeny occurs in the arthropod host then, following infection, merozoites develop in the vascular endothelium of the vertebrate host which subsequently invade erythrocytes and mature into gametocytes. Further development of the gametocytes requires ingestion by a suitable arthropod host in a blood meal (Bermudez 2008).

These protozoa appear to have a commensal relationship with many of their hosts and are pathogenic only under special circumstances. Lameness and respiratory distress followed by death has been described following the infection of domestic ducks with *Haemoproteus* spp. At post-mortem, it was noted that in affected individuals that large numbers of schizonts were causing congestion and oedema due to mechanical interference with the circulation (Julian and Galt 1980).

49.1.5. Hazard identification conclusion

As with *Leucocytozoon* spp. and *Plasmodium* spp., the only way arthropod vectors can be infected with *Haemoproteus* spp. is by sucking blood as they do not feed on meat and cannot be infected from meat. Tissue-based parasites are unlikely to be transmitted in chicken or duck meat because of their complex life cycles requiring specific hosts. *Haemoproteus* spp. are not identified as a potential hazard.

References

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50. *Sarcocystis* spp.

50.1. HAZARD IDENTIFICATION

50.1.1. Aetiological agent

An apicomplexan protozoan of the genus *Sarcocystis* (Bermudez 2008).

50.1.2. OIE list

Not listed.

50.1.3. New Zealand status

Sarcocysts are reported on a regular basis from routine bovine slaughterhouse submissions for the *Taenia saginata* surveillance scheme, and are also seen in sheep and occasionally feral pigs in New Zealand (Bingham 2007). No records of sarcocysts in poultry were identified.

50.1.4. Epidemiology

Avian sarcocystosis has a worldwide distribution but is considered rare in domestic chickens although an incidence of up to 40% has been reported in ducks (Bermudez 2008). *Sarcocystis horvathi* (*S. gallinarum*) is associated with chickens whereas *S. rileyi* (*Balbiani rileyi, S. anatina*) is associated with ducks (Levine 1986).

Sarcocysts have an obligatory two-host lifecycle (Odening 1998), with sexual reproduction occurring in the predator (definitive) host, followed by faecal shedding and subsequent asexual reproduction in the prey (intermediate) host. Sarcocysts are usually found in the skeletal muscle of infected birds and lesions (described as *rice breast disease*) are readily apparent in infected individuals (Tuggle and Friend 1999). Heavy infections may lead to clinical signs (Bermudez 2008).

Cohabitation of young ducks with ducks infected with *S. rileyi* or administration of *S. rileyi* cystozoites to young ducks by oral, intramuscular or intravenous inoculation did not result in infection, demonstrating the need for a definitive host to complete the life cycle. Modern poultry production systems prevent the occurrence of sarcocystosis as the avian intermediate host is not exposed to the oocyst-contaminated excreta of the definitive host (Bermudez 2008). The predator-prey relationship between the intermediate bird host and the definitive carnivore host is thought to be the primary reason why juvenile birds are seldom found to be infected (Tuggle and Friend 1999).

50.1.5. Hazard identification conclusion

Modern commercial poultry production systems prevent poultry intermediate hosts from being exposed to the definitive host of *Sarcocystis* spp. Heavy infections with sarcocysts are normally picked up at routine meat inspection and freezing reduces the viability of sarcocyts during storage and transport (MacDiarmid 1991).

Therefore, *Sarcocystis* spp. are not identified as a potential hazard in the commodity.

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