

***Import risk analysis: Turkey  
meat***

***FINAL***

**March 2011**

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MAF Biosecurity New Zealand  
Pastoral House  
25 The Terrace  
PO Box 2526  
Wellington 6011  
New Zealand

Tel: 64 4 894 0100  
Fax: 64 4 894 0731

Policy and Risk  
MAF Biosecurity New Zealand



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# Contributors to this risk analysis

## 1. Author

Stephen Cobb	Senior Adviser, Risk Analysis (Animal Kingdom)	Biosecurity New Zealand, Wellington
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## 2. Internal Peer Review

Stuart MacDiarmid	Principal International Adviser, Risk Analysis	Biosecurity New Zealand, Wellington
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Howard Pharo	Team Manager, Risk Analysis	Biosecurity New Zealand, Wellington
--------------	-----------------------------	-------------------------------------

Sandy Toy	Senior Adviser, Risk Analysis	Biosecurity New Zealand, Wellington
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Aurelie Castinel	Senior Adviser, Animal Response	Biosecurity New Zealand, Wellington
------------------	---------------------------------	-------------------------------------

## 3. External Scientific Review

Kerry Mulqueen	Veterinary Consultant	Nominated by the Poultry Industry Association of New Zealand
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Neil Christensen	Veterinary Surgeon	Avivet Ltd., Palmerston North, New Zealand
------------------	--------------------	--

David Buckley	Manager, Avian & Non-Ruminants, Animal Biosecurity	The Department of Agriculture, Fisheries and Forestry, Australia
---------------	--	--

David Swayne	Laboratory Director, Southeast Poultry Research Laboratory	USDA/Agricultural Research Service, Athens, Georgia
--------------	--	---

Yehia Mohamed Saif (Chapter 9 only)	Professor/Program Head of Food Animal Health	Food Animal Health Research Programme, The Ohio State University, Ohio
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## Executive Summary

The risks associated with the importation of chilled or frozen meat and meat products derived from turkeys (*Meleagris gallopavo gallopava*) have been examined.

From an initial list of 111 organisms/groups of organisms possibly associated with turkeys, a preliminary hazard list identified 40 organisms/groups of organisms that required further consideration.

Of these 40 preliminary hazards, 15 were considered to be potential hazards in imported whole turkey carcasses and 7 of these were considered to be potential hazards in imports limited to turkey meat. The greater number of potential hazards associated with entire turkey carcasses reflects the likely inefficiencies associated with eviscerators and lung removal machinery used during automated processing of commercial turkeys.

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

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

For imported entire carcasses, risk management measures have also been presented for the following hazards:

- Avian paramyxovirus-2 and -3
- Turkey coronavirus
- The aetiologic agent of turkey viral hepatitis

The assessment and management of risks associated with the consumption of imported food is the responsibility of New Zealand Food Safety Authority (NZFSA). NZFSA is accountable for administering the Food Act 1981 and other food safety legislation which applies to all food imported and sold in New Zealand. Imports of turkey products will be required to meet the requirements of food safety legislation in addition to any biosecurity requirements. NZFSA will evaluate food safety risks associated with imported turkey products and make appropriate risk management decisions.

# 1. Introduction

The development of an import health standard (IHS) for turkey meat from the European Union (EU) has been identified as a priority for MAFBNZ. In view of the highly specific focus of MAF's previous import risk analysis (MAF 1999), it was recommended that an import risk analysis for uncooked turkey meat be drafted to support the development of this IHS. As there was little benefit from limiting the proposed import risk analysis to product derived solely from the EU member states, this analysis examines the risks associated with turkey meat and meat products from all countries.

## 2. Scope

This risk analysis is limited to the description of the risks due to disease-causing organisms associated with the importation of turkey meat.

The assessment and management of human health risks associated with the consumption of imported food is the responsibility of New Zealand Food Safety Authority (NZFSA). NZFSA is accountable for administering the Food Act 1981 and other food safety legislation which applies to all food imported and sold in New Zealand. Imports of turkey products will be required to meet the requirements of food safety legislation in addition to any biosecurity requirements. NZFSA will evaluate food safety risks associated with imported turkey products and make appropriate risk management decisions.

The risk analysis is qualitative.

## 3. Commodity definition

The commodity considered in this import risk analysis is defined as chilled or frozen meat and meat products derived from turkeys (*Meleagris gallopavo gallopava*) that have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate effective Good Management Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes.

The commodities covered in this risk analysis are:

1. whole turkey carcasses<sup>1</sup>; uncooked, unskinned, eviscerated, not containing giblets;
2. bone-in turkey products such as wings or legs;
3. boneless turkey meat products such as breasts, boned-out thighs;
4. reconstituted turkey meat products comprised of turkey meat and skin.

## 4. Risk analysis methodology

The methodology used in this risk analysis follows the guidelines as described in *Biosecurity New Zealand Risk Analysis Procedures – Version 1<sup>2</sup>* and in Section 2 of the *Terrestrial Animal Health Code* of the World Organisation for Animal Health (OIE 2009).

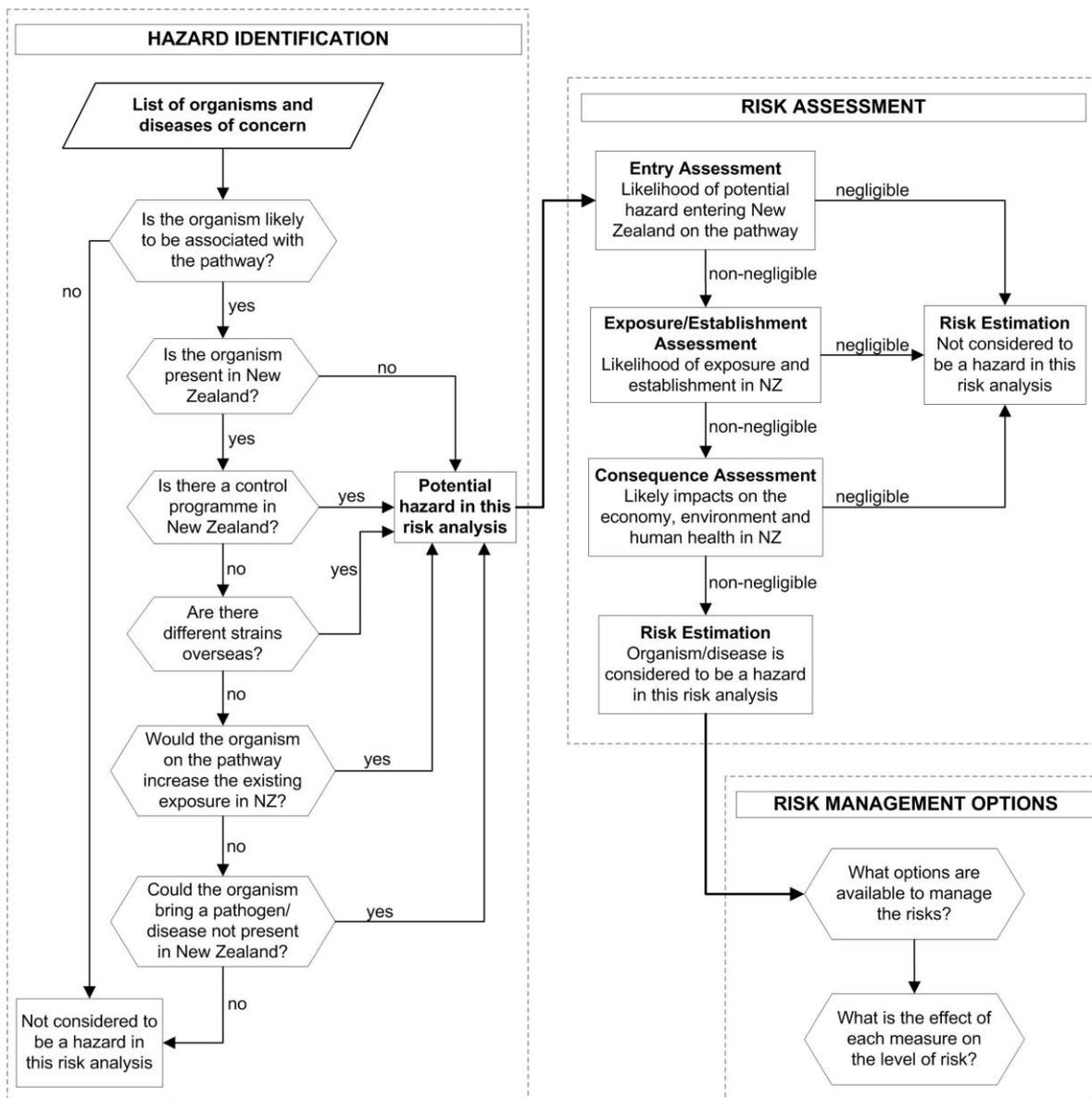
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<sup>1</sup> Including head-and-feet-on carcasses

<sup>2</sup> See [www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf](http://www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf).

The risk analysis process used by the MAF is summarised in Figure 1.

**Figure 1. The risk analysis process**



## 4.1. PRELIMINARY HAZARD LIST

The first step in the risk analysis is hazard identification. The process begins with the collation of a list of organisms that might be associated with turkey meat (the preliminary hazard list). The diseases/agents of interest are those that could be transmitted in turkey meat or meat products and could infect domestic, feral, or wild animals that occur in New Zealand, and man. In this case the preliminary hazard list was compiled from *Diseases of Poultry*, 12<sup>th</sup> 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 considered potential hazards and all organisms considered to be potential hazards are subjected to risk assessment.

**Table 1. List of organisms and diseases of concern**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration
<b>Viral diseases</b>					
Newcastle disease	Avian paramyxovirus serogroup 1 (APMV-1)	Yes	Exotic strains	Yes	Yes
Avian paramyxoviruses 2-9	APMV-2 to 9	No	Exotic serogroups/strains	Yes	Yes
Turkey rhinotracheitis (TRT), swollen head syndrome (SHS), and avian rhinotracheitis (ART)	Avian metapneumovirus	Yes	Exotic	Yes	Yes
Infectious bronchitis	Infectious bronchitis virus (IBV)	Yes	Exotic strains	No	No
IBV is primarily an infection of chickens, with no recorded cases of natural infection in turkeys (Cavanagh 2005). Turkey coronavirus infections are addressed below.					
Laryngotracheitis	Laryngotracheitis virus (LTV)	Yes	Present	N/A	No
Experimental infection of turkeys with LTV has been reported (Winterfield and So 1968) and natural infection of this species with LTV has been recently described (Portz et al 2008). LTV is considered to be present in New Zealand (Howell 1992).					
Influenza	Influenzavirus A	Yes	Exotic serogroups/strains	Yes	Yes
Infectious bursal disease	Infectious bursal disease virus (IBDV)	Yes	Exotic	Yes	Yes
Chicken infectious anaemia	Chicken infectious anaemia virus	No	Present	N/A	No
Chicken infectious anaemia virus is regarded as present in New Zealand (Anonymous 2005).					
Group I adenovirus infections	Fowl adenovirus (FAdV)	No	Some species of <i>Aviadenovirus</i> genus present	Yes	Yes
FAdV-1, 8, and 12 are recognised as present in New Zealand (Saifuddin 1990).					

**Table 1 (continued)**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration
<b>Viral diseases (continued)</b>					
Egg drop syndrome (EDS)	EDS virus	No	Present	N/A	No
EDS is recognised in New Zealand (Howell 1992). Disease outbreaks mostly occur in laying hens although ducks and geese are also thought to be natural hosts of the virus. There is no evidence of naturally occurring infection of turkeys (Adair and Smyth 2008).					
Haemorrhagic enteritis (HE)	HE virus (HEV)	No	Exotic	Yes	Yes
Quail bronchitis (QB)	QB virus	No	Exotic	No	No
Experimental infection of turkeys has been described (Reed and Jack 2008). No reports of natural infections of turkeys could be found.					
Pox	Turkey pox virus	No	Present	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	No
Reovirus (viral arthritis) is recognised in New Zealand (Howell 1992).					
Other reovirus infections	Avian reoviruses	No	Exotic	Yes	Yes
The pathogenic role of other reoviruses in turkey diseases is unclear (Jones 2008). However, reoviruses have been associated with poult enteritis and mortality syndrome (PEMS) (Heggen-Peay et al 2002) so further consideration is considered appropriate here.					
Turkey coronavirus enteritis	Turkey coronavirus	No	Exotic	Yes	Yes
Rotavirus infection	Rotavirus	No	Present	N/A	No
Rotaviruses have been described in New Zealand poultry (Saifuddin et al 1989).					
Astrovirus infection	Turkey astrovirus (TAsTV)	No	Exotic	Yes	Yes
Avian enterovirus-like infection	Enterovirus-like viruses	No	Exotic	Yes	Yes
Turkey viral hepatitis considered separately below.					
Turkey torovirus infection / stunting syndrome	Turkey torovirus	No	Exotic	Yes	Yes
Duck hepatitis	Duck hepatitis virus (DHV) types 1, 2, and 3	Yes	Exotic	No	No
Although experimental infection of turkeys with DHV-1 has been described, natural infection of turkeys with DHV-1,2, or 3 has not been reported (Woolcock 2008).					
Duck virus enteritis (DVE)	Duck enteritis virus	No	Exotic	No	No
Natural susceptibility to DVE is limited to members of the family <i>Anatidae</i> (ducks, geese, and swans) (Sandhu and Metwally 2008).					
Haemorrhagic nephritis enteritis of geese	Goose haemorrhagic polyomavirus (GHPV)	No	Exotic	No	No
Natural infection with GHPV has only been described in geese (Guerin 2008).					
Derzsy's disease	Goose parvovirus	No	Exotic	No	No
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	No
Avian nephritis viruses types 1-3 are recognised as present in New Zealand (Howell 1992).					

**Table 1 (continued)**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration
<b>Viral diseases (continued)</b>					
Arbovirus infections	Eastern equine encephalitis (EEE) virus Western equine encephalitis (WEE) virus Highlands J (HJ) virus Israel turkey meningoencephalitis (IT) virus West Nile (WN) virus	EEE, WEE, and WN listed	Exotic	Yes	Yes
Turkey viral hepatitis	Aetiology thought to be a picornavirus	No	Exotic	Yes	Yes
Avian encephalomyelitis	Avian encephalomyelitis virus	No	Present	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	No	No
Although experimental infection of turkeys has been described, natural HEV infections are limited to chickens (Meng et al 2008).					
Marek's disease	Marek's disease virus	Yes	Exotic strains?	Yes	Yes
Leukosis/sarcoma group	Members of the alpharetrovirus family	No	Exotic strains	No	No
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).					
Reticuloendotheliosis	Reticuloendotheliosis virus	No	Present	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	Unknown	No	No
Disease is most commonly seen in broiler chickens and occasionally in older chickens (Hafner and Goodwin 2008a).					
Multicentric histiocytosis	Unknown	No	Unknown	No	No
Disease is described as a condition of young broiler chickens (Hafner and Goodwin 2008b).					
<b>Bacterial diseases</b>					
Pullorum disease and fowl typhoid	<i>Salmonella</i> Gallinarum-Pullorum	Yes	Exotic	Yes	Yes
Paratyphoid infections	<i>Salmonella</i> spp.	No	Some members exotic	Yes	Yes
Arizonosis	<i>Salmonella</i> arizonae serovar 18Z <sub>4</sub> Z <sub>32</sub>	No	Exotic	Yes	Yes

**Table 1 (continued)**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration
<b>Bacterial diseases (continued)</b>					
Campylobacteriosis	<i>Campylobacter jejuni</i> and others	No	Exotic strains?	Yes	Yes
Colibacillosis	Avian pathogenic <i>Escherichia coli</i> (APEC)	No	Exotic strains	Yes	Yes
Fowl cholera	<i>Pasteurella multocida</i>	Yes	Exotic strains?	Yes	Yes
Fowl cholera due to <i>P. multocida</i> 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 <i>P. multocida</i> in ducks and commercial poultry (Anonymous 2000; Bingham 2006). Diagnostic laboratories have recovered <i>P. multocida</i> from cases of fowl cholera in chickens, turkeys, and quail (Orr 2000; Varney 2004c; Varney 2007). However, it has been suggested that exotic strains of this organism may be more virulent than those present in this country (Mulqueen 2009).					
<i>Riemerella anatipestifer</i> infection	<i>Riemerella anatipestifer</i>	No	Exotic?	Yes	Yes
<i>Ornithobacterium rhinotracheale</i> infection	<i>Ornithobacterium rhinotracheale</i>	No	Exotic	Yes	Yes
Bordetellosis (turkey coryza)	<i>Bordetella avium</i>	No	Exotic	Yes	Yes
Infectious coryza	<i>Avibacterium paragallinarum</i>	No	Exotic	No	No
Turkeys are refractory to experimental infection with <i>Avibacterium paragallinarum</i> (Blackall and Soriano 2008).					
<i>Mycoplasma gallisepticum</i> infection	<i>Mycoplasma gallisepticum</i>	Yes	Exotic strains?	Yes	Yes
<i>Mycoplasma gallisepticum</i> is considered present in New Zealand (Black 1997). However, it has been suggested that exotic strains of this organism may be more virulent than those present in this country (Christensen 2010).					
<i>Mycoplasma meleagridis</i> infection	<i>Mycoplasma meleagridis</i>	No	Exotic	Yes	Yes
<i>Mycoplasma synoviae</i> infection	<i>Mycoplasma synoviae</i>	Yes	Present	N/A	No
<i>Mycoplasma synoviae</i> is considered present in New Zealand (Black 1997).					
<i>Mycoplasma iowae</i> infection	<i>Mycoplasma iowae</i>	No	Exotic	Yes	Yes
Other mycoplasma infections	<i>Mycoplasma</i> spp.	No	Exotic?	Yes	Yes
<i>Mycoplasma imitans</i> , <i>Mycoplasma gallinarum</i> , <i>Mycoplasma pullorum</i> , and <i>Ureaplasma</i> spp. have been found in association with turkeys (Kleven and Ferguson-Noel 2008).					
Ulcerative enteritis (quail disease)	<i>Clostridium colinum</i>	No	Present	N/A	No
Ulcerative enteritis recognised in New Zealand (Black 1997).					
Necrotic enteritis	<i>Clostridium perfringens</i> type A and type C	No	Present	N/A	No
Necrotic enteritis recognised in New Zealand (Black 1997).					
Botulism	<i>Clostridium botulinum</i>	No	Present	N/A	No
Surveillance of wild birds has confirmed the presence of botulism in New Zealand (Alley 2002a).					

**Table 1 (continued)**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration
<b>Bacterial diseases (continued)</b>					
Gangrenous dermatitis	<i>Clostridium perfringens</i> type A, <i>Clostridium septicum</i> , <i>Staphylococcus aureus</i>	No	Present	N/A	No
Gangrenous dermatitis recognised in New Zealand (Black 1997).					
Staphylococcosis	<i>Staphylococcus aureus</i> and <i>Staphylococcus</i> spp.	No	Present	N/A	No
Staphylococcosis recognised in New Zealand (Black 1997).					
Streptococcosis	<i>Streptococcus</i> spp.	No	Present	N/A	No
Streptococcosis recognised in New Zealand (Black 1997).					
Enterococcosis	<i>Enterococcus</i> spp.	No	Present	N/A	No
Enterococcosis recognised in New Zealand (Black 1997).					
Erysipelas	<i>Erysipelothrix rhusiopathiae</i>	No	Present	N/A	No
Erysipelas recognised in New Zealand (Black 1997; Alley 2002a)					
Avian intestinal spirochaetosis	<i>Brachyspira</i> spp.	No	Exotic	Yes	Yes
Tuberculosis	<i>Mycobacterium avium</i>	No	Exotic strains?	Yes	Yes
Other bacterial diseases	<i>Acinetobacter</i> spp.	No	Present (Varney 2005)	N/A	No
	<i>Actinobacillus</i> / <i>Gallibacterium</i> spp.	No	Present (Wilson 2002)	N/A	No
	<i>Arcanobacterium pyogenes</i>	No	Present (Varney 2004a)	N/A	No
	<i>Aegyptianella</i> spp.	No	Exotic	Yes	Yes
	<i>Aerobacter aerogenes</i>	No	Present (Spiller 1964)	N/A	No
	<i>Aeromonas</i> spp.	No	Present (Julian et al 2002)	N/A	No
	<i>Arcobacter</i> spp.	No	Present (McFadden et al 2005)	N/A	No
	<i>Bacillus</i> spp.	No	Present (Wraight 2003)	N/A	No
	<i>Bacteroides</i> spp.	No	Present (McDougall 2005)	N/A	No
	<i>Borrelia</i> spp.	No	Exotic	Yes	Yes
	<i>Citrobacter</i> spp.	No	Present (Julian et al 2002)	N/A	No
<i>Coenonia anatine</i>	No	Exotic	No	No	
<i>Coenonia anatine</i> causes an exudative septicaemia in ducks and geese (Vandamme et al 1999).					

**Table 1 (continued)**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration	
<b>Bacterial diseases (continued)</b>						
Other bacterial diseases (continued)	<i>Enterobacter</i> spp.	No	Present (Thompson 1999)	N/A	No	
	<i>Flavobacterium</i> spp.	No	Present (Ubiquitous – Quinn et al 1994)	N/A	No	
	<i>Hafnia</i> spp.	No	Present (Gartrell et al 2007)	N/A	No	
	<i>Helicobacter</i> spp.	No	Present (Varney and Gibson 2006)	N/A	No	
	<i>Klebsiella</i> spp.	No	Present (Varney 2004b)	N/A	No	
	<i>Lactococcus</i> spp.	No	Present (Stone 2005)	N/A	No	
	<i>Lawsonia intracellularis</i>	No	Present (Smits et al 2002)	N/A	No	
	<i>Listeria monocytogenes</i>	No	Present (Varney 2005)	N/A	No	
	Long-segmented filamentous organisms (LSFOs)	No	Unknown	Yes	Yes	
	<i>Moraxella</i> spp.	No	Present (Vermunt and Parkinson 2000)	N/A	No	
	<i>Mycobacterium avium</i> subsp. <i>paratuberculosis</i>	Yes	Exotic strains	No	No	
	Natural infections of poultry with <i>Mycobacterium avium</i> subsp. <i>paratuberculosis</i> have not been reported (Barnes and Nolan 2008).					
	<i>Neisseria</i> spp.	No	Present (Alley 2002b)	N/A	No	
	<i>Nocardia</i> spp.	No	Present (Orchard 1979)	N/A	No	
	<i>Oerskovia</i> spp.	No	Unknown	No	No	
	Infections with <i>Oerskovia</i> spp. recorded in pigeons (Barnes and Nolan 2008).					
<i>Pelistega</i> spp.	No	Unknown	No	No		
<i>Pelistega europaea</i> is a newly described bacterium associated with respiratory disease in pigeons (Barnes and Nolan 2008).						
<i>Peptostreptococcus</i> spp.	No	Present (Graham 1998)	N/A	No		
<i>Planococcus</i> spp.	No	Exotic	No	No		
<i>Planococcus</i> spp. are usually associated with the marine environment. <i>P. halophilus</i> has been associated with multifocal hepatic necrosis in a layer flock (Abdel Gabbar et al 1995) but no reports suggesting an association with turkeys have been found.						
<i>Plesiomonas</i> spp.	No	Present (Staples 2000)	N/A	No		

**Table 1 (continued)**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration
<b>Bacterial diseases (continued)</b>					
Other bacterial diseases (continued)	<i>Proteus</i> spp.	No	Present (Orr 1995)	N/A	No
	<i>Pseudomonas</i> spp.	No	Present (Coats 1998)	N/A	No
	<i>Rothia</i> spp.	No	Present (Thompson 1999)	N/A	No
	<i>Streptobacillus moniliformis</i>	No	Present (Sakalkale et al 2007)	N/A	No
	<i>Vibrio</i> spp.	No	Present (Staples 2000)	N/A	No
Avian chlamydiosis	<i>Chlamydophila psittaci</i>	Yes	Exotic strains?	Yes	Yes
<b>Fungal diseases</b>					
Aspergillosis	<i>Aspergillus</i> spp.	No	Present	N/A	No
<i>Aspergillus</i> spp. (including <i>Aspergillus fumigatus</i> ) are listed as organisms known to be present in New Zealand on the NZFUNGI database (see: <a href="http://nzfungi.landcareresearch.co.nz/html/mycology.asp">http://nzfungi.landcareresearch.co.nz/html/mycology.asp</a> ).					
Candidiasis (thrush)	<i>Candida</i> spp.	No	Present	N/A	No
Candidiasis is recognised in New Zealand (McCausland 1972).					
Dermatophytosis (favus)	<i>Microsporium gallinae</i>	No	Exotic?	Yes	Yes
Dactylariosis	<i>Dactylaria gallopava</i>	No	Present	N/A	No
Although clinical dactylariosis has not been reported in New Zealand, <i>Dactylaria gallopava</i> is an environmental fungal organism which causes sporadic opportunistic infections and is found in New Zealand (see <a href="http://nzfungi.landcareresearch.co.nz/html/mycology.asp">http://nzfungi.landcareresearch.co.nz/html/mycology.asp</a> ).					
Histoplasmosis	<i>Histoplasma capsulatum</i>	No	Exotic	Yes	Yes
Cryptococcosis	<i>Cryptococcus neoformans</i>	No	Present	N/A	No
<i>Cryptococcus neoformans</i> is recognised in New Zealand (Varney 2005).					
Zygomycosis (Phycomycosis)	Fungi belonging to the genera <i>Mucor</i> , <i>Rhizopus</i> , <i>Absidia</i> , <i>Rhizomucor</i> , and <i>Mortierella</i> .	No	Present	N/A	No
All genera are recognised in New Zealand (see <a href="http://nzfungi.landcareresearch.co.nz/html/mycology.asp">http://nzfungi.landcareresearch.co.nz/html/mycology.asp</a> ).					
Macrorhabdosis (Megabacteria)	<i>Macrorhabdus ornithogaster</i>	No	Present	N/A	No
Megabacteriosis has been described in New Zealand (Christensen et al 1997).					
<b>Parasitic diseases</b>					
Nematodes and acanthocephalans	Various	No	Some exotic	Yes	Yes
Cestodes and trematodes	Various	No	Some exotic	Yes	Yes

**Table 1 (continued)**

Disease	Agent	OIE notifiable	NZ status	Disease associated with turkeys?	Requires further consideration
<b>Parasitic diseases (continued)</b>					
Coccidiosis	<i>Eimeria meleagrimitis</i> , <i>E. adenoides</i> , <i>E. melegridis</i> , <i>E. dispersa</i> , <i>E. gallopavonis</i>	No	Present	Yes	No
Coccidiosis recognised in New Zealand turkeys (Black 1997).					
Cryptosporidiosis	<i>Cryptosporidium baileyi</i> , <i>C. meleagridis</i>	No	Present	N/A	No
Cryptosporidiosis has been described in New Zealand poultry (Anonymous 1999).					
<i>Cochlosoma anatis</i> infection	<i>Cochlosoma anatis</i>	No	Exotic	Yes	Yes
Histomoniasis (blackhead)	<i>Histomonas meleagridis</i>	No	Present	N/A	No
Histomoniasis described in New Zealand (Black 1997).					
Trichomoniasis	<i>Trichomonas gallinae</i>	No	Present	N/A	No
Canker due to <i>Trichomonas gallinae</i> is considered common in New Zealand (Anonymous 1975).					
Hexamita	<i>Spironucleus meleagridis</i>	No	Exotic?	Yes	Yes

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

For any organism, if the answer to question one is “yes” (and the answer to question 2 is “yes” in the cases of organisms requiring a vector) and the answers to either questions three or four are “yes”, it is classified 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.

If importation of the commodity is considered likely to result in an increased exposure of people to a potentially zoonotic organism already present in New Zealand, then that organism is also considered to be a potential hazard.

### 4.3. RISK ASSESSMENT

In line with the MAF Biosecurity New Zealand 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 potential hazard.
- c) Consequence assessment - the consequences of entry, establishment or spread of the organism.
- 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 hazard.

It is important to note that all of the above steps may not be necessary in all risk assessments. The MAF Biosecurity New Zealand 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.

### 4.4. RISK MANAGEMENT

For each organism classified as a hazard, 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 hazard, 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 hazards. 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 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).

## 4.5. RISK COMMUNICATION

MAF releases draft import risk analyses for a six-week period of public consultation to verify the scientific basis of the risk assessment and to seek stakeholder comment on the risk management options presented. Stakeholders are also invited to present alternative risk management options that they consider necessary or preferable.

Following public consultation on the draft risk analysis, MAF produces a review of submissions and determines whether any changes need to be made to the draft risk analysis as a result of public consultation, in order to make it a final risk analysis.

Following this process of consultation and review, the Imports Standards team of MAF Biosecurity New Zealand decides on the appropriate combination of sanitary measures to ensure the effective management of identified risks. These are then presented in a draft IHS which is released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to the draft IHS are reviewed before a final IHS is issued.

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## 5. Avian paramyxovirus-1

### 5.1. HAZARD IDENTIFICATION

#### 5.1.1. 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 6.

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 an 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, which 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 can only be 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 2009) 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.

#### 5.1.2. OIE list

Listed.

#### 5.1.3. 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 considered to be unwanted notifiable organisms (MAF 2009).

#### 5.1.4. 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 velogenic 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, 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 viral 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 carcass 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).

#### **5.1.5. Hazard identification conclusion**

Following infection, 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 5.1.1 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 considered to be a potential hazard in the commodity.

## **5.2. RISK ASSESSMENT**

### **5.2.1. Entry assessment**

Historically, Lancaster (1966) stated that poultry carcasses 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 titre in muscle of infected chickens was about  $10^4$  EID<sub>50</sub> (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<sub>50</sub> (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 for NDV is assessed to be non-negligible.

## **5.2.2. Exposure assessment**

### **Backyard poultry**

NDV may be regarded as a heat labile virus and heat inactivation studies have shown that it is likely to be inactivated by domestic cooking (Alexander and Manvell 2004). 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 carcasses for 121 days.

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

There is considered to be a negligible likelihood of backyard poultry being exposed to NDV from cooked turkey meat scraps and a non-negligible likelihood of exposure to NDV from raw scraps generated during the domestic preparation of imported turkey meat.

### **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 turkey 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, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008).

However, the likelihood that commercial poultry will be subject to secondary exposure from infected free-living avian species or backyard flocks should be considered.

#### **Exposure of commercial poultry from free-living avian species**

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 ensure that the likelihood of commercial poultry being exposed to free-living avian species is very low.

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.

It is therefore concluded that there is a non-negligible likelihood of commercial poultry being exposed to NDV through infected wild birds.

#### Exposure of commercial poultry from backyard flocks

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).

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

#### 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.

#### 5.2.3. 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.

#### 5.2.4. Risk estimation

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

## 5.3. RISK MANAGEMENT

### 5.3.1. Options

Article 10.13.14 of the current OIE *Code* (OIE 2009) 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 10.13.22 to 10.13.26. 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 10.13.22 to 10.13.26 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 although 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 10.13.23 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 can be assessed by determining the ICPI or by using molecular techniques (reverse-transcription polymerase chain reaction and sequencing). 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 four 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.

MAF's import risk analysis for chicken meat products and turkey preparations (MAF 1999) and subsequent modelling work (MAF 2000) demonstrated that, to ensure chicken meat contains APMV-1 at a titre no higher than  $-9 \log_{10} \text{CID}_{50}/\text{g}$ , chicken meat should be cooked at 80°C for 5 minutes or 70°C for 30 minutes. MAF estimated that under such a cooking regime the risk of NDV introduction, if imported poultry meat were consumed at a rate equivalent to 20% of the current consumption, would be one outbreak per 1000 importation years. It would be reasonable to conclude that cooking imported turkey meat under these conditions would effectively manage the risk of introducing NDV.

Article 10.13.21 of the *Code* describes cooking at 80°C for 203 seconds or 70°C for 574 seconds as being suitable for the inactivation of NDV in meat. Although this Article is currently described as 'under study', these less stringent conditions could also be considered to effectively manage the risk of introducing NDV when this amendment is adopted.

### Option 1

Imported turkey 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 10.13.22 to 10.13.26 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 or a validated molecular test has demonstrated freedom from NDV at slaughter could be considered eligible for import.

### Option 3

Imported turkey meat could be cooked at 80°C for 5 minutes or 70°C for 30 minutes.

### Option 4

Imported turkey meat could be cooked at 80°C for 203 seconds or 70°C for 574 seconds.

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## 6. Avian paramyxoviruses 2-9

### 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. APMV-1 is considered in Chapter 5 of this risk analysis.

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

**Table 2. Prototype viruses and host range of avian paramyxoviruses (from Alexander and 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.

#### 6.1.2. OIE list

Not listed.

#### 6.1.3. New Zealand status

APMV-2, -3, and -5 are listed as unwanted exotic organisms (MAF 2009).

Stanislawek et al (2002) recovered APMV-4 from live healthy mallard ducks in New Zealand and 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).

#### **6.1.4. Epidemiology**

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). A subsequent survey of 37 turkey flocks indicated that APMV-2 was widespread in the United States, with 27 flocks showing serological evidence of exposure (Bankowski et al 1968). However, since the move of turkeys from range to indoor rearing, disease due to APMV-2 is now considered uncommon in the United States (Swayne 2010).

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).

APMV-2 infection of turkeys may be subclinical or associated with mild respiratory signs including occasional coughing, ocular discharge and encrusted nostrils (Bankowski et al 1968; Bradshaw and Jensen 1979). Lang et al (1975) described three severe respiratory disease outbreaks in turkey flocks in Canada where APMV-2 was recovered, although in each of these cases other pathogenic organisms were also recovered which were more likely to be the cause of the signs described. Experimental infection of turkey hens with APMV-2 was shown to have a profound effect on egg hatchability and poult yield (Bankowski et al 1981).

APMV-3 has been isolated from turkeys in the United States and Canada (Alexander 1980) and serologically related viruses have been reported in several countries in Europe (Alexander and Senne 2008). Natural infection of chickens with APMV-3 has not been described although experimental infection of 1-day-old chicks results in severe growth impairment whereas no clinical signs follow experimental infection of 6-week-old birds (Alexander and Collins 1982). Natural infection of turkey flocks with APMV-3 has been associated with reduced egg production in breeder farms (Alexander et al 1983; MacPherson et al 1983) and mild respiratory disease (Tumova et al 1979).

APMV-6 has been isolated on one occasion from turkeys with reduced egg production and mild respiratory problems (Alexander 2000).

APMV-7 has been isolated from an outbreak of respiratory disease in a turkey breeder flock in the United States. Experimental inoculation of specific-pathogen-free poults with this isolate resulted in rhinitis and airsacculitis (Saif et al 1997).

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).

### **6.1.5. Hazard identification conclusion**

APMV-2, -3, -6, and -7 have been associated with turkeys.

There is only a single report of natural APMV-7 infection of turkeys with no subsequent reports during the last 12 years and APMV-6 is recognised in New Zealand. APMV-6 and -7 are not considered to be potential hazards in the commodity.

Replication of APMV-2 and APMV-3 is limited to the intestinal and respiratory tracts so these viruses are not considered to be potential hazards in turkey meat and turkey meat products.

Although respiratory and intestinal tissues will be removed from turkey carcasses, remnants of these tissues may remain following automated processing (Mulqueen 2009; Christensen 2010). Automated lung removal machinery is quoted to be 90-92% efficient and automated eviscerators are quoted to be 87-94% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection. APMV-2 and APMV-3 are therefore considered to be a potential hazard in imported entire turkey carcasses.

## **6.2. RISK ASSESSMENT**

### **6.2.1. Entry assessment**

Infection with APMV-2 or APMV-3 may be associated with mild clinical signs so infected flocks may not be detected during routine ante and post-mortem inspection. Infected tissues will be limited to any remnants of respiratory or intestinal tissues remaining in turkey carcasses after processing. The likelihood of entry is therefore considered to be very low but non-negligible.

### **6.2.2. Exposure assessment**

#### **Backyard poultry**

The heat sensitivity of APMV-2 and APMV-3 is likely to be similar to that of APMV-1. Therefore, there is considered to be a negligible likelihood of backyard poultry being exposed to APMV-2 and APMV-3 from scraps of turkey carcasses following domestic cooking.

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

Any respiratory or intestinal tissue remnants in imported turkey carcasses 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.

There is therefore considered to be a very low but non-negligible likelihood of backyard poultry exposure from raw scraps generated during the domestic preparation of imported turkey carcasses.

## Wild birds

APMV-2 has been isolated from captive or free-ranging Passeriformes, hanging parrots, mynahs, *Neophema* sp., lovebirds, and African grey parrots, and APMV-3 has been isolated from Passeriformes, Galliformes, waterfowl, and Psittaciformes (Ritchie 1995).

The likelihood of free-living avian species being infected with APMV-2 or APMV-3, 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, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008).

However, the likelihood that commercial poultry will be subject to secondary exposure from infected free-living avian species or backyard flocks should be considered.

### Exposure of commercial poultry from free-living avian species

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 ensure that the likelihood of commercial poultry being exposed to free-living avian species will be very low. However, 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).

It is therefore concluded that there is a non-negligible likelihood of commercial poultry being exposed to APMV-2 or APMV-3 through infected wild birds.

### Exposure of commercial poultry from backyard flocks

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 or APMV-3 infection from backyard flocks to commercial poultry so there is a negligible likelihood of commercial poultry being exposed to APMV-2 or APMV-3 through infected backyard flocks.

## Exposure assessment conclusion

In conclusion, the likelihood of exposure of backyard poultry, wild birds, and commercial poultry to APMV-2 or APMV-3 is assessed to be non-negligible.

### 6.2.3. Consequence assessment

APMV-2 infection of turkeys may be subclinical, associated with mild respiratory signs, or result in reduced egg hatchability and poult yield, and APMV-3 infections have also been associated with reduced egg production and mild respiratory disease. APMV-2 infection of chickens has been associated with mild respiratory signs and natural infection of chickens with APMV-3 has not been described.

Most APMV-2 infections of Passeriformes are mild and self-limiting; infection of psittacines can lead to severe clinical signs including pneumonia, mucoid tracheitis, diarrhoea, and high mortality. APMV-3 infections of *Neophema* sp. have been associated with neurological signs and up to 40% mortality. Neurological signs have also been described in parakeets, Cockatiels, and a Cockatoo infected with APMV-3. Conjunctivitis, anorexia, diarrhoea, and dyspnoea due to APMV-3 has been described in the Gouldian finch, blue waxbill, common canary, white-rumped canary, orange-cheeked waxbill, black-throated grassfinch, double-barred finch, and avadavat (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 or APMV-3 would have negligible consequences for human health.

The introduction of APMV-2 or APMV-3 in the commodity would be associated with non-negligible consequences to the New Zealand poultry industries and wildlife. The consequences are therefore assessed as non-negligible.

### 6.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and APMV-2 and APMV-3 are classified as hazards in imported whole turkey carcasses. Therefore, risk management measures can be justified.

## 6.3. RISK MANAGEMENT

### 6.3.1. Options

The *Code* contains no recommendations for sanitary measures appropriate to manage the risk of APMV-2 or APMV-3 in poultry meat. However, recognition of country, zone, or compartment freedom from NDV could be extended to include freedom from APMV-2 and APMV-3.

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 and APMV-3.

MAF's import risk analysis for chicken meat products and turkey preparations (MAF 1999) and subsequent modelling work (MAF 2000) demonstrated that, to ensure chicken meat contains APMV-1 at a titre no higher than  $-9 \log_{10} \text{CID}_{50}/\text{g}$ , chicken meat should be cooked at 80°C for 5 minutes or 70°C for 30 minutes. MAF estimated that under such a cooking regime the risk of NDV introduction, if imported poultry meat were consumed at a rate equivalent to 20% of the current consumption, would be one outbreak per 1000 importation years. It would be reasonable to conclude that cooking imported turkey carcasses under these conditions would effectively manage the risk of introducing APMV-2 and APMV-3.

Article 10.13.21 of the *Code* describes cooking at 80°C for 203 seconds or 70°C for 574 seconds as being suitable for the inactivation of NDV in meat. Although this Article is currently described as ‘under study’, these less stringent conditions could also be considered to effectively manage the risk of introducing APMV-2 and APMV-3 when this amendment is adopted.

### Option 1

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

### Option 2

Imported turkey carcasses could be derived from birds kept in a country, zone or compartment free from APMV-2 and APMV-3 since they were hatched or for at least the past 21 days.

### Option 3

Carcasses derived from flocks where virus isolation has demonstrated freedom from APMV-2 and APMV-3 at slaughter could be considered eligible for import.

### Option 4

Imported turkey carcasses could be cooked at 80°C for 5 minutes or 70°C for 30 minutes.

### Option 5

Imported turkey carcasses could be cooked at 80°C for 203 seconds or 70°C for 574 seconds.

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## 7. Turkey rhinotracheitis, swollen head syndrome, and avian rhinotracheitis

### 7.1. HAZARD IDENTIFICATION

#### 7.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Metapneumovirus* (Gough and Jones 2008). Avian metapneumoviruses (aMPV) have 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).

#### 7.1.2. OIE list

TRT is an OIE listed disease.

#### 7.1.3. New Zealand status

In New Zealand the clinical syndrome of TRT has never been reported and a small serological survey in 1988 found no evidence of TRT antibodies in five different flocks (Horner 1993).

Turkey rhinotracheitis virus is listed as an unwanted exotic organism (MAF 2009).

#### 7.1.4. Epidemiology

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).

Typical clinical signs associated with natural infection of turkeys include snicking, rales, sneezing, nasal discharge, foamy conjunctivitis, swollen infraorbital sinus and submandibular oedema. Laying birds may experience a drop in egg production of up to 70%. Morbidity in an infected flock can be up to 100%, with mortality ranging from 0.4% to 50% (Gough and Jones 2008).

Experimentally infected birds 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).

Bacteria play an important role as secondary pathogens in field and experimental cases of TRT, 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). Shin et al (2002) demonstrated transmission of aMPV to mallard ducks from a neighbouring turkey flock experiencing a severe TRT outbreak.

Sequence analysis has shown a high sequence identity among wild bird isolates and between wild bird and turkey isolates, which suggests 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 turkeys 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 (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 carcasses or processed products.

#### **7.1.5. Hazard identification conclusion**

Following infection of turkeys, virus replication is limited to the respiratory tract tissues. There is no evidence of virus in any other tissues. aMPVs are not considered to be a potential hazard in turkey meat and turkey meat products.

Although respiratory tract tissues will be removed from turkey carcasses, remnants of these tissues may remain following automated processing (Mulqueen 2009; Christensen 2010). Automated lung removal machinery is quoted to be 90-92% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection. aMPVs are therefore considered to be a potential hazard in imported entire turkey carcasses.

## **7.2. RISK ASSESSMENT**

### **7.2.1. Entry assessment**

Turkey flocks infected with aMPVs can be expected to report high morbidity and mortality rates although these will be affected by management factors and the presence of secondary infections (Gough and Jones 2008). It is therefore likely that an infected flock would be detected during ante-mortem inspection.

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 some upper respiratory tract tissue will remain in around 0.2% of processed chicken carcasses (MAF 1999). In the absence of any evidence to the contrary, it is assumed that a similar figure would apply to turkey carcasses.

Considering the above, the likelihood of entry in imported turkey carcasses is considered to be very low but non-negligible.

### **7.2.2. 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 turkey carcasses following domestic cooking.

Any respiratory tissue remnants in imported turkey carcasses 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 considered to be negligible.

### 7.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and aMPVs are not assessed to be a hazard in the commodity. Therefore, risk management measures cannot be justified.

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## 8. Avian influenza

### 8.1. HAZARD IDENTIFICATION

#### 8.1.1. 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).

#### 8.1.2. 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 2009) states that, for the purposes of international trade, avian influenza 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.

#### 8.1.3. New Zealand status

Influenzavirus type A (exotic avian strains) and avian influenza H5 and H7 are listed as unwanted notifiable organisms (MAF 2009).

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 feral pigeons found no evidence of AI infections (Black 2004).

#### **8.1.4. 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 turkey 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 turkey 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 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, airsacculitis, and pneumonia) with histological lesions and viral antigen distribution restricted to the lungs and trachea although pancreatic necrosis is also reported in turkeys (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 titres 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 titres in muscle tissue ( $10^{2.2-3.2}$  EID<sub>50</sub> virus/g) and feeding of susceptible birds with this meat did not transmit infection, whereas an H5N1 isolate achieved a much higher titre in muscle tissue ( $10^{7.3}$  EID<sub>50</sub> 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.

### **8.1.5. 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. Automated lung removal machinery is quoted to be 90-92% efficient and automated eviscerators are quoted to be 87-94% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection.

Notwithstanding the likelihood that some respiratory tract tissues may be present in imported turkey carcasses, given the wide range of LPAI viruses that have been described in New Zealand, LPAI is not considered to be a potential hazard in imported turkey 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 considered to be a potential hazard.

## **8.2. RISK ASSESSMENT**

### **8.2.1. 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 turkeys, based on these findings it is reasonable to conclude that the likelihood of HPAI being present in imported turkey meat is non-negligible.

### **8.2.2. 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 titre and the authors concluded that the U.S. Department of Agriculture Food Safety and Inspection Service time-temperature guidelines<sup>3</sup> 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 turkey meat are likely to be able to transmit infection.

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

<sup>3</sup> See: [http://www.fsis.usda.gov/OPPDE/rdad/FSISNotices/RTE\\_Poultry\\_Tables.pdf](http://www.fsis.usda.gov/OPPDE/rdad/FSISNotices/RTE_Poultry_Tables.pdf)

There is a negligible likelihood of backyard poultry being exposed to HPAI from cooked turkey meat and a non-negligible likelihood of exposure to HPAI from scraps of raw meat generated during the domestic preparation of imported turkey meat.

### **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 turkey 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, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008b).

However, the likelihood that commercial poultry will be subject to secondary exposure to HPAI from infected free-living avian species or backyard flocks must be considered.

#### **Exposure of commercial poultry from free-living avian species**

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 ensure that the likelihood of commercial poultry being exposed to free-living avian species will be very low. However, the introduction of AI viruses to commercial poultry by migratory waterfowl is documented (Halvorson et al 1985) so the likelihood of exposure of commercial poultry from free-living avian species is assessed as non-negligible.

#### **Exposure of commercial poultry from backyard flocks**

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).

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).

Unlike Newcastle disease (see Section 5.2.2), there have been no reported cases of HPAI infection being introduced into commercial poultry from an infected backyard flock. It is therefore concluded that there is a negligible likelihood of commercial poultry being exposed to HPAI through infected backyard flocks.

### **Exposure assessment conclusion**

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

#### **8.2.3. 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, therefore, 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. The consequences are assessed to be non-negligible.

#### **8.2.4. Risk estimation**

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

## 8.3. RISK MANAGEMENT

### 8.3.1. Options

Article 10.4.20 of the current OIE *Code* (OIE 2009) 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 10.4.28 to 10.4.34, 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 10.4.28 to 10.4.34 has been carried out during that three month period. Restricting imports of turkey meat to countries 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 risk of HPAI virus infection being present in imported turkey meat.

Article 10.4.27 of the OIE *Code* describes cooking procedures recognised to achieve the inactivation of AI virus in poultry meat as shown in Table 3 (below):

**Table 3. Temperature/time requirements to inactivate AI virus in poultry meat**

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

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

### Option 1

Imported turkey 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 10.4.28 to 10.4.34 of the *Code*.

### Option 2

Meat derived from 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 turkey meat could be cooked in accordance with Article 10.4.27 of the OIE *Code*.

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## 9. Infectious bursal disease

### 9.1. HAZARD IDENTIFICATION

#### 9.1.1. Aetiological agent

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

#### 9.1.2. OIE list

Listed.

#### 9.1.3. New Zealand status

Exotic, notifiable disease (MAF 2009). Ongoing industry surveillance has detected no cases of IBD in commercial poultry since 1999 (Brooks 2003; Gerber 2008).

#### 9.1.4. Epidemiology

Natural infection of turkeys with IBDV (designated TY89) was first reported in 1979 (McNulty et al 1979). Further work identified this TY89 isolate as the prototype strain for IBDV serotype 2 (McFerran et al 1980). Jackwood et al (1982a) found widespread seroconversion to “IBDV serotype II” in turkey flocks from Ohio, North Carolina and Indiana and it was subsequently demonstrated that the American IBDV serotype II and the British IBDV serotype 2 belonged to the same serotype (McNulty and Saif 1988).

A United States survey of 23 turkey breeder flocks and 22 commercial turkey flocks found 75% of breeder flocks and 77% of commercial flocks were seropositive to IBDV-2. This survey also found antibodies to IBDV-1 in 70% of breeder flocks, all of which had been vaccinated using a commercial IBDV-1 vaccine (Jackwood et al 1982b).

Ten Iowa turkey flocks showed seroconversion to IBDV-2 from five to eight weeks of age (Barnes et al 1982). In these flocks there were also low to negligible titres to IBDV-1, which were suggested to be due to cross-reactivity associated with the use of chicken IBDV vaccines in turkey breeder hens. No clinical disease resembling IBDV infection in chickens was identified.

In California, a survey of 15 turkey flocks found 15% of 342 samples to be seropositive for IBDV-1 whilst 89% were seropositive for IBDV-2. Serological titres to IBDV-1 were low compared with those to IBDV-2. All flocks sampled had originated from IBDV-1 vaccinated parent flocks. The authors suggested that the presence of high antibody titres to IBDV-2 had caused a falsely high titre to IBDV-1 which would account for the high number of flocks found to be positive to IBDV-1 in this survey (Chin et al 1984). Similarly, high serological titres to IBDV-2 were seen in five Minnesota turkey flocks alongside low titres to IBDV-1 (Sivanandan et al 1984).

Eddy et al (1985) surveyed 32 turkey flocks in England. They found antibodies to IBDV-2 in 29 flocks and no turkey flocks with antibodies to IBDV-1 despite widespread infection of English chickens with IBDV-1.

In 1978 it was speculated that there could be an association between natural IBDV infections in young turkeys and concurrent respiratory problems (Page et al 1978). Turkeys in problem flocks were reported to have antibodies to IBDV, but no virus was isolated.

It was later reported that poults recovering from rhinotracheitis had high titres of antibodies to IBDV (Johnston et al 1980) and it was suggested that immunosuppression by IBDV might predispose poults to other diseases such as adenoviruses, Newcastle disease, or *Alcaligenes faecalis*.

However, two years later a study found that infection of poults with IBDV-2 did not predispose to *Alcaligenes* rhinotracheitis (*Alcaligenes faecalis*) (Jackwood et al 1982a). It was reported that no gross or histological lesions were observed (apart from those due to *Alcaligenes* rhinotracheitis) in exposed poults.

Experimental infection of day-old turkey poults with IBDV-2 resulted in no clinical disease or histological changes in the bursa, spleen, or thymus although a suppression of the cellular immune system and a decrease in the plasma cell population of the Harderian gland were described (Nusbaum et al 1988).

Reddy and Silim (1989) isolated IBDV from the synovial fluid of turkeys with swollen hock joints and respiratory disease. The isolate recovered (IBDV QT-1) was not conclusively demonstrated to be either IBDV-1 or IBDV-2 although the authors suggested that ability of IBDV-1 commercial chicken antiserum to neutralise this virus might suggest a vaccinal origin. Furthermore, a number of bacterial pathogens were cultured from the joint fluid of infected turkeys so the significance of this viral isolate was undetermined. Subsequent work has suggested antigenic similarities between this IBDV QT-1 isolate and IBDV-2 (Reddy et al 1992).

Giambrone (1978) experimentally infected turkey poults with an IBDV-1 isolate that had been passaged through turkeys six times in order to increase the isolate's pathogenicity in this species. The resulting infections were subclinical with no morbidity, mortality, or gross lesions observed. However, microscopic changes were seen in the lymphoid organs of infected poults and similar changes were seen in uninfected poults housed with the experimentally-infected birds. Similarly, experimental infection of turkey poults with IBDV-1 was shown to result in microscopic changes in the bursa of Fabricius and impairment of the immune system although the changes observed were only partial and in no way comparable to those seen in chickens infected with IBDV-1 (Perelman and Heller 1982). More recently Oladele et al (2009) experimentally infected chickens, turkeys and ducks with IBDV-1 and found all three species could be infected with virus although there was no bursal damage and minimal viral replication in ducks and turkeys. The authors concluded that the chicken host has a facilitating inherent "factor" which permits maximal replication of the IBD virus compared with turkeys and ducks.

Owoade et al (2004) sequenced four IBDV isolates recovered from turkey flocks in Nigeria. Viral isolates were recovered from turkeys that showed no macroscopic signs of IBDV infection at necropsy although one flock reported a high (27%) mortality. A further 40 IBDV isolates recovered from chickens were characterised in this study. Sequence analysis of these isolates was claimed to show they were all vvIBDV variants and the genetic diversity of these variants was >50% higher than the genetic diversity seen between all other vvIBDV isolates identified in Europe, Latin America and Asia. The authors of this study went on to suggest that, based on the expected mutation rate for this virus and the high level of virus diversity in Nigeria, vvIBDV may have been circulating in West Africa for several hundred years. The finding of no macroscopic lesions associated with IBDV in turkey flocks sampled suggests

that the vvIBDV isolates recovered from this study were not pathogenic. Furthermore, molecular differentiation of vvIBDV strains should be interpreted with caution as the virulence markers are not known and the reliable indicator of vvIBDV remains *in vivo* pathogenicity testing (Ashraf et al 2007). It has also been suggested that incorrect identification of viruses in Nigeria is not unusual and these findings should be regarded with caution unless confirmed by another laboratory (Swayne 2010).

Chickens are the only animals known to develop clinical disease and distinct lesions when exposed to IBDV (Etteradossi and Saif 2008). Serotype 1 and 2 viruses have been isolated from chickens (McFerran et al 1980).

Infection of chickens with IBDV-1 can lead to diarrhoea, anorexia, depression, ruffled feathers, trembling, and death. Flock mortality may be zero although it can be as high as 20-30%. Mortality rates of 90-100% have been associated with vvIBDV. The cloacal bursa is the primary target organ and infection leads initially to cloacal oedema and hyperaemia which is followed by atrophy around five days after infection. Microscopic lesions in other lymphoid tissues are described (Etteradossi and Saif 2008).

Although Sivanandan et al (1986) reported bursal necrosis and atrophy in specific-pathogen-free 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).

#### **9.1.5. Hazard identification conclusion**

The study by Owoade et al (2004) suggests vvIBDV may be present in turkeys in Nigeria although the reliability of these findings has been questioned. Notwithstanding these reservations, IBDV-1 (including vvIBDV) should be considered a potential hazard in turkey meat originating from this part of the world.

There is no evidence for natural infection of turkeys with IBDV-1 strains present elsewhere in the world. There is serological evidence of exposure to IBDV-1 in commercial turkey flocks in some countries although this is associated with the use of IBDV-1 vaccines.

IBDV-1 is not a potential hazard in turkey meat originating from anywhere other than West Africa.

Natural infection of turkeys with IBDV-2 is widespread in commercial turkey flocks and IBDV-2 should be considered a potential hazard in the commodity.

## **9.2. RISK ASSESSMENT**

### **9.2.1. Entry assessment**

Appendix 4 of MAF's *import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom* described a quantitative assessment of the risk of backyard flocks becoming infected with IBDV-2 following the importation of turkey meat preparations from the United Kingdom (MAF 1999). This model assumed that turkeys could be infected with IBDV-2 from one day of age to 12 weeks (the approximate age of slaughter in commercial turkey flocks), and most birds would be infected between 4 and 7 weeks old (Chettle et al 1985; MAF 1999). Studies

commissioned by MAF and the Chief Veterinary Officer of Australia in chickens using IBDV-1 demonstrated that IBDV is recoverable from muscle tissue of chickens for 2-6 days post-infection and it was assumed that these figures provided a reasonable approximation for the duration of IBDV-2 infectivity in turkey muscle tissues. Based on these figures the probability that imported turkey muscle meat will be infected with IBDV-2 at slaughter was calculated to be approximately 0.001 (i.e. <0.1%) (MAF 1999).

The likelihood of IBDV-2 being present in imported turkey meat is therefore assessed to be very low but non-negligible.

There is insufficient information to estimate the likelihood that IBDV-1 (including vvIBDV) might be present in turkey meat imported from Nigeria. However, turkey production in Nigeria remains at the smallholder level (Okoli et al 2006). Furthermore, MAF is unaware of any turkey meat export industry in West Africa and has received no representations to open such a trade. For these reasons the entry assessment for IBDV-1 is considered to be negligible and this issue will not be pursued further in this risk analysis.

### **9.2.2. Exposure assessment**

#### **Backyard poultry**

Heat inactivation studies have shown that there is a high probability that IBDV would survive at infectious titres in domestically cooked chicken, especially in deep tissues (MAF 1999). Although these studies have not been repeated in turkey meat, it is reasonable to assume that IBDV would also persist in infected turkey meat after domestic cooking. It has previously been calculated that, for consumer-ready turkey preparations, we can be 95% confident that with a 10% market penetration there are likely to be fewer than four IBDV-2 introductions per thousand years and for 50% market penetration there are likely to be fewer than two IBDV-2 introductions per hundred years (MAF 1999).

However, the modelling that provided this result estimated that there was a less than 1% likelihood of edible turkey scraps being disposed of from the imported commodity. Whilst it would be quite reasonable to assume this to be a valid assumption for a consumer-ready product, the likelihood of scrap generation from imported turkey meat requiring home preparation is likely to be greater than this 1% value used in MAF's previous model.

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

Backyard flocks may therefore be exposed to IBDV in either raw or cooked imported turkey meat. Based on MAF's 1999 quantitative model, and accepting that the likelihood of scrap generation from turkey meat is likely to be greater than scrap generation from consumer-ready preparations, the likelihood of backyard poultry flocks being exposed to IBDV-2 from turkey meat imported from any country 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<sub>50</sub>) 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-2, 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 but non-negligible.

### **Commercial poultry**

As described above, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008).

However, the likelihood that commercial poultry will be subject to secondary exposure to IBDV from infected free-living avian species or backyard flocks must be considered.

#### **Exposure of commercial poultry from free-living avian species**

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 ensure that the likelihood of commercial poultry being exposed to free-living avian species will be very low.

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/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.

#### Exposure of commercial poultry from backyard flocks

IBDV is highly contagious and the virus is persistent in the poultry house environment (Etteradossi 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 (Etteradossi 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  $5 \times 10^{4.3}$  EID<sub>50</sub> 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 IBDV-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 birds (Christensen 2009).

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

#### Exposure assessment conclusion

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

### 9.2.3. Consequence assessment

IBDV-2 infection is not associated with any clinical disease in turkeys and experimentally-infected chickens. There are no reports of IBDV-2 causing disease in free-living avian species. Local industry has previously raised concerns that the introduction of IBDV-2 would interfere with ongoing IBDV-1 testing and eradication from New Zealand chicken flocks (MAF 1999). However, as the likelihood of exposure of commercial poultry is negligible, there is considered to be a negligible likelihood of IBDV-2 introduction into commercial poultry flocks associated with the importation of turkey meat.

Therefore, the consequences of IBDV-2 associated with turkey meat from any country are assessed to be negligible.

### 9.2.4. Risk estimation

Since the consequence assessment for IBDV-2 associated with turkey meat from all countries is assessed to be negligible, under the methodology used in this risk analysis (see Section 4.3) the risk is estimated to be negligible and sanitary measures cannot be justified.

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## 10. Group I adenovirus infections

### 10.1. HAZARD IDENTIFICATION

#### 10.1.1. Aetiological agent

Family: *Adenoviridae*, Genus: *Aviadenovirus*. Most members of this genus do not have a well-defined role as pathogens, with the exceptions of fowl adenovirus-1 (FAdV-1) strains which cause quail bronchitis and fowl adenovirus-4 (FAdV-4) strains associated with hydropericardium syndrome (Adair and Fitzgerald 2008).

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

<i>Mastadenovirus</i>	Mammalian adenoviruses
<i>Aviadenovirus</i>	Group I avian adenoviruses, considered in this chapter
<i>Siadenovirus</i>	Group II avian adenoviruses, considered in Chapter 11 of this risk analysis
<i>Atadenovirus</i>	Group III avian adenoviruses (Egg drop syndrome virus and related viruses; see Section 4.1)

#### 10.1.2. OIE list

Not listed.

#### 10.1.3. New Zealand status

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

Strains of FAdV-1, -8, and -12 have been isolated from New Zealand poultry (Saifuddin 1990; Saifuddin and Wilks 1990; Saifuddin and Wilks 1991; Saifuddin et al 1992).

#### 10.1.4. Epidemiology

Different serotypes of avian adenoviruses and different strains of the same serotype vary with regard to their ability to cause disease (Cook 1974; Dhillon and Winterfield 1984). It has been suggested that many avian adenoviruses cause disease only in the presence of a secondary agent (Adair and Fitzgerald 2008).

A survey of 26 turkey flocks in Northern Ireland demonstrated widespread adenovirus infection without significant illness (Scott and McFerran 1972) and serological evidence of exposure to adenoviruses is widespread in turkeys (Adair and Fitzgerald 2008).

A group I avian adenovirus was associated with inclusion body hepatitis in day-old turkey poults (Guy and Barnes 1997) and Shivaprasad et al (2001) recovered a group I avian adenovirus from 4-week-old turkeys with inclusion body hepatitis.

Group I avian adenoviruses have also been associated with respiratory disease in turkeys. However, attempts to reproduce disease using recovered virus were unsuccessful, even when inoculated into poults stressed with deoxycorticosterone (Easton and Simmons 1977; Sutjipto

et al 1977). The role of group I avian adenoviruses as either primary or secondary respiratory pathogens has not been established (Crespo et al 1998).

Similarly, adenoviruses have been recovered from cases of poult enteritis although their pathogenic role has not been established (Pantin-Jackwood et al 2007; Woolcock and Shivaprasad 2008).

Following infection, avian adenoviruses can be recovered from the tracheal and nasal mucosa, and kidneys, although the highest titres are found in the faeces (Burke et al 1959; Scott and McFerran 1972; Cowen et al 1978; Adair and Fitzgerald 2008). From the available evidence it seems that most, if not all, strains of avian adenovirus follow the same pattern of infection. Following initial multiplication there is probably a viraemia, resulting in virus spread to virtually all organs with the main sites of virus replication in the respiratory and alimentary system, especially the trachea and caecae (McFerran and Adair 1977).

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 turkeys) or inclusion body hepatitis (which is recognised in New Zealand).

#### 10.1.5. Hazard identification conclusion

Adenovirus infections are widespread in poultry in New Zealand. There is limited evidence to support a pathogenic role for group I avian adenoviruses in turkeys and there is no evidence that exotic serotypes/strains likely to be present in turkeys are more pathogenic than those in New Zealand. Group I avian adenoviruses are therefore not assessed to be a potential hazard.

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# 11. Haemorrhagic enteritis

## 11.1. HAZARD IDENTIFICATION

### 11.1.1. Aetiological agent

Family: *Adenoviridae*, Genus: *Siadenovirus*, haemorrhagic enteritis virus (HEV). Marble spleen disease of pheasants is caused by a virus (MSDV) that is serologically indistinguishable from HEV and varies only slightly at the genomic level (Pierson and Fitzgerald 2008).

### 11.1.2. OIE list

Not listed.

### 11.1.3. New Zealand status

Haemorrhagic enteritis has been described in a New Zealand turkey flock with 10% of 3000 ten-week-old birds, hatched from imported eggs, dying over a seven-day period (Howell 1992). There is no other evidence that this virus is established here (MAF 1999).

### 11.1.4. Epidemiology

HEV infects turkeys over 4-weeks-old, causing depression, bloody droppings, and death, with clinical disease persisting in affected flocks for 7-10 days (Pierson and Fitzgerald 2008). Flock mortality is reported to range from less than 1% to over 60% (Gross and Moore 1966).

Naturally-occurring infection with HEV has also been reported in commercial broiler-breeder flocks (Domermuth et al 1979), guinea fowl (Cowen et al 1988; Massi et al 1995), and psittacines (Gómez-Villamandos et al 1995).

A serological survey found no evidence of HEV infection amongst wild birds in Florida (618 birds from 42 species) (Domermuth et al 1977). Similarly, none of forty-five wild turkeys trapped for relocation in Arkansas had seroconversion to HEV (Hopkins et al 1990).

Following infection, viral replication occurs in the reticuloendothelial cells, primarily in the spleen (Wyand et al 1972; Carlson et al 1973; Fujiwara et al 1975; Itakura and Carlson 1975; Tolin and Domermuth 1975). More recent studies (Suresh and Sharma 1995; Suresh and Sharma 1996) have shown that HEV may infect and replicate in B lymphocytes and macrophages, leading to immunosuppression, which may be the underlying mechanism for the haemorrhagic intestinal lesions seen in infected birds.

Studies have shown that the most consistently infected organs are the spleen, caecal tonsil, and intestine, although virus has also been demonstrated less frequently (and in a lower concentration) in liver, bone marrow, bursa, pancreas and kidney (Silim and Thorsen 1981; Fasina and Fabricant 1982; Fitzgerald et al 1992; Trampel et al 1992; Hussain et al 1993). HEV is not found in the lungs of infected turkeys (Hussain et al 1993).

The most likely pathogenesis of HEV infection in turkeys is that virus replication occurs in lymphoid cells of the spleen. Replication does not occur in other organs and virus-infected lymphocytes from the spleen then circulate to other organs (Saunders et al 1993).

Infection is spread in the faeces of infected birds, although faeces are only infectious when birds are showing acute clinical signs (Gross and Moore 1966). Itakura et al (1974) were able

to transmit infection using an inoculum of spleen and intestinal content given orally and by the cloacal route.

#### **11.1.5. Hazard identification conclusion**

Following infection, HEV replication is limited to reticuloendothelial tissues and infectivity is concentrated in the spleen, caecal tonsil, and intestines. Some infectivity may also be found in liver, bone marrow, bursa, pancreas and kidney. HEV is not considered to be a potential hazard in turkey meat and turkey meat products.

Although evisceration will remove the vast majority of infectivity from turkey carcasses, remnants viscera may remain following automated processing (Mulqueen 2009; Christensen 2010). Automated eviscerators are quoted to be 87-94% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection. HEV is therefore considered to be a potential hazard in imported entire turkey carcasses.

### **11.2. RISK ASSESSMENT**

#### **11.2.1. Entry assessment**

HEV infection may be associated with marked clinical signs which are likely to be detected by ante-mortem and post-mortem inspection. However, infection of mature turkeys is often subclinical (McCracken and Adair 1993), which would not be detected.

Gross and Moore (1966) experimentally infected 60 turkeys with HEV and found that the degree of intestinal congestion and haemorrhage showed a marked peak at five days after inoculation. They went on to demonstrate that intestinal content taken from birds during this one day peak of haemorrhagic intestinal lesions was capable of transmitting the disease whereas samples taken immediately before and after this period were not.

Using an agar gel precipitin test, Domermuth et al (1972) were able to detect HEV antigen in 6 of 7 spleens, though not in the intestinal contents, bone marrow, liver, blood, lung, brain, or eye tissues of any of the same birds and concluded that the spleen was the most infectious of the tissues studied.

Silim and Thorsen (1981) demonstrated that, although low levels of viral antigen could be detected sporadically in the liver, intestine, kidney and bone marrow of experimentally infected poults, HEV is found principally in the spleen of infected birds. Similarly, immunofluorescence studies have shown that although scattered HEV-positive cells can be seen in a number of tissues of experimentally infected turkeys, the most consistently affected organs are the spleen, caecal tonsil, and intestine (Fasina and Fabricant 1982) and these results are supported by more recent studies of HEV antigen distribution in experimentally infected turkeys using an antigen capture ELISA (Hussain et al 1993).

HEV Infectivity is concentrated in the spleen. 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 turkey carcasses following automated evisceration.

Intestinal content is only considered infectious at the peak of clinical disease which is likely to be detected during inspection. Furthermore, whilst faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria viruses will not multiply on the carcass surface (MAF 1999).

Considering the above, the likelihood of HEV entry in imported turkey carcasses is considered to be negligible.

### 11.2.2. Risk estimation

Since the likelihood of entry is considered to be negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and HEV is not assessed to be a hazard in the commodity. Therefore, risk management measures cannot be justified.

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## 12. Reovirus infections

### 12.1. HAZARD IDENTIFICATION

#### 12.1.1. 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).

#### 12.1.2. OIE list

Not listed.

#### 12.1.3. 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).

There are no reports of reovirus isolates being recovered from turkeys in New Zealand.

#### 12.1.4. 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 immunosuppression (Dees et al 1972; Sterner et al 1989; Rosenberger and Olson 1997; van der Heide 2000).

Both Levisohn et al (1980) and Page et al (1982) recovered reoviruses from turkeys with tenosynovitis and injection of recovered virus into the footpads of a day-old poult produced tenosynovitis (Page et al 1982). However, three chicken reoviruses and three turkey reoviruses produced no lesions when injected into the footpads of day-old poults whereas the same viral isolates were found to cause erosive arthritis and tenosynovitis in chicks, suggesting that turkeys are much more resistant than chickens to the induction of arthritis/tenosynovitis by reoviruses from either species (Al-Afaleq and Jones 1989). Experimental co-infection of day-old turkey poults with reovirus and *Mycoplasma synoviae* recovered from commercial turkeys with severe synovitis was found to result in only microscopic lesions in a single bird from 20 inoculated (Al-Afaleq et al 1989). A similar experimental study performed on chickens found that co-infection with *Mycoplasma synoviae* and reovirus resulted in marked synovitis, dyspnoea, and foci of liver necrosis (Bradbury and Garuti 1978).

Reoviruses have been recovered from turkey intestines in both clinically normal birds (Simmons et al 1972) and birds with enteric disease (Dees et al 1972; Gershowtiz and Wooley 1972; Nersessian et al 1986).

Poult enteritis and mortality syndrome (PEMS) is considered to be a component of poult enteritis complex (PEC) (Barnes et al 2000), and was first identified in the United States (Jones 2008) and subsequently in the United Kingdom (Culver et al 2006). PEMS presents clinically as stunting together with poor feed utilisation and in more severe forms 100% morbidity and 100% mortality is reported (Jones 2008). Reoviruses have been implicated in the aetiology of PEMS (Heggen-Peay et al 2002) although there is likely to be a multifactorial basis for this syndrome which may also include enteropathogenic *E. coli* (Pakpinyo et al 2002), turkey coronavirus (Guy et al 2000), and turkey astrovirus (Koci and Schultz-Cherry 2002).

Sequence analysis of a turkey reovirus isolate associated with PEMS in the United States suggests that it is a separate virus species, distinct from reoviruses of chicken or duck origin (Kapczynski et al 2002). Pathogenicity studies have indicated that turkey reovirus isolates are probably not a primary cause of PEMS as clinical signs associated with experimental infection of poults are minimal, gross lesions are absent, microscopic lesions in the intestines are mild and non-specific, and the ability of these isolates to replicate in intestinal tissues is poor (Spackman et al 2005). Furthermore, these turkey reovirus isolates caused no disease in chickens and their ability to replicate in chickens is poor or nil.

#### 12.1.5. 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 remains unclear (Jones 2008). There is no evidence to suggest that turkey reoviruses should be considered as primary pathogens. Reoviruses are not considered to be a potential hazard.

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## 13. Turkey coronavirus enteritis

### 13.1. HAZARD IDENTIFICATION

#### 13.1.1. 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.

#### 13.1.2. OIE list

Not listed.

#### 13.1.3. New Zealand status

Coronavirus has not been recorded in the New Zealand turkey population. Turkey coronavirus is not listed on the unwanted organisms register (MAF 2009).

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

#### 13.1.4. Epidemiology

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).

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<sub>50</sub> 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).

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  $5 \times 10^6$  EID<sub>50</sub> TCV/ml were able to transmit infection to poult, 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.

#### **13.1.5. Hazard identification conclusion**

TCV is not considered to be a potential hazard in turkey meat and turkey meat products.

Prolonged shedding of TCV can occur following recovery from clinical disease and the bursa of Fabricius is recognised as a source of virus. As fragments of bursal tissue may be present in poultry carcasses after processing (MAF 1999) and TCV is exotic to New Zealand, this virus is assessed to be a potential hazard in imported entire turkey carcasses.

### **13.2. RISK ASSESSMENT**

#### **13.2.1. Entry assessment**

As stated above, the bursa of Fabricius is recognised as a concentrated source of TCV. Because commercial turkeys are likely to be slaughtered between 8 and 20 weeks old and the maximal bursal weight in this species occurs at 87 to 115 days (Hoskins 1977), it is likely that fragments of bursal tissue may be present in turkey carcasses after processing. The likelihood of entry of TCV in imported turkey carcasses is assessed to be non-negligible.

#### **13.2.2. Exposure assessment**

##### **Backyard poultry**

Turkey coronaviruses have been shown to be resistant to 50°C for 1 hour (Guy 2008). No reports of thermal inactivation studies of TCV at higher temperatures have been located. However, 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).

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

Turkeys are believed to be the only natural host for TCV. Although statistics for which poultry species are kept in backyard flocks in New Zealand are not available, turkeys can often be seen for sale on on-line auction sites which suggests that backyard turkey flocks are not unusual.

There is considered to be a negligible likelihood of turkeys in backyard poultry flocks being exposed to TCV from cooked turkey meat scraps and a non-negligible likelihood of exposure to TCV from raw scraps generated during the domestic processing of imported turkey carcasses.

##### **Wild birds**

Turkeys are believed to be the only natural host for TCV, so the likelihood of free-living avian species being infected with TCV, 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 negligible.

### **Commercial poultry**

As described above, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008).

However, the likelihood that commercial poultry will be subject to secondary exposure to TCV from infected backyard turkey flocks must be considered.

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 mealworms have been experimentally shown to be able to transmit TCV to turkeys, the authors of that study concluded that they may well be involved in the transmission of disease within a turkey house during an active outbreak but they are less likely to transmit disease from field soils to a turkey house after the land application of litter (Watson et al 2000).

Calibeo-Hayes et al (2003) demonstrated that a single housefly exposed to an inoculum of TCV was able to transmit infection to a seven-day-old turkey poult as a mechanical vector and that the virus could persist in the fly crop for up to 9 hours after exposure. It is therefore likely that flies from an infected backyard flock could transmit infection to a commercial poultry flock.

There is a non-negligible likelihood of commercial poultry being exposed to TCV from infected backyard turkey flocks.

#### **13.2.3. Consequence assessment**

TCV is the cause of an acute highly contagious enteric disease of turkey characterised by depression, anorexia, diarrhoea, and decreased weight gain (Guy 2008). Infection of commercial turkey flocks or backyard turkey flocks with TCV would be associated with non-negligible consequences.

As turkeys are believed to be the only natural host for TCV, there would be negligible consequences for other commercial poultry species, free-living avian species, or human health.

#### **13.2.4. Risk estimation**

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and TCV is classified as a hazard in imported turkey carcasses. Therefore, risk management measures can be justified.

### **13.3. RISK MANAGEMENT**

#### **13.3.1. Options**

TCV distribution in an infected bird is limited to the intestinal tract and the bursa of Fabricius. Turkey meat products which do not contain bursal material could be imported without further sanitary measures.

TCV has been identified in the United States, Canada, Brazil, Italy, the United Kingdom, and Australia. Turkey meat could be imported without further sanitary measures from countries where structured surveillance, carried out in accordance with Chapter 1.4 of the *Code*, has demonstrated freedom from this disease.

Reverse transcriptase polymerase chain reaction (RT-PCR) tests for the detection of TCV have been described (Breslin et al 2000; Velayudhan et al 2003) which are claimed to have a high sensitivity and specificity and may detect the virus as soon as one day after infection. The RT-PCR test could be used to test pooled faeces or intestinal content from a flock to demonstrate freedom from infection.

Enzyme-linked immunosorbent assays (ELISA) (Loa et al 2000; Guy et al 2002) and competitive ELISAs (Breslin et al 2001; Guy et al 2002) have been developed to detect antibodies to TCV in turkeys. Both of these tests have been shown to be highly sensitive and highly specific and could be used to screen turkey flocks to demonstrate freedom from infection. However, these tests would not detect the early stages of infection in a bird prior to seroconversion.

Turkey coronaviruses have been shown to be resistant to 50°C for 1 hour (Guy 2008) and no reports of thermal inactivation studies of TCV at higher temperatures have been located. However, 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 imported turkey meat under the conditions described to manage the risk associated with NDV (see Section 5.3.1) would be sufficient to manage the risk of TCV.

#### **Option 1**

Turkey meat products that do not contain remnants of the bursa of Fabricius could be considered eligible for import.

#### **Option 2**

Turkey meat could be imported from countries where TCV has not been recognised.

#### **Option 3**

Turkey meat could be imported from a flock where testing of pooled faeces or intestinal content by RT-PCR has demonstrated no infection with TCV on the day of slaughter.

## Option 4

Imported turkey meat could be cooked in accordance with the conditions required to manage the risk associated with NDV (see Section 5.3.1).

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## 14. Astrovirus infection

### 14.1. HAZARD IDENTIFICATION

#### 14.1.1. Aetiological agent

Family: *Astroviridae*, Genus: *Avastrovirus*. Member of the Avastrovirus genus infect avian species. Two distinct strains of turkey astrovirus (TAsTV) are described, TAsTV-1 and TAsTV-2 (Fauquet et al 2005; Reynolds and Schultz-Cherry 2008)

#### 14.1.2. OIE list

Not listed.

#### 14.1.3. New Zealand status

Unwanted exotic organism (MAF 2009).

#### 14.1.4. Epidemiology

Avian astroviruses were first described in 11-day-old and 6-day-old poults with diarrhoea and increased mortality (McNulty et al 1980).

Subsequently, Reynolds and Saif (1986) used electron microscopy to detect astrovirus in intestinal samples taken from poults in California, Missouri, North Carolina and Wisconsin. Similarly, Reynolds et al (1987) demonstrated astrovirus in four turkey flocks in Ohio, and Yu et al (2000a) identified astrovirus in turkey flocks in Indiana. A turkey enterovirus-like virus that was recovered from turkeys with enteritis in North Carolina in 1988 (Guy and Barnes 1991) was subsequently identified as an astrovirus (Guy et al 2004).

A survey of four commercial turkey flocks in Ohio demonstrated that poults are usually infected within the first four weeks of life (Reynolds et al 1987) and results in growth depression, decreased thymus size, and a profuse watery diarrhoea (Koci et al 2003).

In situ hybridisation studies have shown that viral replication is limited to the intestines (Behling-Kelly et al 2002; Koci et al 2003), although RT-PCR and virus isolation have demonstrated that a transient viraemia occurs which can result in virus detection in skeletal muscle five days after infection at the peak of viraemia (Koci et al 2003).

Poult enteritis mortality syndrome (PEMS) is a highly infectious disease of commercial turkeys that is associated with diarrhoea, immunosuppression, and high mortality (Schultz-Cherry et al 2001). Astroviruses have been associated with PEMS (Koci et al 2000a; Koci et al 2000b; Yu et al 2000a; Yu et al 2000b), although a number of other viruses, bacteria, and protozoa have also been suggested as the cause of this disease (Schultz-Cherry et al 2001). The exact role of astroviruses in PEMS remains unclear (Reynolds and Schultz-Cherry 2008).

#### 14.1.5. Hazard identification conclusion

Natural infection of turkeys with astrovirus occurs before they are four weeks old and is followed by a transient viraemia during which low titres of virus may be detected in skeletal muscle for a short period. Commercial turkeys are likely to be slaughtered at or after eight weeks of age (MAF 1999) so there is a negligible likelihood of virus being present in turkey meat when birds are slaughtered. Astroviruses are not assessed to be a potential hazard in the commodity.

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## 15. Avian enterovirus-like infection

### 15.1. HAZARD IDENTIFICATION

#### 15.1.1. Aetiological agent

A group of nine genera within the Family *Picornaviridae*, enterovirus-like viruses (ELVs) (Guy et al 2008).

Turkey viral hepatitis is addressed separately in Chapter 18 of this risk analysis.

#### 15.1.2. OIE list

Not listed.

#### 15.1.3. 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).

#### 15.1.4. Epidemiology

ELV infections have been reported in turkeys throughout the world, including the United Kingdom (McNulty et al 1979), the United States (Saif et al 1985; Reynolds et al 1987; Saif et al 1990), and France (Andral and Toquin 1984).

Natural ELV infections have been described in turkeys, chickens, guinea fowl, partridges, pheasants, ostriches, and psittacine species (Guy et al 2008).

Horizontal transmission through ingestion of faeces is considered most likely (Guy et al 2008) although Spackman et al (1984) identified enteroviruses in the merconium 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 poult was found to be already infected with ELVs when obtained from the hatchery.

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).

Experimental infection of poults with ELVs results in depression, ruffled feathers, watery droppings, and pasted vents. Clinical signs develop three days after infection and resolve after a further three to five 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).

#### 15.1.5. Hazard identification conclusion

The pathogenic role of turkey ELVs is not completely understood. Infectivity is restricted to the intestinal tract and there is no evidence for infectivity elsewhere in the carcass. Reflecting this, ELVs are not considered to be a potential hazard.

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## 16. Turkey torovirus infection / stunting syndrome

### 16.1. HAZARD IDENTIFICATION

#### 16.1.1. Aetiological agent

Order: *Nidovirales*, Genus: *Torovirus*. The enteric virus formerly known as stunting syndrome agent (SSA) has been determined to be a torovirus and SSA is now referred to as turkey torovirus (Ali and Reynolds 1997; Ali and Reynolds 2000; Reynolds and Ali 2008).

#### 16.1.2. OIE list

Not listed.

#### 16.1.3. New Zealand status

Turkey torovirus has not been described in New Zealand and is considered exotic.

#### 16.1.4. Epidemiology

The worldwide distribution is unknown. Around 30% of USA turkey flocks experiencing enteric disease were found to be positive for torovirus and seropositive turkey flocks have been described in Israel (Reynolds and Ali 2008).

Infected poults have clinical signs lasting seven to ten days including diarrhoea, stunting, and poor feed conversion (Angel et al 1990). Mortality is usually low but morbidity is high (Reynolds and Ali 2008). Turkeys older than four weeks may have mild disease or subclinical infection. Chickens are refractory to infection with the virus (Reynolds and Ali 2008).

Toroviral antigen can be detected in the intestinal tissue of infected birds and gross lesions associated with infection are limited to the intestinal tract. Torovirus associates with intestinal epithelial cells and its demonstration from intestines/faeces may require concentration from large volumes of clinical specimens (Reynolds and Ali 2008)

Equine and bovine toroviruses are the best-studied members of this genus (Koopmans and Horzinek 1994), although the relationship between turkey torovirus and toroviruses from other animal species has not been determined (Reynolds and Ali 2008). The equine Berne virus (BEV) was the first torovirus identified in 1972 and is considered the torovirus prototype and the bovine Breda virus (BRV) was identified seven years later. Experimental infection of gnotobiotic calves has shown BRV can cause gastroenteritis and that this virus mainly infects differentiating epithelial cells in the crypts of the intestinal villi (Koopmans and Horzinek 1994).

#### 16.1.5. Hazard identification conclusion

The pathogenesis of turkey torovirus infections is not completely understood. However, it appears that infectivity is restricted to the intestinal tract and there is no evidence for infectivity elsewhere in the carcass. Although splitting of the intestinal tract during automated processing may result in carcass contamination (Christensen 2010), as virus associates with intestinal epithelial cells and its demonstration from intestines/faeces may require concentration from large volumes of clinical specimens (Reynolds and Ali 2008), turkey torovirus is not considered to be a potential hazard.

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## 17. Arbovirus infections

### 17.1. HAZARD IDENTIFICATION

#### 17.1.1. 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)

#### 17.1.2. OIE list

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

#### 17.1.3. New Zealand status

EEEV and WEEV are listed as unwanted notifiable organisms and HJV is listed as an unwanted exotic organism (MAF 2009).

#### 17.1.4. 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 turkey flocks with EEEV has been described (Spalatin et al 1961; Ficken et al 1993; Wages et al 1993) and it is regarded as a sporadic disease in this species. 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 turkeys is associated with muscular incoordination, tremors, and paralysis of legs or wings (Spalatin et al 1961). Following experimental intramuscular infection of turkeys, there is a short period of viraemia (1-2 days), with subsequent histological lesions described in the bursa of Fabricius, thymus, spleen, heart, pancreas and kidney (Guy et al 1993). Lesions in the central nervous system have also been described (Spalatin et al 1961) and experimental infection of toms has been shown to result in viral shedding in semen (Guy et al 1995). Immunohistochemistry has been used to demonstrate EEEV in the brain of infected pheasants (Williams et al 2000) and viral RNA can be detected by RT-PCR in liver, brain, and intestine samples collected from infected birds (Vodkin et al 1993).

The mosquito vector of WEEV is *Culiseta tarsalis* (Chamberlain 1958). WEEV is rarely associated with disease in avian species (Guy and Malkinson 2008) although natural infection has been described in turkey flocks presenting with high mortality and encephalitis

(Woodring 1957) or a sudden onset drop in egg production (Cooper and Medina 1999). Experimental infection of two-week-old turkey poults with WEEV resulted in a short duration (3 day) viraemia with no subsequent mortality or clinical signs (Cooper et al 1997). Earlier studies on experimentally-infected chickens also demonstrated that viraemia lasted no longer than 3 days (Hammon and Reeves 1946). The global distribution is limited to Central America, South America, and western parts of the USA and Canada (Guy and Malkinson 2008).

Natural HJV infection of turkey flocks has been associated with drops in egg production (Wages et al 1993) and mortality of young birds (Ficken et al 1993). Experimental infection of turkeys has been shown to result in clinical and pathological changes which closely resemble those associated with EEEV (Guy et al 1993; Guy et al 1994; Guy and Malkinson 2008).

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). Infection results in a non-purulent meningo-encephalitis, presenting as progressive paralysis (Komarov and Kalmar 1960), clinical signs are only seen in birds older than ten weeks and mortality in affected flocks ranges from 1% to 30% (Samberg et al 1972). Experimental infection of turkeys produces a viraemia lasting 5 to 8 days and virus can be recovered from the brain, blood, spleen and liver of infected birds (Ianculescu et al 1973). The mosquito vector of ITV has not been clearly defined although virus has been isolated from unsorted pools of mosquitoes (*Aedes* spp. and *Culex pipiens*) and *Culicoides* spp. trapped near affected turkey flocks (Braverman et al 1981).

No natural cases of WNV infection have been reported in turkeys (Guy and Malkinson 2008). Experimental infection of three-week-old poults produced a very low level viraemia lasting 2 to 10 days with no clinical signs seen over the duration of the study (Swayne et al 2000).

#### **17.1.5. Hazard identification conclusion**

As natural infection of turkeys has not been described, WNV is not a potential hazard in imported turkey meat. Furthermore, 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.

Sporadic infections of turkey flocks with EEEV, WEEV, HJV and ITV have been described. These infections are characterised by short periods of viraemia and subsequent recovery of virus from a range of body tissues. For the purposes of this risk analysis these four arboviruses are considered to be potential hazards in imported turkey meat.

## **17.2. RISK ASSESSMENT**

### **17.2.1. Entry assessment**

Natural infection of turkey flocks with EEEV, WEEV, HJV and ITV has been described.

Arboviruses have been recovered from a range of body tissues following infection and it is reasonable to assume that virus may be present in the meat of turkeys if infected shortly before slaughter. These infections are characterised by a short-duration low level viraemia so the likelihood of arboviruses being present in imported turkey meat is assessed to be very low but non-negligible.

### 17.2.2. Exposure assessment

Arboviruses replicate in bloodsucking arthropods and are transmitted by bite to a vertebrate host.

Since 1991 New Zealand has operated an arbovirus and *Culicoides* surveillance programme (Tana and Holder 2007). In a typical year seroconversion did not occur in sentinel cattle to bluetongue, epizootic haemorrhagic disease, Akabane and Palyam which are all viruses transmitted by *Culicoides* spp. In addition *Culicoides* spp. were not found in 15,000 insects collected from light traps (Motha et al 1997). The *Culicoides* monitoring programme has continued up to the present time with annual reports of the serology programme appearing in the MAF *Surveillance* magazine. Seroconversion to the arboviruses has not been detected in sentinel cattle and no *Culicoides* have been trapped.

New Zealand has a very limited mosquito population and none of those species recognised in this country are considered to be likely vectors of EEEV, WEEV, HJV or ITV (Holder et al 1999)

There is sufficient evidence to consider New Zealand to be free of the likely arthropod vectors of arboviruses that might be found in imported turkey meat. Furthermore, 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 the likelihood of exposure is negligible.

### 17.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and arboviruses are not assessed to be a hazard in the commodity. Therefore, risk management measures cannot be justified.

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## **18. Turkey viral hepatitis**

### **18.1. HAZARD IDENTIFICATION**

#### **18.1.1. Aetiological agent**

The aetiological agent of turkey viral hepatitis (TVH) has not been characterised (Guy 2008) although it is considered to be a picornavirus-like virus (Klein et al 1991)

#### **18.1.2. OIE list**

Not listed.

#### **18.1.3. New Zealand status**

No records of TVH in New Zealand have been found.

#### **18.1.4. Epidemiology**

TVH has been described in a number of countries including the United States (Snoeyenbos et al 1959), Canada (Mongeau et al 1959), Great Britain (MacDonald et al 1982), and Italy (Mandelli et al 1966). The disease is frequently subclinical and, as there are no serological tests for TVH, the true distribution is unknown (Guy 2008).

TVH is usually not associated with clinical signs other than sporadic mortality of apparently normal birds (Snoeyenbos et al 1959; MacDonald et al 1982; Snoeyenbos 1991). Morbidity and mortality are usually very low although morbidity rates up to 100% have been reported and 25% mortality has been reported in an infected flock. Mortality is restricted to birds under six weeks old (Snoeyenbos 1991). The severity of disease is considered likely to be influenced by the presence of concurrent infections or other stressor agents (Snoeyenbos 1991; Guy 2008). Studies performed in the absence of concomitant factors and in conditions which minimise stress report no remarkable mortality in experimentally infected birds (Klein et al 1991).

Tzianabos and Snoeyenbos (1965) were able to experimentally infect turkeys with TVH and showed that rabbits, chickens, pheasants, and quail are refractory to infection. Transmission between birds is thought to occur principally through faecal exposure (Guy 2008).

Gross and histological lesions in infected birds are confined to the liver and pancreas (Guy 2008). Foci of hepatocytic necrosis and hepatic congestion are consistently reported together with large degenerative foci in the pancreas (Mongeau et al 1959; Snoeyenbos et al 1959; MacDonald et al 1982; Klein et al 1991).

Large aggregates of particles resembling picornaviruses in the cytoplasm of degenerate hepatocytes are visible by electron microscopy (MacDonald et al 1982). Liver, pancreas, and intestinal tissues have been shown to transmit infectivity (Klein et al 1991).

#### **18.1.5. Hazard identification conclusion**

The aetiological agent of TVH has not been fully characterised. Gross and histological lesions and infectivity are restricted to the liver, pancreas, and intestinal tissues and there is no evidence for infectivity elsewhere in the carcass. The agent of TVH is not considered to be a potential hazard in turkey meat and turkey meat products.

Although viscera will be removed from turkey carcasses, remnants may remain following automated processing (Mulqueen 2009; Christensen 2010). Automated eviscerators are quoted to be 87-94% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection. The agent of TVH is therefore considered to be a potential hazard in imported entire turkey carcasses.

## **18.2. RISK ASSESSMENT**

### **18.2.1. Entry assessment**

TVH is a generally subclinical disease of turkeys so it is unlikely that infected birds will be detected during ante-mortem inspection. Individuals with TVH generally have enlarged livers with (possibly extensive) grey focal lesions (Guy 2008) which are likely to be detected during post-mortem inspection of slaughtered birds.

Considering the above, the likelihood of entry in imported turkey carcasses is considered to be low but non-negligible.

### **18.2.2. Exposure assessment**

#### **Backyard poultry**

The agent of TVH survives exposure to 60°C for 6 hours and 56°C for 14 hours (McFerran 1993) so domestic cooking would be unlikely to inactivate any TVH agent contamination of imported turkey carcasses. Rabbits, chickens, pheasants, and quail are refractory to infection with the TVH agent (Tzianabos and Snoeyenbos 1965).

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

Therefore, there is considered to be a non-negligible likelihood of turkeys in a backyard flock being infected with the agent of TVH through exposure to raw or cooked turkey meat scraps generated from imported carcasses.

#### **Wild birds**

Free-living avian species may be exposed to the agent of TVH, either following exposure to an infected backyard turkey flock or through consumption of scraps of cooked or uncooked turkey meat in kitchen waste disposed of at sites accessible to wild avian species. However, there is no evidence for infection of wild birds or other wildlife with the agent of TVH (McFerran 1993).

#### **Commercial poultry**

As described above, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008).

However, the likelihood that commercial poultry will be subject to secondary exposure from infected backyard flocks should be considered.

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, exposure assessment for wild birds and commercial poultry is considered to be negligible. The exposure assessment for turkeys in backyard poultry flocks is considered to be non-negligible.

#### **18.2.3. Consequence assessment**

As described above, TVH has only been recognised in turkeys (Guy 2008). Rabbits, chickens, pheasants, and quail are refractory to infection with the TVH agent (Tzianabos and Snoeyenbos 1965) and there is no evidence for infection of wild birds, other wildlife, or man (McFerran 1993). The consequences of infection would therefore be limited to turkeys.

TVH is generally a subclinical disease although morbidity rates of up to 100% have been reported and a 25% mortality rate was reported in one flock (Guy 2008).

The economic significance of TVH is not known and there is no evidence to suggest that TVH virus is transmissible to human beings or other mammalian species (Guy 2008).

The consequences of the introduction of the TVH agent in imported turkey carcasses are considered to be very low but non-negligible for infected backyard turkey flocks.

#### **18.2.4. Risk estimation**

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

### **18.3. RISK MANAGEMENT**

#### **18.3.1. Options**

The agent of TVH is associated with the liver, pancreas, and intestinal tract of infected birds. Turkey meat products which do not contain remnants of these tissues could be imported without further sanitary measures.

Previously MAF has recommended sourcing birds from flocks with no history of unusually high liver condemnations at slaughter to manage the risk of introducing TVH (MAF 1999).

Given that the agent of TVH survives exposure to 60°C for 6 hours and 56°C for 14 hours, cooking would not be appropriate to manage the risk of introducing the agent of TVH in imported turkey carcasses.

## Option 1

Turkey meat products that do not contain remnants of liver, pancreas, and intestinal tract could be considered eligible for import.

## Option 2

Imported turkey carcasses could be required to originate from flocks with no history of unusually high liver condemnations at slaughter.

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## 19. Marek's disease

### 19.1. HAZARD IDENTIFICATION

#### 19.1.1. Aetiological agent

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

#### 19.1.2. OIE list

Listed.

#### 19.1.3. 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 2009).

#### 19.1.4. 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 two to three 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).

Experimental infection of turkeys has been described, producing variable results. Witter et al (1974) inoculated day-old poults intra-abdominally with four different MDV isolates and reported gross lesions (mainly hepatosplenomegaly) in 14% of the infected birds, whereas another study (Paul et al 1977) reported 70% mortality when day-old poults were experimentally infected. A more recent study (Davidson et al 2002) reported 60-70% mortality in turkeys experimentally infected with MDV isolates.

MD-like tumours have been occasionally reported in turkeys (Andrews and Glover 1939; Voûte and Wagenaar-Schaafsma 1974) and natural outbreaks of the disease in turkeys are considered to be rare (Schat and Venugopal 2008). However, clinical outbreaks of MD in commercial turkey flocks have been reported in Israel (Davidson et al 2002), Germany (Voelckel et al 1999), and Scotland (Pennycott and Venugopal 2002) although close contact between these infected turkey flocks and flocks of chickens are commonly described.

#### **19.1.5. 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 airborne route (Purchase 1976). Virus could persist in the skin (in feather follicles) of imported carcasses 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 should therefore be considered a potential hazard in imported turkey meat.

## **19.2. RISK ASSESSMENT**

### **19.2.1. Entry assessment**

Natural outbreaks of MD in turkeys are considered to be rare although sporadic outbreaks have been reported in commercial turkey flocks throughout the world.

As described above, cell-free infectious MDV is only associated with the epithelial cells of feather follicles so virus may be present in the skin of infected turkeys at slaughter. The processing of turkey carcasses will remove dust and dander and is likely to significantly reduce the amount of virus present on the skin surface.

MDV is inactivated when stored at 4°C for 2 weeks (Calnek and Adldinger 1971). Chilling of turkey 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 very low but non-negligible.

### **19.2.2. Exposure assessment**

#### **Backyard poultry**

Cell-free MDV obtained from the skin of infected chickens is inactivated at 60°C after 10 minutes (Calnek and Adldinger 1971). Therefore, cooking can be considered to inactivate any virus present on the skin of imported turkey carcasses.

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

The route of infection is recognised to be respiratory, by the inhalation of infectious dust. Any uncooked kitchen scraps originating from the preparation of turkey meat will almost certainly be moist, and hence dust-free. The likelihood of backyard poultry becoming infected from virus present on the surface of the skin of raw scraps generated from imported turkey meat is considered to be negligible.

### **Wild birds**

Chickens are by far the most important natural host for MDV, but quail, turkeys, and pheasants are also susceptible to infection and disease. Most other avian species, including ducks, sparrows, partridges, pigeons, and peafowl are probably refractory (Schat and Venugopal 2008).

The likelihood of free-living avian species being infected with MDV, through consumption of uncooked turkey meat in kitchen waste disposed of at sites accessible to wild avian species is assessed to be negligible.

### **Commercial poultry**

As described above, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008).

As the likelihood of free-living avian species or backyard flocks being infected from imported turkey meat is assessed to be negligible, the likelihood of commercial flocks being exposed indirectly to virus is also assessed to be negligible.

### **Exposure assessment conclusion**

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

### **19.2.3. Risk estimation**

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and MDV is not considered to be a hazard in the commodity. Therefore, risk management measures cannot be justified.

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## 20. Pullorum disease and fowl typhoid

### 20.1. HAZARD IDENTIFICATION

#### 20.1.1. 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).

#### 20.1.2. OIE list

Pullorum disease and fowl typhoid are both OIE listed diseases.

#### 20.1.3. 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). There has been no serological survey of commercial turkeys in this country.

A small serological survey of Old English Game fowl in 2005 found no evidence of exposure to *S. Pullorum* (Christensen 2006).

Both *S. Pullorum* and *S. Gallinarum* are listed as unwanted notifiable organisms (MAF 2009).

#### 20.1.4. Epidemiology

*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 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).

Chickens are the natural host for *S. Gallinarum-Pullorum* although according to Shivaprasad and Barrow (2008) outbreaks have rarely been described in turkeys. Pullorum disease was described in turkey poults that had been hatched in an incubator used for hatching chicks (Hewitt 1928) and fowl typhoid has been described in adult turkeys that had contact with infected chickens (Martinaglia 1929; Hinshaw 1930). Johnson and Pollard (1940) also described fowl typhoid in turkey poults. More recently, Iliadis (1987) described *S. Gallinarum-Pullorum* infection of commercial turkeys.

Bassiouni and El-Mossalami (1968) experimentally infected one-year-old turkeys with *S. Gallinarum-Pullorum*. Birds infected orally showed transient infection with the organism recovered from faeces for up to 19 days whereas birds infected intramuscularly laid infected eggs and the organism was recovered from heart blood, liver, gall bladder, ovaries, oviduct, and intestinal contents at necropsy.

Although earlier reports found infection in turkeys where they were in close contact with infected chickens, a survey of poultry kept in Egyptian villages found widespread infection of chickens with *S. Gallinarum-Pullorum* but no infection in local turkeys (Shalaby et al 1981). Similarly, more recent investigations of outbreaks of pullorum disease in chickens in the United States have found no seroconversion in turkeys kept on the same premises (Rhorer 2009).

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). In experimental studies, ducks have been shown to be resistant to infection (Buchholz and Fairbrother 1992; Barrow et al 1999).

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  $1.3 \times 10^9$  to  $10 \times 10^9$  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  $5 \times 10^8$  organisms, although no clinical disease was noted (Badi et al 1992).

### 20.1.5. Hazard identification conclusion

Chickens are recognised as the natural host of *S. Gallinarum-Pullorum* and apart from the report by Iliadis in 1987, the only other descriptions of natural infection of turkeys are around 70 years old. Although *S. Gallinarum-Pullorum* may have been recorded historically in turkeys, there is no evidence of infection in the modern commercial turkey industry.

Recovery of the organism from muscle tissue has not been documented and chicken meat contamination with *S. Gallinarum-Pullorum* has been described only in environments with poor hygiene practices. As described in the commodity definition, imported turkey meat will be derived only from turkeys that have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate effective Good Management Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes.

For the above reasons, *S. Gallinarum-Pullorum* is not assessed to be a potential hazard in the commodity.

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## 21. Paratyphoid infections

### 21.1. HAZARD IDENTIFICATION

#### 21.1.1. 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).

#### 21.1.2. OIE list

The *Code* contains sections concerned with the prevention, detection, and control of *Salmonella* in poultry (Chapter 6.5) and with *Salmonella* Enteritidis and *Salmonella* Typhimurium in poultry (Chapter 6.6). However, paratyphoid infections of poultry are not OIE listed diseases (OIE 2009).

#### 21.1.3. 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 2009a). 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 2009b).

#### 21.1.4. 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).

The vast majority of poultry isolates from turkeys in the USA are either *S. Senftenberg*, *S. Heidelberg*, *S. Hadar*, *S. Muenster*, or *S. Kentucky* (Ferris et al 2003; Nayak et al 2003). *S. Heidelberg* is most prevalent serotype recovered from turkeys in Canada (Guerin et al 2005) and Denmark (Pedersen et al 2002).

A recent survey of the prevalence of *Salmonella* in turkey flocks in the EU cultured five environmental faecal samples from 539 turkey breeding flocks and 3,769 fattening turkey flocks (EFSA 2008). The five most frequently isolated *Salmonella* serovars from fattening turkey flocks in the EU, in decreasing order, were *S. Bredeney*, *S. Hadar*, *S. Derby*, *S. Saintpaul* and *S. Kottbus*. This survey identified a total of 50 different *Salmonella* serotypes in EU turkey flocks, 37 of which have been described in New Zealand (ESR 2003a-2008b).

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 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).

#### **21.1.5. Hazard identification conclusion**

A limited pool of paratyphoid *Salmonella* spp. have been described in turkeys, the 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 turkeys overseas should be considered any more pathogenic than the serotypes recognised as present in New Zealand.

The assessment and management of risks associated with the consumption of imported food is the responsibility of New Zealand Food Safety Authority (NZFSA). NZFSA is accountable for administering the Food Act 1981 and other food safety legislation which applies to all food imported and sold in New Zealand. Imports of turkey products will be required to meet the requirements of food safety legislation in addition to any biosecurity requirements. NZFSA will evaluate food safety risks associated with imported turkey products and make appropriate risk management decisions.

Paratyphoid salmonellae are not considered to be a potential hazard in the commodity.

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## 22. Arizonosis

### 22.1. HAZARD IDENTIFICATION

#### 22.1.1. Aetiological agent

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

*S. arizonae* represent 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).

#### 22.1.2. OIE list

Not listed.

#### 22.1.3. New Zealand status

*S. arizonae* has never been reported in animals or birds in New Zealand (MAF 1999) and is listed as an unwanted exotic organism (MAF 2009).

#### 22.1.4. 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).

Avian arizonosis is most frequently seen in turkeys although infections of chickens (Edwards et al 1947; Silva et al 1980) and ducks (Edwards et al 1956) have occasionally been reported.

*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 as 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).

In adult turkeys, *S. arizonae* is confined primarily to the intestinal tract but has also been recovered from the spleen, gall bladder, lungs, peritoneum, heart, bursa, and kidneys (Hinshaw and McNeil 1946; Sato and Adler 1966a). Vertical transmission has been described (Edwards et al 1943; Bruner and Peckham 1952; Goetz et al 1954; Edwards et al 1959) and the organism has been recovered from the ovaries and oviducts of adult turkeys (Hinshaw and McNeil 1946; Gauger 1946; Sato and Adler 1966a). In addition, faecal contamination of eggs can lead to penetration of the shell and shell membranes (Sato and Adler 1966b; Williams and Dillard 1968).

*S. arizonae* spreads rapidly in an infected flock and shedding from infected birds gradually subsides and stops when birds are 13 to 14 weeks old. Long term carriers have been

described associated with local infections in the peritoneal cavity, air sacs, intestinal wall, or eye (Adler and Rosenwald 1968).

Infected poultts may be listless with anorexia, diarrhoea, leg paralysis, and twisted necks. In addition, corneal opacity, huddling, paralysis, torticollis, opisthotonus, and convulsions have been described. Mortality rates of between 10 and 70% have been reported in poultts and chicks although these can be exacerbated by intercurrent infections (Shivaprasad 2008). Clinical signs are rarely seen in adult birds (Sato and Adler 1966a).

Histopathological examination of infected poultts consistently shows a marked fibrinosuppurative inflammation in the central nervous and ocular tissues (West and Mohanty 1973; Sári et al 1979; Silva et al 1980; Crespo et al 2004) and chronic inflammatory lesions (eosinophilia and/or degeneration) have also been described in the lungs, spleen, heart, liver, pancreas, and kidneys (West and Mohanty 1973).

#### **22.1.5. Hazard identification conclusion**

*S. arizonae* primarily localises to the intestinal tract of adult birds although widespread dissemination of the organism has also been described. This organism has been described in commercial turkeys throughout the world and is considered to be exotic to New Zealand. *S. arizonae* is a potential hazard in the commodity.

### **22.2. RISK ASSESSMENT**

#### **22.2.1. Entry assessment**

In an infected flock, shedding of *S. arizonae* from turkeys 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 carcasses at retail (Izat 1991). The likelihood of entry in imported turkey meat is considered to be low but non-negligible.

#### **22.2.2. Exposure assessment**

##### **Backyard poultry**

In New Zealand, commercial 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 will not feed food scraps to their birds whereas non-commercial poultry flocks containing 100 or fewer birds are not required to have an RMP and are likely to feed food scraps to their birds (Wintle 2010). For the purposes of this risk analysis, backyard flocks are considered to include these non-commercial flocks of 100 or fewer birds.

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 turkey meat after it has been cooked.

*S. arizonae* may be present on scraps of raw turkey 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, 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 turkey meat in kitchen waste disposed of at accessible sites.

### **Commercial poultry**

As described above, there is a negligible likelihood that commercial poultry will be exposed directly to scraps of imported turkey meat. There is a voluntary agreement in place between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand although there are no legislative controls to prevent this (Wintle 2010). If imported turkey meat were to be incorporated into poultry feed it would be subject to rendering which would inactivate any pathogens present in such material (MAFBNZ 2008).

However, the likelihood that commercial poultry will be subject to secondary exposure to *S. arizonae* from infected free-living avian species or backyard flocks should be considered.

#### **Exposure of commercial poultry from free-living avian species**

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 ensure that the likelihood of commercial poultry being exposed to free-living avian species will be very low. 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.

#### **Exposure of commercial poultry from backyard flocks**

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 will 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.

#### **22.2.3. Consequence assessment**

*S. arizonae* is known to infect chickens, ducks, 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.

#### **22.2.4. Risk estimation**

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

### **22.3. RISK MANAGEMENT**

#### **22.3.1. Options**

To effectively manage the risk of imported turkey 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.

Turkey 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 turkey flocks (Snoeyenbos and Smyser 1969; Greenfield and Bigland 1971). Serological tests for turkeys to detect exposure to *S. arizonae* have been described although these have not been entirely effective when used for detecting or controlling arizonosis (Shivaprasad 2008).

Timms (1971) summarised the difficulties encountered in the detection of arizonosis in a turkey flock as follows:

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

Chapter 6.4 of the *Code* (OIE 2009a) describes hygiene and disease security procedures in poultry breeding flocks and hatcheries including guidelines for monitoring poultry breeding flocks and hatcheries for *Salmonella*. Article 6.4.9 requires bacteriological examination of dead in shell and culled chicks at hatcheries and of faeces, dead or culled birds, or chick box liners at breeding flocks. In addition, culture of environmental samples (drag swabs, litter, feather, down, and dust) for *Salmonella* is recommended. Chapter 6.5 for the *Code* (OIE 2009b) 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*.

Turkey meat derived from breeding flocks, hatcheries, and rearing farms that comply with the guidelines in Chapter 6.4 and 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  $5 \times 10^7$  *Salmonella* Typhimurium/ml. Cooking imported turkey meat to a core temperature of 79°C would ensure destruction of *S. arizonae* in the imported commodity.

### Option 1

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

### Option 2

Imported turkey 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 6.4 and 6.5 of the *Code*.

### Option 3

Imported turkey meat could be cooked to reach a core temperature of 79°C.

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## 23. Campylobacteriosis

### 23.1. HAZARD IDENTIFICATION

#### 23.1.1. Aetiological agent

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

#### 23.1.2. OIE list

Not listed.

#### 23.1.3. New Zealand status

No *Campylobacter* spp. are listed on the unwanted organisms register (MAF 2009).

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).

#### 23.1.4. 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 three 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 carcass contamination in retail poultry (Jeffrey et al 2001) and surveys of chicken carcasses 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 carcasses in their study were found to be contaminated with *Campylobacter*, only 14% of turkey carcasses 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).

### 23.1.5. 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 turkeys overseas are more virulent than those found in this country.

The assessment and management of risks associated with the consumption of imported food is the responsibility of New Zealand Food Safety Authority (NZFSA). NZFSA is accountable for administering the Food Act 1981 and other food safety legislation which applies to all food imported and sold in New Zealand. Imports of turkey products will be required to meet the requirements of food safety legislation in addition to any biosecurity requirements. NZFSA will evaluate food safety risks associated with imported turkey products and make appropriate risk management decisions.

For the above reasons, *Campylobacter* spp. are not considered to be potential hazards in the commodity.

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## 24. Colibacillosis

### 24.1. HAZARD IDENTIFICATION

#### 24.1.1. 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).

#### 24.1.2. OIE list

Not listed.

#### 24.1.3. 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, air sac disease, colisepticaemia, coligranuloma, synovitis, and ophthalmitis (Black 1997).

#### 24.1.4. 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 carcass rejections were due to colisepticaemia (Yogarathnam 1995) and *E. coli* was also found to be responsible for the majority of infections resulting in the condemnation of broiler carcasses 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 airsacculitis 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 poultry although *E. coli* O157 has been identified in both chickens (Pilipčinec et al 1999) and turkeys (Heuvelink et al 1999) and human disease has been associated with contaminated turkey meat (Doyle and Schoeni 1987; Griffin and Tauxe 1991). Turkey meat (Johnson et al 2005) and chicken meat (Johnson et al 2003) have also been recognised as a source of *E. coli* with virulence and antimicrobial resistance factors.

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 pathologies in poultry associated with *E. coli* are 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 polyarthrititis) (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).

#### 24.1.5. 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.

The assessment and management of risks associated with the consumption of imported food is the responsibility of New Zealand Food Safety Authority (NZFSA). NZFSA is accountable for administering the Food Act 1981 and other food safety legislation which applies to all food imported and sold in New Zealand. Imports of turkey products will be required to meet the requirements of food safety legislation in addition to any biosecurity requirements. NZFSA will evaluate food safety risks associated with imported turkey products and make appropriate risk management decisions.

APEC is not considered to be a potential hazard in the commodity.

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## 25. Fowl cholera

### 25.1. HAZARD IDENTIFICATION

#### 25.1.1. Aetiological agent

Fowl cholera is caused by *Pasteurella multocida*, a gram-negative, nonmotile, non-spore-forming rod (Glisson et al 2008).

#### 25.1.2. OIE list

Listed

#### 25.1.3. New Zealand status

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 presenting with sudden death and commercial poultry with peritonitis (Anonymous 2000; Bingham 2006). Diagnostic laboratories have recovered *P. multocida* from chickens with ill-thrift and decreased egg production, turkeys with unilateral or bilateral head swelling and mortality, and quail presenting with sudden death (Orr 2000; Varney 2004; Varney 2007).

#### 25.1.4. Epidemiology

Based on bacterial colony morphology when viewed under obliquely transmitted light, Hughes (1930) described 3 types of *P. multocida* from a collection of isolates recovered from cases of fowl cholera – “fluorescent” colonies considered to be highly virulent and associated with acute outbreaks of disease, low virulence “blue” colonies found in flocks where fowl cholera was endemic, and a third “intermediate” type.

Based on DNA homology studies (Mutters et al 1985) three subspecies are recognised (*P. multocida* subsp. *multocida*, *P. multocida* subsp. *septica*, and *P. multocida* subsp. *gallicida*). All three subspecies have been associated with fowl cholera (Snipes et al 1990; Hirsh et al 1990; Fegan et al 1995) although *P. multocida* subsp. *multocida* is the predominant isolate recovered from chickens and turkeys (Glisson et al 2008)

Conventionally, *P. multocida* isolates have been classified based on capsule serogroup antigens by using passive haemagglutination tests (Carter 1955). Five capsular serogroups of *P. multocida* are currently recognised (A, B, D, E, and F) (Rimler and Rhoades 1987). Somatic serotyping has also been used to distinguish *P. multocida* isolates. 16 somatic serotypes are currently described (Brogden et al 1978), with different serovars being predominant in different geographic locations (Glisson et al 2008). More recently, several molecular techniques have also been described to differentiate avian strains of *P. multocida* (Glisson et al 2008). These methods have indicated that wild birds may be a source of infection for domestic poultry (Christensen and Bisgaard 2000).

The presence of a bacterial capsule is considered to be a major virulence factor, although no single factor has been observed to determine bacterial virulence (Christensen and Bisgaard 2000). Endotoxins (Rhoades 1964; Ficken et al 1991; Lee et al 1992), outer membrane proteins (Truscott and Hirsh 1988), iron binding systems (Ogunnariwo et al 1991; Zhao et al 1995), heat shock proteins (Love and Hirsh 1994), neuraminidase production (Ifeanyi and Bailie 1992; Lee et al 1994), and antibody cleaving enzymes (Pouedras et al 1992) have all been suggested as possible virulence factors for *P. multocida*.

The route of infection for *P. multocida* is via the mucous membranes of the pharynx and upper respiratory tract. Birds orally inoculated using virulent strains of *P. multocida* do not become infected (Hughes and Pritchett 1930).

All species of birds are thought to be susceptible to infection. Turkeys are considered to be more susceptible than chickens and clinical disease is most commonly associated with young mature turkeys (Glisson et al 2008). Introduction of chronically infected carriers is thought to be the major source of infection in flocks, with birds harbouring the organism in nasal clefts (Pritchett et al 1930a; Pritchett et al 1930b). The spread of *P. multocida* within a flock occurs via contaminated nasal, oral and conjunctival excretions (Glisson et al 2008) although the organism is also rarely found in faeces (Reis 1941). Although *P. multocida* has been recovered from many farmed species, only those isolates recovered from swine are considered pathogenic in poultry (Glisson et al 2008).

*P. multocida* may become disseminated throughout the carcass of birds that die with acute fowl cholera. The organism has been isolated from the blood of naturally infected chickens up to 49 days before death and can remain viable for 2 months at 5-10°C (Hendrickson and Hilbert 1932). However, *P. multocida* is a fairly delicate organism, which is easily inactivated by common disinfectants, sunlight, drying, or heat (Christensen and Bisgaard 2000).

Acute fowl cholera often presents as sudden death, with signs of fever, anorexia, ruffled feathers, diarrhoea, and oral discharge present for only a few hours preceding this. Those birds that survive this acute septicaemia (and those infected with less virulent strains of *P. multocida*) may go on to develop chronic fowl cholera, characterised by swollen joints, bursitis, swollen wattles, conjunctivitis and torticollis. Birds with chronic fowl cholera may remain infected for long periods, die, or recover. Infected flocks may have 17-68% mortality (Glisson et al 2008).

Acute disease is seen as septicaemia at post mortem, with widespread petechial and ecchymotic haemorrhages, accompanied by peritonitis and pericarditis and necrotic liver foci. Pneumonia is often seen in turkeys (Glisson et al 2008).

Chronic fowl cholera usually present post mortem as localised suppurative infections, often involving the respiratory tract, hock joints, foot pads, peritoneal cavity or oviduct (Glisson et al 2008). Birds showing torticollis may have localised infections in the cranial bones, middle ear, and meninges (Olson et al 1966).

#### **25.1.5. Hazard identification conclusion**

Acute and chronic fowl cholera are recognised in New Zealand. 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.

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*.

*P. multocida* is not considered to be a potential hazard in the commodity.

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## 26. *Riemerella anatipestifer* infection

### 26.1. HAZARD IDENTIFICATION

#### 26.1.1. 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).

#### 26.1.2. OIE list

Not listed.

#### 26.1.3. 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).

#### 26.1.4. 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 three 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 cohemolysin with virulence and demonstrated expression of this cohemolysin 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 turkeys (Zehr and Ostendorf 1970; Helfer and Helmboldt 1977; Smith et al 1987; Frommer et al 1990). *R. anatipestifer* has also been recovered from pheasants (Bruner et al 1970), chickens (Rosenfeld 1973), 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 guillemots, 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 been suggested for turkeys in California (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, airsacculitis, 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).

#### **26.1.5. Hazard identification conclusion**

Infection with *R. anatipestifer* may be accompanied by marked clinical signs in live birds and significant post-mortem pathology. Imported turkey 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 would go undetected.

The history outlined in 26.1.3 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 assessed to be a potential hazard in the commodity.

## **26.2. RISK ASSESSMENT**

### **26.2.1. 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.

### 26.2.2. 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 there is a negligible likelihood of *R. anatipestifer* persisting in scraps of turkey meat following domestic cooking.

Hendrickson and Hilbert (1932) found that feeding pure cultures of *R. anatipestifer* to ducklings over a ten 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 were 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 three 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 ( $0.5 \times 10^2$  CFU to  $0.5 \times 10^6$  CFU) given via the subcutaneous, intravenous, oral, and nasal routes. Whilst inoculation via the intravenous and subcutaneous routes were associated with significant mortality at all challenge doses, there were no deaths associated with oral inoculation using a dose of either  $0.5 \times 10^2$  CFU or  $0.5 \times 10^4$  CFU and only one death (n=11) recorded following oral inoculation with a dose of  $0.5 \times 10^6$  CFU.

Considering the above evidence, there is a negligible likelihood of *R. anatipestifer* being transmitted to susceptible species through the ingestion of uncooked turkey meat scraps. The likelihood of exposure is considered to be negligible.

### 26.2.3. Risk estimation

Since the exposure assessment for *R. anatipestifer* is negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and this organism is not assessed to be a hazard in the commodity. Therefore, risk management measures cannot be justified.

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## 27. *Ornithobacterium rhinotracheale* infection

### 27.1. HAZARD IDENTIFICATION

#### 27.1.1. Aetiological agent

*O. 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).

#### 27.1.2. OIE list

Not listed.

#### 27.1.3. New Zealand status

*O. rhinotracheale* has not been isolated in New Zealand (Black 1997) and is listed as an unwanted exotic organism (MAF 2009)

#### 27.1.4. 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 and turkey isolates. Different serotypes are associated with different geographical origins and pathogenicity varies between isolates (Chin et al 2008).

Outbreaks of disease associated with *O. rhinotracheale* have been reported in turkey flocks throughout the world including Belgium (Devriese et al 1995), Canada (Abdul-Aziz and Weber 1999; Joubert et al 1999), France (Leroy-Sétrin et al 1998), Slovenia (Zorman-Rojs et al 2000), and the United States (Roepke et al 1998). Outbreaks have also been described in chicken flocks in 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 *Chlamydophila psittaci* (Van Loock et al 2005). Experimental infection studies using turkeys have also shown a synergistic effect between *O. rhinotracheale* and avian pneumovirus (Jirjis et al 2004; Marien 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 and turkeys (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 depression, coughing, and decreased feed intake seen in 22-week-old turkeys within 24 hours of experimental infection (Sprenger et al 1998).

Infection of young poults (which usually occurs between 2 and 8 weeks) is associated with mortality rates of 1 to 15% which may rise up to 15% depending on environmental conditions and the presence of concomitant infections. Initial clinical signs include coughing, sneezing, and nasal discharge which may progress to include severe respiratory distress, dyspnoea, and sinusitis. Neurological signs and paralysis have also been reported (Chin et al 2008).

Post mortem findings include oedema and pulmonary consolidation with a fibrinous exudate on the pleura. These lesions may be accompanied by fibrinosuppurative airsaccultitis, pericarditis, peritonitis, and mild tracheitis (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 organisms 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).

#### **27.1.5. 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, pathological lesions and infectivity are restricted mainly to the respiratory tissues. *O. rhinotracheale* is not considered to be a potential hazard in those commodities that exclude respiratory tract material, namely turkey meat and turkey meat products.

Although respiratory tract tissues will be removed from turkey carcasses, remnants of these tissues may remain following automated processing (Mulqueen 2009; Christensen 2010). Automated lung removal machinery is quoted to be 90-92% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection. *O. rhinotracheale* is therefore considered to be a potential hazard in imported entire turkey carcasses.

## **27.2. RISK ASSESSMENT**

### **27.2.1. Entry assessment**

The clinical signs associated with *O. rhinotracheale* infection are extremely variable (Chin et al 2008) so it is unlikely that an infected flock 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 carcasses (MAF 1999). In the absence of any evidence to the contrary, it is assumed that a similar figure would apply to turkey carcasses.

Considering the above, the likelihood of *O. rhinotracheale* entry in imported turkey carcasses is considered to be very low but non-negligible.

### 27.2.2. 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 considered that there is a negligible likelihood of *O. rhinotracheale* persisting in scraps of turkey meat following domestic cooking.

Any respiratory tissue remnants in imported turkey carcasses 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 turkey meat discarded from imported carcasses would not transmit infection so the likelihood of exposure is considered to be negligible.

### 27.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and *O. rhinotracheale* is not assessed to be a hazard in the commodity. Therefore, risk management measures cannot be justified.

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## 28. Bordetellosis (turkey coryza)

### 28.1. HAZARD IDENTIFICATION

#### 28.1.1. Aetiological agent

*Bordetella avium* is a gram-negative, nonfermentative, motile, strictly anaerobic bacillus, previously described as *Alcaligenes faecalis* (Jackwood and Saif 2008).

#### 28.1.2. OIE list

Not listed.

#### 28.1.3. New Zealand status

*B. avium* has not been isolated in New Zealand (Black 1997) and is listed as an unwanted exotic organism (MAF 2009).

#### 28.1.4. Epidemiology

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).

Turkeys are considered to be the natural host of *B. avium* although the organism has also been recovered from chickens and 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)

Disease is usually seen in turkeys from 2 to 6 weeks (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).

#### **28.1.5. 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 considered to be a potential hazard in those commodities that exclude upper respiratory tract material, namely turkey meat and turkey meat products.

Although respiratory tract tissues will be removed from turkey carcasses, remnants of these tissues may remain following automated processing (Mulqueen 2009; Christensen 2010). Automated lung removal machinery is quoted to be 90-92% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection. *B. avium* is therefore considered to be a potential hazard in imported entire turkey carcasses.

## **28.2. RISK ASSESSMENT**

### **28.2.1. Entry assessment**

Infection with *B. avium* may be associated with mild clinical signs unless concomitant infections are present so it is unlikely that an infected flock 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 carcasses (MAF 1999). In the absence of any evidence to the contrary, it is assumed that a similar figure would apply to turkey carcasses.

Considering the above, the likelihood of *B. avium* entry in imported turkey carcasses is considered to be very low but non-negligible.

### **28.2.2. 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 considered that there is a negligible likelihood of *B. avium* persisting in scraps of turkey meat following domestic cooking.

Any respiratory tissue remnants in imported turkey carcasses 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 turkey meat discarded from imported carcasses would not transmit infection.

The likelihood of exposure is considered to be negligible.

### 28.2.3. Risk estimation

Since the exposure assessment is negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and *B. avium* is not assessed to be a hazard in the commodity. Therefore, risk management measures cannot be justified.

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## 29. *Mycoplasma* spp. infections

### 29.1. HAZARD IDENTIFICATION

#### 29.1.1. Aetiological agent

*Mycoplasma meleagridis*, *M. iowae*, *M. gallisepticum*, *M. imitans*, *M. gallinarum*, *M. pullorum*, *M. synoviae* and *Ureaplasma* spp. Small prokaryotes devoid of a cell wall, bounded only by a plasma membrane (Kleven 2008).

#### 29.1.2. OIE list

Avian mycoplasmosis due to *M. gallisepticum* or *M. synoviae* are listed diseases.

#### 29.1.3. New Zealand status

Serological evidence of exposure to *M. meleagridis* was reported in a commercial turkey flock during the investigation of airsaccultitis in 1969 (Pohl 1969) and, historically, serosurveillance routinely demonstrated exposure of turkeys to this organism (Anonymous 1994a - 1996d). However, since 1997 surveys of commercial turkey flocks in New Zealand have found no serological evidence of *M. meleagridis* infection (Anonymous 1997a - 2001; Poland 2004; Poland 2005; Tana 2007).

*M. iowae* is listed as an unwanted exotic organism.

*M. gallisepticum* and *M. synoviae* are both present in New Zealand (Black 1997) although it has been suggested that more virulent strains of *M. gallisepticum* may be present overseas (Christensen 2010). 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 it is reasonable to assume that exotic strains of *M. gallisepticum* may be more virulent than those currently present (Anonymous 1994a -1999).

No records could be found of the recovery of *M. imitans*, *M. gallinarum*, *M. pullorum*, or *Ureaplasma* spp. from New Zealand poultry although there have been no surveys to look for these organisms.

#### 29.1.4. Epidemiology

##### *M. meleagridis*

*M. meleagridis* is considered to be a common pathogen of turkeys found worldwide including Australia (Grimes 1972; Rosenfeld and Grimes 1972), Canada (Bigland and Benson 1968; Bigland 1969), Guatemala (Mátzer Ovalle 1972), Japan (Shimizu and Yagihashi 1980), the United Kingdom (Wise et al 1973), and the United States (Adler et al 1958), although efforts over the last twenty years have significantly reduced the prevalence of *M. meleagridis* in the major turkey-producing areas of the world (Chin et al 2008).

*M. meleagridis* is only pathogenic in turkeys. Chickens have been shown to be refractory to infection (Adler 1958; Yamamoto and Bigland 1964; Yamamoto et al 1965). *M. meleagridis* has been isolated from free-ranging birds of prey in Germany with no clinical signs of respiratory disease (Lierž et al 2000) and serological surveys have found evidence of infection in less than 3% of lesser prairie-chickens in Kansas (Hagen et al 2002) and in a peafowl in Michigan (Hollamby et al 2003).

Vertical transmission is primarily responsible for the spread of *M. meleagridis*, with hens being infected during their embryonic development. Insemination with mycoplasma-contaminated semen also plays a major role in sustaining the egg-transmission rate during the laying season (Kumar and Pomeroy 1969; Yamamoto and Ortmayer 1969; Kleven and Pomeroy 1971; Matzer and Yamamoto 1974). Horizontal transmission of *M. meleagridis* has been described in a hatchery (Kumar and Pomeroy 1969), flock (Yamamoto and Ortmayer 1967), or between flocks (Yan Ghazikhanian et al 1980). Infection due to horizontal transmission usually results in localised infections of the sinus and trachea (Mohamed and Bohl 1967; Yamamoto and Ortmayer 1967; Kumar and Pomeroy 1969).

Infection of poult with *M. meleagridis* results in a high rate of airsaccultitis, which is rarely accompanied by clinical signs. *M. meleagridis* has no effect on egg production or fertility although it has been associated with late incubation mortality in naturally infected turkey embryos, and has been estimated to produce a 5-6% loss of fertile eggs. Airsaccultitis may result in carcase condemnations at slaughter, especially where co-infections or exacerbating environmental factors are present. *M. meleagridis* may also adversely affect weight gain in infected poults (Chin et al 2008).

Airsaccultitis deficiency syndrome (also known as Turkey Y disease or Turkey syndrome '65) is characterised by stunting, poor feathering, and leg bone abnormalities and has been associated with *M. meleagridis* infection (Gordon et al 1965, Wise et al 1973). However, this syndrome has also been reproduced using *M. gallisepticum* isolates (Wannop and Butler 1968; Wannop et al 1971) and is likely to be due to a combination of nutritional factors alongside a mycoplasmal infectious component (Grasso 1968; Wannop et al 1971; Wise et al 1973). Airsaccultitis deficiency syndrome has been described in New Zealand (Pohl 1969).

*M. meleagridis* may also act synergistically with other mycoplasma isolates to produce a severe form of airsaccultitis (Rhoades 1981) and *M. meleagridis* has been shown to act synergistically with *M. synoviae* to produce synovitis in experimentally infected turkeys (Rhoades 1977).

A study of 300 naturally-infected turkey embryos demonstrated that *M. meleagridis* localises to the respiratory tissues, with the organism detected in the sinus, peritoneum, lung, trachea and air sacs (Bigland 1972) and experimental infection of embryos has shown that the organism also localises to the intestine and bursa of Fabricius (Reis and Yamamoto 1971). Although gross lesions in natural infections are limited to the air sacs (Chin et al 2008), experimental infections using *M. meleagridis* have been associated with sternal bursitis (Yamamoto and Bigland 1965), synovitis (Saif et al 1970), and ascites (Wise and Fuller 1975).

### ***M. iowae***

*M. iowae* is found worldwide including Europe (Jordan and Amin 1980; Benčina et al 1987a), India (Rathore et al 1979), Japan (Shimizu et al 1979), and the United States (Yoder and Hofstad 1962).

The natural host of *M. iowae* is the turkey (Bradbury and Kleven 2008) although it is also found in chickens (Yoder and Hofstad 1962; Benčina et al 1987a) and parrots (Bozeman et al 1984), as well as in geese and other exotic birds (Bradbury and Kleven 2008).

Transmission of *M. iowae* is predominantly vertical although infected semen may also play a role in dissemination of the organism (Bradbury and Kleven 2008).

*M. iowae* is not associated with clinical disease in live turkeys although infection is associated with a reduction in poult hatchability in the order of 2% to 5% (Bradbury and Kleven 2008) and one report has associated *M. iowae* with leg weakness (Trampel and Goll 1994).

Following experimental infection of one-day-old poults, *M. iowae* was recovered predominantly from the oesophagus and air sacs of live birds although isolations became less frequent with age and the organism could not be recovered from birds over 12 weeks (Bradbury et al 1988). *M. iowae* has also been recovered from the semen and phallus of sexually mature toms (Shah-Majid and Rosendal 1986) and from the oviduct of chickens (Yoder and Hofstad 1962; Rathore et al 1979).

### ***M. gallisepticum***

*M. gallisepticum* has a world-wide distribution (Levisohn and Kleven 2000) and naturally occurs primarily in gallinaceous birds, especially commercial chickens and turkeys (Ley 2008). *M. gallisepticum* has also been recovered from pheasants, chukar partridge, peafowl, and Japanese quail (Reece et al 1986b; Cookson and Shivaprasad 1994; Murakami et al 2002; Benčina et al 2003) as well as from ducks (Benčina et al 1988), geese (Buntz et al 1986), a yellow-naped Amazon parrot (Bozeman et al 1984), and greater flamingos and white pelicans (El-Shater 1996). PCR testing has detected *M. gallisepticum* DNA in mature rooks in Scotland (Pennycott et al 2005).

In 1994 *M. gallisepticum* was recognised as the cause of peri-orbital swelling and conjunctivitis in free-ranging house finches in the United States (Ley et al 1996; Luttrell et al 1996; Fischer et al 1997; Luttrell et al 1998). Conjunctivitis associated with *M. gallisepticum* infection was subsequently also reported in a blue jay, a purple finch, and goldfinches in the United States (Ley et al 1997; Hartup et al 2000) and in evening gosbeaks and pine gosbeaks in Canada (Mikaelian et al 2001).

Isolates and strains of *M. gallisepticum* vary widely in their relative pathogenicity (Ley 2008). For example the R strain of *M. gallisepticum* is used for challenge studies (Glisson et al 1989; Kleven et al 1998). Low passage strains of this organism ( $R_{low}$ ) are pathogenic and capable of adhesion and cell invasion whereas high passage strains ( $R_{high}$ ) are avirulent (Papazisi et al 2002; May et al 2006). Following infection  $R_{low}$  strains can be recovered from internal organs whereas  $R_{high}$  strains cannot (Much et al 2002).

The incubation period varies from 6 to 21 days depending on strain virulence although sinusitis will often develop in turkeys within 6-10 days of experimental exposure (Ley 2008). Transmission of *M. gallisepticum* occurs via the upper respiratory tract or conjunctiva following exposure to aerosols or droplets although the organism cannot survive outside the host for more than a few days (Ley 2008). Vertical transmission of *M. gallisepticum* is recognised (Lin and Kleven 1982; Glisson and Kleven 1985; Ortiz et al 1995).

In the United States, backyard flocks (McBride et al 1991; Ewing et al 1996), multi-age commercial layer flocks (Kleven 1996) and free-ranging songbirds (Fischer et al 1997; Ley et al 1997; Ley et al 2006) are considered potential reservoirs of the organism. However, Stallknecht et al (1998) were able to show that transmission of *M. gallisepticum* from infected finches to naïve chickens required direct contact over a period of at least 10 weeks and that minimal biosecurity measures to restrict direct contact between chickens and finches should significantly reduce the potential for transmission.

Following infection turkeys develop sinusitis, respiratory distress, depression, decreased feed intake, and weight loss. Clinical signs, morbidity and mortality can be highly variable in infected flocks (Ley 2008). Concurrent infections or environmental stressors are associated with more severe disease (Kleven 1998).

Infection leads to catarrhal exudate in the nasal and paranasal passages, trachea, bronchi, and air sacs and a caseous exudate may be found in the air sacs. Microscopically, there is a marked thickening of the mucous membranes due to mononuclear infiltration and mucous gland hyperplasia (Hitchner 1949). Encephalitis has also been reported in turkeys infected with *M. gallisepticum* (Chin et al 1991). The upper respiratory tract and conjunctiva are generally accepted as the portals of entry for naturally acquired *M. gallisepticum* infections and the organism is considered to be a surface parasite of the respiratory tract and conjunctiva (Ley 2008). However, transient systemic infections have been described which may result in infection at other sites (Thomas et al 1966; Chin et al 1991).

*M. gallisepticum* can be cultured from suspensions of tracheal or air sac exudates, turbinates, lungs, or sinus exudate and has also been recovered from the oviduct and cloaca of infected birds (Domermuth et al 1967; Amin and Jordan 1979; MacOwan et al 1983; Nunoya et al 1997). More virulent strains are more likely to be recovered from a wider range of tissues including the bursa, spleen, liver and kidney following experimental infection (Varley and Jordan 1978).

#### **Other *Mycoplasma* spp. and *Ureaplasma* spp.**

*M. imitans* has been isolated from ducks, geese, and partridges (Kleven and Ferguson-Noel 2008). Experimental infection of day-old turkey poults with a partridge isolate of *M. imitans* that had been passaged through turkey poults results in mild upper respiratory tract disease, with the organism establishing in the sinuses, eyes, and upper trachea (Ganapathy et al 1998). Natural infection of commercial turkeys has not been described.

*M. gallinarum* is found throughout the world in domestic poultry, including turkeys (Jordan and Amin 1980; Benčina et al 1987b), as well as in jungle fowl (Shah-Majid 1987), ducks (El Ebeedy et al 1987), and pigeons (Reece et al 1986a). *M. gallinarum* is generally not considered to be a pathogenic mycoplasma although one report has linked this organism to airsaccultitis in broiler chickens (Kleven et al 1978).

*M. pullorum* has been isolated from chickens, quail, partridge, pheasants and turkeys (Kleven and Ferguson-Noel 2008). An isolate of *M. pullorum* was recovered from a specific-pathogen-free turkey flock in France that was able to induce embryonic mortality when experimentally inoculated into chicken and turkey eggs (Moalic et al 1997). Natural infection of commercial turkeys has not been described.

A number of reports have described the recovery of ureaplasma strains from poultry (Koshimizu et al 1982; Harasawa et al 1985). An investigation of infertility in a turkey-breeding farm in Eastern Europe was associated with the presence of ureaplasma in tom semen (Stipkovits et al 1983) and experimental infection of turkeys and chickens with a ureaplasma isolate was reported to result in mild upper respiratory clinical signs with a serofibrinous airsaccultitis and peritonitis seen at necropsy (Stipkovits et al 1978).

#### **29.1.5. Hazard identification conclusion**

There is historical evidence for the presence of *M. meleagridis* in commercial turkeys in New Zealand although more recent surveys have consistently found no evidence of exposure to this organism. *M. meleagridis* localises predominantly in the tissues of the respiratory tract.

Following infection, *M. iowae* has a limited tissue distribution in turkeys and can only be recovered from the reproductive tract of adult birds.

*M. gallisepticum* can be found predominantly in respiratory tissues although more virulent strains may disseminate more widely following infection.

There is little evidence to suggest that other *Mycoplasma* spp. and *Ureaplasma* spp. have a pathogenic role in natural infections. Where experimental infections have resulted in clinical disease, these organisms are confined to the upper respiratory tract tissues.

*Mycoplasma* spp. and *Ureaplasma* spp. are not considered to be potential hazards in those commodities that exclude upper respiratory tract material, reproductive tract tissues and abdominal viscera (turkey meat and turkey meat products).

Although upper respiratory tract material, reproductive tract tissues and abdominal viscera will be removed from turkey carcasses, remnants of these tissues may remain following automated processing (Mulqueen 2009; Christensen 2010). Automated lung removal machinery is quoted to be 90-92% efficient whilst automated eviscerators could be expected to be 87-94% efficient (Land 2010) although some of the remaining tissue could be removed during manual inspection. *M. meleagridis*, *M. iowae*, exotic strains of *M. gallisepticum*, *M. imitans*, *M. gallinarum*, *M. pullorum*, and *Ureaplasma* spp. are therefore considered to be potential hazards in imported entire turkey carcasses.

## 29.2. RISK ASSESSMENT

### 29.2.1. Entry assessment

Mycoplasmal infections are rarely associated with marked clinical signs unless accompanied by concurrent infections or environmental stressors. However, airsacculitis may result in carcass condemnations at slaughter.

It is generally accepted that organisms belonging to the *Mollicutes* class are unstable and die rapidly in liquid media. However, it is also known that mycoplasmas can exist for a long period within or on animal tissues (Nagatomo et al 2001). Chandiramani et al (1966) intravenously inoculated chickens with  $3 \times 10^9$  to  $1.2 \times 10^{10}$  *M. gallisepticum* organisms and demonstrated that the organism could be recovered from muscle tissue for up to 49 days if stored at 6°C and from whole carcasses for up to 4 weeks when stored under conditions varying between 2°C and 24°C.

Although experimental infections have been associated with more widespread dissemination, *Mycoplasma* spp. and *Ureaplasma* spp. localise principally to respiratory and reproductive tissues following natural infection. As noted above, remnants of these tissues may remain following automated processing as automated lung removal machinery is quoted to be 90-92% efficient whilst automated eviscerators could be expected to be 87-94% efficient.

Considering the above, the likelihood of *M. meleagridis*, *M. iowae*, exotic strains of *M. gallisepticum*, *M. imitans*, *M. gallinarum*, *M. pullorum*, and *Ureaplasma* spp. entry in imported turkey carcasses is considered to be very low but non-negligible.

### 29.2.2. 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 considered that there is a negligible likelihood of *Mycoplasma* spp. or *Ureaplasma* spp. persisting in scraps of turkey meat following domestic cooking.

Any respiratory or reproductive tissue remnants in imported turkey carcasses 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.

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). Fresh or frozen poultry meat products produced for human consumption are not ordinarily considered risks for *M. gallisepticum* infection (Levisohn and Kleven 2000).

Considering the above, the likelihood of exposure for *M. meleagridis*, *M. iowae*, exotic strains of *M. gallisepticum*, *M. imitans*, *M. gallinarum*, *M. pullorum*, and *Ureaplasma* spp. is considered to be negligible.

### 29.2.3. Risk estimation

Since the exposure assessment for *M. meleagridis*, *M. iowae*, exotic strains of *M. gallisepticum*, *M. imitans*, *M. gallinarum*, *M. pullorum*, and *Ureaplasma* spp is negligible, under the methodology used in this risk analysis (see Section 4.3) the risk estimation is negligible and these organisms are not assessed to be hazards in the commodity.

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## 30. Avian intestinal spirochaetosis

### 30.1. HAZARD IDENTIFICATION

#### 30.1.1. 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).

#### 30.1.2. OIE list

Not listed.

#### 30.1.3. 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).

#### 30.1.4. 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).

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 ceca 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).

#### 30.1.5. Hazard identification conclusion

Avian intestinal spirochaetosis is principally a disease of chickens. However, the disease has been described in commercial turkeys although it was associated with *B. pilosicoli*, which is recognised as being present in New Zealand.

Infectivity is confined to the lower intestinal tract, which is removed from the commodities under consideration here.

Reflecting the above, the agents of avian intestinal spirochaetosis are assessed not to be potential hazards.

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## 31. Tuberculosis

### 31.1. HAZARD IDENTIFICATION

#### 31.1.1. Aetiological agent

Avian tuberculosis is caused by *Mycobacterium avium* (Fulton and Sanchez 2008). At least 28 serovars of *M. avium* are recognised (Wayne et al 1993). *M. avium* is further subdivided into *M. avium* sbsps. *paratuberculosis*, *M. avium* sbsps. *silvaticum*, and *M. avium* subsp. *avium*.

Avian tuberculosis in poultry is associated with *M. avium* sbsps. *avium* serovars 1, 2, and 3 (Thoen et al 1981).

#### 31.1.2. OIE list

Not listed.

#### 31.1.3. New Zealand status

Avian tuberculosis is recognised in New Zealand poultry (McCausland 1972; Black 1997) and is considered to be reasonably common in the minority of free range layer flocks where the average age of birds exceeds two years (Christensen 2010). Tuberculosis has also been diagnosed following necropsy of a captive kiwi and faecal sampling identified another kiwi excreting *M. avium* (Davis et al 1984).

#### 31.1.4. Epidemiology

Avian tuberculosis is found throughout the world and is rarely diagnosed in commercial poultry. Agricultural statistics from the United States show that no more than 0.001 % of young chickens, 0.02% of mature chickens and 0.0000004% of mature turkeys were condemned at slaughter because of avian tuberculosis (Fulton and Sanchez 2008).

Avian tuberculosis has been described in turkeys (Hinshaw et al 1932) although it is very uncommon and usually contracted from infected chickens (Fulton and Sanchez 2008). Infection of birds in zoological collections with *M. avium* is much more common than infection of domestic poultry (Montali et al 1976).

The principal means of spread of the organism is through infected faeces containing vast numbers of tubercle bacilli from ulcerated tuberculous lesions in the intestines of infected poultry. *M. avium* can also be transmitted in carcasses of tuberculous fowl (Fulton and Sanchez 2008).

Clinical signs associated with infection include progressive weight loss, icterus, lameness, and paralysis. Gross lesions (multiple granulomas) can be seen in the liver, spleen, intestine, and bone marrow and, less frequently, in the heart, ovaries, testes, and skin. In turkeys, lesions are seen mainly in the liver and spleen (Fulton and Sanchez 2008).

#### 31.1.5. Hazard identification conclusion

Avian tuberculosis is extremely rare in commercial poultry, especially turkeys. Gross lesions in infected birds are likely to be easily spotted at slaughter. Imported turkey meat will be derived from birds that have passed ante-mortem and post-mortem inspection.

Furthermore, 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.

For the above reasons, avian tuberculosis is assessed not to be a potential hazard.

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## **32. *Aegyptianella* spp.**

### **32.1. HAZARD IDENTIFICATION**

#### **32.1.1. Aetiological agent**

*Aegyptianella* spp. are obligate intracellular organisms in the family Anaplasmataceae (Barnes and Nolan 2008).

#### **32.1.2. OIE list**

Not listed.

#### **32.1.3. New Zealand status**

*Aegyptianella* spp. are listed as unwanted exotic organisms (MAF 2009).

#### **32.1.4. 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 exotic to New Zealand (McKenna 1996; Loth 2005)

#### **32.1.5. 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 considered exotic to New Zealand.

For the above reasons, *Aegyptianella* spp. are assessed not to be potential hazards.

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## 33. *Borrelia* spp.

### 33.1. HAZARD IDENTIFICATION

#### 33.1.1. Aetiological agent

*Borrelia anserina* causes borreliosis in a number of avian species including chickens, turkeys, pheasants, geese, and ducks (Barnes and Nolan 2008).

#### 33.1.2. OIE list

Not listed.

#### 33.1.3. New Zealand status

*Borrelia anserina* is listed as an unwanted exotic organism (MAF 2009).

#### 33.1.4. 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 exotic to 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).

#### 33.1.5. 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 considered exotic to New Zealand.

For the above reasons, *B. anserina* is assessed not to be a potential hazard.

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## 34. Long-segmented filamentous organisms

### 34.1. HAZARD IDENTIFICATION

#### 34.1.1. 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 generic name for this group of organisms (Snel et al 1995).

#### 34.1.2. OIE list

Not listed.

#### 34.1.3. New Zealand status

No reports have been found of LSFO infections in New Zealand poultry.

#### 34.1.4. 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 turkeys including diarrhoea, huddling, poor growth, and increased mortality, 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)

One experimental study did associate LSFOs with a stunting syndrome in turkey poults (Angel et al 1990) although a subsequent study by this group using filtered inoculates demonstrated that LSFOs were not primary causative agents of this disease (Sell et al 1992). Other experimental studies have suggested that LSFO infections may be associated with a depression in the growth rate of turkey poults (Morishita et al 1992).

#### 34.1.5. 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 assessed not to be a potential hazard.

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## 35. Avian chlamydiosis

### 35.1. HAZARD IDENTIFICATION

#### 35.1.1. Aetiological agent

Obligate intracellular gram-negative bacteria, *Chlamydophila psittaci* (Andersen and Vanrompay 2008).

#### 35.1.2. OIE list

Listed.

#### 35.1.3. 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 feral pigeons, with a prevalence rate of between 9.5% and 25% (Motha et al 1995).

A survey of faecal samples from captive and wild endangered and threatened avian species was reported to have detected *C. psittaci* in a number of species, including kakapo, takahe, and kiwi although it was subsequently suggested that a large number of these findings were false positive results due to the choice of test (Motha et al 1995).

#### 35.1.4. Epidemiology

There are 8 known serovars of *C. psittaci*, with serotypes D and E mainly associated with turkeys although other serovars have also been isolated from this species. Serovar D is considered to be highly virulent and especially noted to be a risk for veterinarians and poultry workers (Andersen and Vanrompay 2008).

*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).

Highly virulent strains of *C. psittaci* cause acute disease epidemics resulting in the death of 5-30% of affected birds whilst less virulent strains cause slowly progressive epidemics. Highly virulent serovar D strains are most often isolated from turkeys (Winsor and Grimes 1988) although less virulent strains are occasionally recorded in turkeys (Tappe et al 1989).

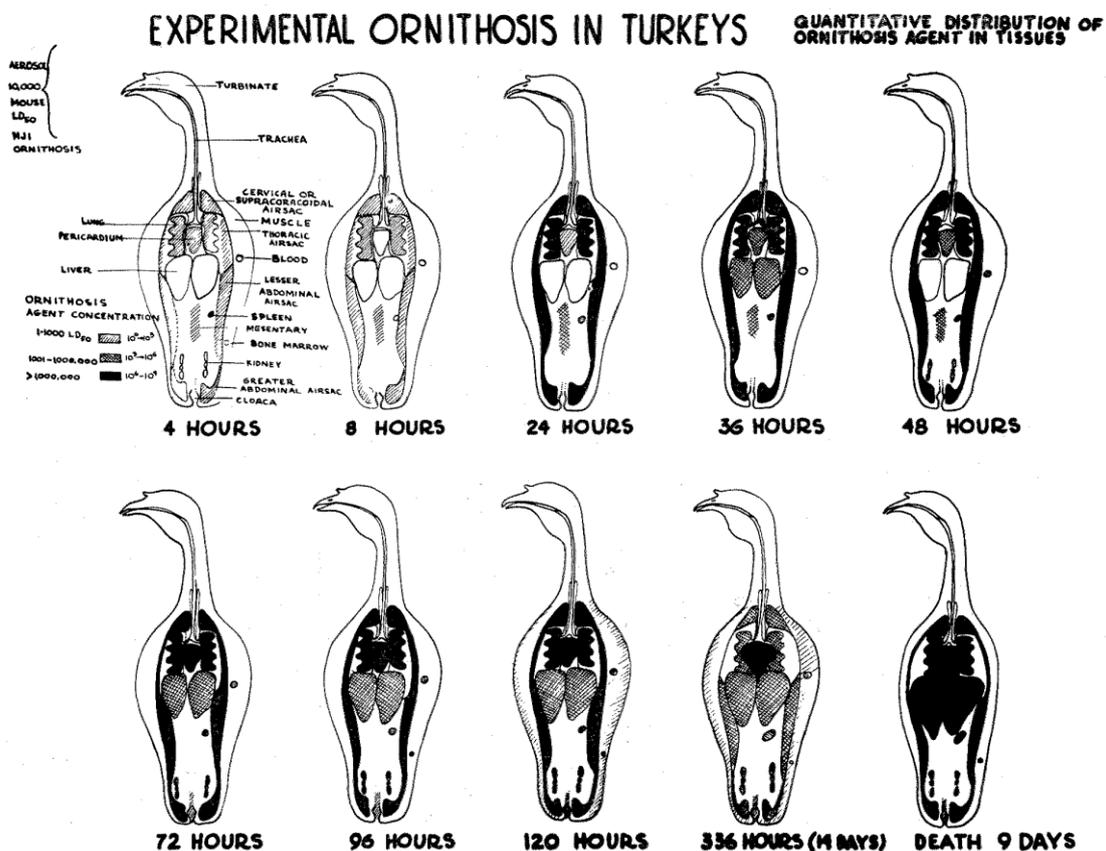
Historically, chlamydiosis in turkeys has been associated with explosive outbreaks in free-ranging birds with high mortality associated with serovar D strains of *C. psittaci*. However, more recent studies indicate that less virulent strains may be endemic in commercial turkeys and only result in respiratory disease in association with other agents such as avian pneumovirus or *Ornithobacterium rhinotracheale* (Van Loock et al 2005; Andersen and Vanrompay 2008).

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<sub>50</sub>. 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 1995b). 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)



Gross lesions associated with *C. psittaci* infection include pulmonary congestion and cardiomegaly together with a fibrinous pleuritis and fibrinous pericarditis. These lesions in the heart and lungs are considered to be the major cause of mortality in disease outbreaks. Other lesions seen include hepatosplenomegaly, thickening of the air sacs, and a fibrinous peritonitis (Andersen and Vanrompay 2008).

Histological studies of infected turkeys have described ocular lesions (epithelial erosions and fibrin deposits in the conjunctivae and corneal ulcerations), and cardiac lesions (fibrinous pericarditis and myocarditis), as well as lesions in the respiratory tract (bronchopneumonia, fibrinous necrotising airsaccultitis, epithelial pneumonitis and tracheitis), and lesions in the

abdominal viscera (interstitial nephritis, hepatitis, peritonitis, splenitis, orchitis, and catarrhal enteritis) (Beasley and Grumbles 1959; Vanrompay et al 1995a).

Clinical signs described following infection with a virulent strain of *C. psittaci* include cachexia, pyrexia, anorexia, conjunctivitis, and respiratory distress. Laying hens may show an acute 10-20% drop in egg production. Less virulent strains are associated with anorexia, loose droppings and a less marked decline in egg production. 50-80% of birds in a flock infected with a virulent strain may show clinical signs with 10-30% mortality. Less virulent strains are associated with 5-20% morbidity and 1-4% mortality (Andersen and Vanrompay 2008).

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).

#### **35.1.5. Hazard identification conclusion**

It is not known which serovars of *C. psittaci* are present in New Zealand although there are no reports of explosive disease outbreaks in turkey flocks that would normally be associated with virulent serovar D strains of *C. psittaci*. It is therefore assumed that virulent strains of the organism associated with turkeys in other countries do not occur in New Zealand.

Infectivity is concentrated in the respiratory tissues and intestinal tract, which are both removed from the commodities under consideration. However, some infectivity can be detected in muscle and renal tissues.

Infection with a highly virulent strain would be likely to result in carcass 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 assessed to be a potential hazard in the commodity.

### **35.2. RISK ASSESSMENT**

#### **35.2.1. 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 not 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).

Reflecting the above, the likelihood of entry of viable *C. psittaci* is assessed to be negligible.

### 35.2.2. Risk estimation

Since the entry assessment for *C. psittaci* associated with turkey meat from all countries is assessed to be negligible, the risk is estimated to be negligible and sanitary measures cannot be justified.

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## 36. Dermatophytosis (favus)

### 36.1. HAZARD IDENTIFICATION

#### 36.1.1. Aetiological agent

Initially identified as *Epidermophyton gallinae*, then *Achorion gallinae* and *Trichophyton gallinae*. The aetiological agent is currently described as *Microsporum gallinae* (Charlton et al 2008).

#### 36.1.2. OIE list

Not listed.

#### 36.1.3. New Zealand status

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).

#### 36.1.4. Epidemiology

Ringworm (favus) is a fungal condition of the skin caused principally by *M. gallinae* in poultry. The disease is found sporadically worldwide where it appears most commonly in backyard flocks (Fonseca and Mendoza 1984; Droual et al 1991; Bradley et al 1993). The disease is rare in large-scale commercial operations (Charlton et al 2008).

Following infection, *M. gallinae* colonises the epidermis and results in epidermal hyperplasia and hyperkeratosis initially in the unfeathered skin (comb, wattle, shanks). Lesions slowly expand concentrically and infection is confined to the non-viable superficial layers of the skin. Infection is usually limited to individual animals in a group and is spread slowly to other birds by direct contact (Brown and Jordan 2001; Charlton et al 2008)

The majority of reports of favus are found in early literature and few reports of this disease exist in recent publications (Droual et al 1991; Bradley et al 1993). An extensive literature search found no reports of dermatophytosis in commercial turkey farms.

#### 36.1.5. Hazard identification conclusion

Dermatophytes have a worldwide distribution. Although there have been no surveys of poultry in New Zealand that have tested for *M. gallinae* it is unlikely that this organism can be considered exotic.

*M. gallinae* is considered rare in modern commercial poultry farms and there are no records of *M. gallinae* in commercial turkey flocks.

Reflecting the above, *M. gallinae* is assessed not to be a potential hazard.

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## 37. Histoplasmosis

### 37.1. HAZARD IDENTIFICATION

#### 37.1.1. Aetiological agent

*Histoplasma capsulatum* var *capsulatum*, a dimorphic facultative intracellular fungal parasite (Rosas-Rosas et al 2004).

#### 37.1.2. OIE list

Not listed.

#### 37.1.3. New Zealand status

*Histoplasma capsulatum* var *farciminosum* (epizootic lymphangitis) is listed as an unwanted notifiable organism (MAF 2009).

No record could be found of histoplasmosis affecting animals in New Zealand and *Histoplasma capsulatum* is considered to be exotic (Hill 1999).

#### 37.1.4. Epidemiology

*H. capsulatum* var *capsulatum* is found mainly in areas with humid temperate climates. Infection is acquired through inhalation of the infective mycelial phase and the organism then parasitises the mononuclear-phagocytic system and becomes disseminated throughout the body of the susceptible host (Rosas-Rosas et al 2004).

Because outbreaks of histoplasmosis in humans were repeatedly observed to be associated with cleaning chicken coops and handling pigeon excreta, poultry were suspected to be involved in the epidemiology of this disease and both pigeons and chickens were found to be able to be infected with *H. capsulatum* when given by intravenous injection (Schwarz et al 1957).

However, no reports of natural infection of poultry 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).

It should be noted that *H. capsulatum* var. *farciminosum* is distinct from *H. capsulatum* var. *capsulatum*. Inhalation of *H. capsulatum* var. *capsulatum* spores in dust generally associated with bird or bat droppings is associated with histoplasmosis in humans. Whilst exudates from equine *H. capsulatum* var. *farciminosum* infections have been used to experimentally infect rabbits, mice, and guinea pigs, no reports have been located which confirmed human infection with this organism (Coetzer 2004; Picard and Vismar 2004).

#### 37.1.5. Hazard identification conclusion

*H. capsulatum* var *capsulatum* is not considered to be a disease of birds. Therefore it is not a potential hazard.

### References

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## 38. Nematodes and Acanthocephalans

### 38.1. HAZARD IDENTIFICATION

#### 38.1.1. Aetiological agent

The nematodes and acanthocephalans that have been identified in turkeys are summarised below in Table 4.

#### 38.1.2. OIE list

Not listed.

#### 38.1.3. New Zealand status

McKenna (1998) described the nematode species recognised in New Zealand birds. *Ascaridia* sp. and *Heterakis gallinarum* were recorded in domestic turkeys and *Ascaridia galli*, *Capillaria annulata*, *Capillaria caudinflata*, *Capillaria obsignata*, *Heterakis gallinarum*, *Heterakis vesicularis*, and *Syngamus trachea* were recorded in domestic fowl. No acanthocephalan parasites were described in domestic poultry.

#### 38.1.4. 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 turkeys are summarised below in Table 4.

**Table 4. Nematodes and acanthocephalans of turkeys (Yazwinski and Tucker 2008)**

Parasite	Location	Intermediate host	Pathogenicity
<b>Upper digestive tract</b>			
<i>Capillaria annulata</i>	Mucosa of oesophagus and crop	Earthworms ( <i>Eisenia foetida</i> and <i>Allolobophora caliginosus</i> )	Inflammation of crop and oesophageal walls with masses of worms found in sloughing tissue
<i>Capillaria contorta</i>	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
<i>Capillaria combologiodes</i>	Crop	Unknown	Not described
<i>Gongylonema ingluvicola</i>	Mucosa of crop and sometimes in oesophagus and proventriculus	Beetle ( <i>Copris minutus</i> ) and cockroaches	Limited local lesions in crop mucosa
<i>Cyanea colini</i>	Proventriculus wall at junction with gizzard	Cockroach ( <i>Blatella germanica</i> )	Little or no pathology associated with this parasite
<i>Dispharynx nasuta</i>	Proventriculus wall, sometimes in oesophagus and rarely in the small intestine	Pillbug ( <i>Armadillidium vulgare</i> ) and sowbug ( <i>Porcellio scaber</i> )	Proventricular ulceration and thickening with parasites concealed beneath the proliferating tissue.
<i>Tetrameres americana</i>	Proventriculus wall	Grasshoppers ( <i>Melanoplus femurrubrum</i> and <i>M. differentialis</i> ) and cockroach ( <i>Blatella germanica</i> )	Thickening of proventricular wall
<i>Tetrameres fissispina</i>	Proventriculus	Amphipods, grasshoppers, earthworms, and cockroaches all described	Considerable tissue reaction with degeneration, oedema and leukocyte infiltration
<i>Tetrameres confusa</i>	Proventriculus	Unknown	Not described
<i>Cheilospirura hamulosa</i>	Cardiac and/or pyloric regions of the gizzard	Grasshoppers, beetles, weevils, and sandhoppers	Heavy infestations may be associated with damage to the gizzard wall
<i>Oncicola canis</i>	Under epithelial lining of the oesophagus	Not described	Adult worm normally in dog and coyote. Finding in turkeys likely to be accidental occurrence in unsuitable host
<b>Small intestine</b>			
<i>Ascaridia dissimilis</i>	Lumen and wall of small intestine	None	Intestinal inflammation results in lowered feed efficiency and poor performance. Aberrant migration of larvae may also cause hepatic foci and granulomas
<i>Ascaridia galli</i>	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
<i>Capillaria obsignata</i>	Small intestine	None	Catarrhal exudate and thickening of intestinal wall associated with heavy infestations
<i>Capillaria caudinflata</i>	Small intestine mucosa	Earthworms ( <i>Allolobophora caliginosa</i> or <i>Eisenia foetida</i> )	None described
<i>Capillaria bursata</i>	Small intestine mucosa	None	None described
<i>Capillaria anatis</i>	Small intestine and caecum	Unknown	None described

**Table 4 (continued)**

Parasite	Location	Intermediate host	Pathogenicity
<b>Caecum</b>			
<i>Heterakis gallinarum</i>	Caecum	None	Inflammation and thickening of the caecal walls. Carrier of <i>Histomonas meleagridis</i>
<i>Heterakis meleagris</i>	Caecum	Unknown	None described
<i>Subulura brumpti</i>	Caecum	Beetles or cockroaches	None described
<i>Subulura suctorica</i>	Caecum and small intestine	Beetles	Very little
<i>Strongyloides avium</i>	Caecum and small intestine	None	Marked thickening of caecal wall
<i>Trichostrongylus tenuis</i>	Caecum and small intestine	None	Caecal distension and congestion with thickening of caecal wall. Anaemia associated with heavy infestation
<b>Respiratory tract</b>			
<i>Cyathostoma bronchialis</i>	Larynx, trachea, bronchi and abdominal air sacs	Earthworms may act as paratenic hosts but not necessary	Hyperplasia of bronchial epithelium, pneumonitis and respiratory distress
<i>Syngamus trachea</i>	Trachea, bronchi and bronchioles	Earthworms ( <i>Eisenia foetida</i> and <i>Allolobophora caliginosa</i> ), slugs and snails may act as paratenic hosts but not necessary	Obstruction of tracheal lumen may lead to suffocation
<b>Other tissues</b>			
<i>Oxyspirura mansoni</i>	Beneath nictitating membrane and in conjunctival sacs and nasolacrimal ducts	Cockroach ( <i>Pycnoscelus (Leucophaea) surinamensis</i> )	Ophthalmia, possibly progressing to destruction of eyeball.
<i>Aproctella stoddardi</i>	Body cavity	Unknown, possibly a biting arthropod	Generally not pathogenic although has been associated with a granulomatous pericarditis
<i>Singhfilaria hayesi</i>	Subcutaneous	Unknown	Little described

### 38.1.5. Hazard identification conclusion

A wide range of nematodes and acanthocephalans of turkeys 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 assessed not to be potential hazards.

### References

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## 39. Cestodes and trematodes

### 39.1. HAZARD IDENTIFICATION

#### 39.1.1. Aetiological agent

Tapeworms (Class: *Cestoda*) and flukes (Class: *Trematoda*). The cestode and trematode parasites of turkeys identified by Taylor et al (2007) are shown below in Table 5.

**Table 5. Turkey cestode and trematode parasites (Taylor et al 2007)**

Parasite	Location	Intermediate host	Pathogenicity
<b>Cestodes</b>			
<i>Raillietina cesticillus</i>	Small intestine	Various genera of beetles	Heavy infestations associated with catarrhal enteritis. Birds may show a reduced growth rate, emaciation, and weakness
<i>Raillietina echinobothrida</i>	Small intestine	Ants of the genera <i>Pheidole</i> and <i>Tetramorium</i>	Hyperplastic enteritis at the site of attachment may result in caseous nodules in the intestinal wall
<i>Davainea proglottina</i>	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
<i>Choanotaenia infundibulum</i>	Small intestine	Housefly, beetles, and grasshoppers	None described
<i>Metroliaesthes lucida</i>	Small intestine	Grasshoppers	None described
<i>Hymenolepis carioca</i>	Small intestine	Dung and flour beetles, sometimes <i>Stomoxys</i> spp.	Considered to be of low pathogenicity
<i>Hymenolepis cantianiana</i>	Small intestine	Beetles	None described
<b>Trematodes</b>			
<i>Hypoderaeum conoideum</i>	Small intestine	Snails, frogs, freshwater clams, and mussels	Large numbers may be associated with enteritis and diarrhoea
<i>Brachylaemus commutatus</i>	Caeca	Land snails	Large numbers may be associated with enteritis and diarrhoea
<i>Postharmostomum commutatum</i>	Caeca	Snails	Large numbers may be associated with enteritis and diarrhoea
<i>Prosthogonimus pellucidus</i>	Large intestine and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal
<i>Prosthogonimus macrorchis</i>	Cloacal bursa and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal
<i>Prosthogonimus ovatus</i>	Rectum and oviduct	Aquatic snails and nymph stage of dragonflies	Light infestations affect egg production, heavy infestations can be fatal
<i>Plagiorchis arcuatus</i>	Rectum and oviduct	Snails, crustacea, molluscs, and insects	Light infestations affect egg production, heavy infestations can be fatal
<i>Notocotylus attenuatus</i>	Oviduct	Snails	Large numbers may be associated with enteritis and diarrhoea
<i>Collyriclum faba</i>	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

#### 39.1.2. OIE list

Not listed.

### 39.1.3. New Zealand status

McKenna (1998) identified the cestodes *Davainea proglottina*, *Raillietina cesticillus*, and *Choanotaenia infundibulum*, and the trematode *Notocotylus attenuatus* in New Zealand.

### 39.1.4. Epidemiology

Cestode infestation is associated with free-range rearing or backyard flocks but 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. 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 which 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 turkeys 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).

### 39.1.5. Hazard identification conclusion

The intestinal tract is removed from all commodities considered here and commercially reared turkeys would be unlikely to be raised in wet marshy areas required for the persistence of the trematode *Collyriclum faba*. Cestodes and trematodes are assessed not to be potential hazards.

## References

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**Taylor MA, Coop RL and Wall RL (2007)** Parasites of poultry and gamebirds. In *Veterinary Parasitology* 3<sup>rd</sup> Edition, Blackwell Publishing, 459-557.

## 40. *Cochlosoma anatis* infection

### 40.1. HAZARD IDENTIFICATION

#### 40.1.1. Aetiological agent

*Cochlosoma anatis* is a flagellate protozoan initially identified in the European domestic duck (Bermudez 2008).

#### 40.1.2. OIE list

Not listed.

#### 40.1.3. New Zealand status

No record of *C. anatis* infection in New Zealand could be found.

#### 40.1.4. 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).

*C. anatis* can be found throughout the intestinal tract of young poults and localises to 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).

#### 40.1.5. 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 assessed not to be a potential hazard.

## References

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**Campbell JG (1945)** An infectious enteritis in young turkeys associated with *Cochlosoma* sp. *The Veterinary Journal* 101, 255-259.

**Cooper GL, Shivaprasad HL, Bickford AA, Nordhausen R, Munn RJ and Jeffrey JS (1995)** Enteritis in turkeys associated with an unusual flagellated protozoan (*Cochlosoma anatis*). *Avian Diseases* 39, 183-190.

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**McNeil E and Hinshaw WR (1942)** *Cochlosoma rostratum* from the turkey. *Journal of Parasitology* 28, 349-350.

## 41. Hexamita

### 41.1. HAZARD IDENTIFICATION

#### 41.1.1. Aetiological agent

Hexamitiasis is caused by the protozoan *Spironucleus meleagridis*, commonly known by the generic name *Hexamita* (McDougald 2008).

#### 41.1.2. OIE list

Not listed.

#### 41.1.3. New Zealand status

No record of hexamitiasis in New Zealand could be found. Given the importation of game birds and turkeys over many years, it has been suggested that they were likely to have arrived carrying this organism although hexamitiasis is reported to be unseen in New Zealand game birds (Christensen 2010).

#### 41.1.4. Epidemiology

Poults infected with hexamitiasis have watery diarrhoea which may progress to listlessness, convulsions, and coma. At necropsy, watery distension of the small intestine is seen with large number of *Hexamita* present in the intestinal mucus and in intestinal crypts under microscopic examination (Wilson and Slavin 1955; McDougald 2008). Histological examination of a flock of naturally infected chukar partridges also demonstrated that *Hexamita* can be found intracellularly within the cells of the intestinal mucosal epithelium and lamina propria (Cooper et al 2004).

Heavy infections of *Hexamita* in the small intestine of game birds result in a marked reduction in absorption across the intestinal wall with subsequent diarrhoea, depression, and weight loss (Lloyd et al 2005). Further work by this group demonstrated that hexamitiasis was responsible for a significant lowering of plasma total protein, albumin, osmolality and electrolyte concentrations (Lloyd and Gibson 2006).

Hexamitiasis is now rarely seen in commercial turkeys but remains common in game birds (Wood and Smith 2005; McDougald 2008)

#### 41.1.5. Hazard identification conclusion

Hexamitiasis is unlikely to be present in commercial turkeys. Furthermore, infectivity is confined to the intestinal tract which is removed from the commodities under consideration here. *Hexamita* are assessed not to be potential hazards.

### References

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