

***Import risk analysis:***  
**Passerine hatching eggs**  
**from the European Union**

**1 February 2006**



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European Union***

**Biosecurity New Zealand  
Ministry of Agriculture and Forestry  
Wellington  
New Zealand**



**1 February 2006**

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*Import risk analysis:* Passerine hatching eggs from the European Union

1 February 2006

Approved for general release

Debbie Pearson  
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# 1 EXECUTIVE SUMMARY

This risk analysis considers the biosecurity risks associated with the importation of hatching eggs of birds in the order Passeriformes from the European Union.

From a preliminary hazard list of organisms, those that were considered to be potential hazards in the commodity were subjected to individual risk assessments.

As a result of the individual risk assessments, it was concluded that the risk in the commodity was non-negligible for the following organisms:

- avian influenza viruses
- avian paramyxoviruses types 1, 2 and 3

These organisms were classified as hazards in the commodity, and sanitary measures were recommended to manage their risks to an acceptable level. These measures include:

- layer flocks of origin will be tested for the presence of these organisms prior to collection of the hatching eggs, and eggs will be collected only from test-negative flocks
- the imported eggs will be hatched in a post-arrival quarantine facility in New Zealand
- hatchlings from the imported eggs will be tested for the presence of these organisms
- a biosecurity clearance will be issued for the birds hatched from the imported eggs only if all laboratory tests for these organisms are negative

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## 2 INTRODUCTION

This risk analysis examines the biosecurity risks posed by the importation of hatching eggs of birds in the taxonomic order Passeriformes from the European Union into New Zealand.

### 2.1 Commodity definition

The commodity is hatching eggs of any species of the order Passeriformes from the EU. The eggs must be clean (free of faeces) when collected, unwashed and have intact shells (uncracked). Following collection the eggs must be disinfected in accordance with Appendix 3.4.1 of the OIE Terrestrial Animal Health Code.

### 2.2 Background

Zoological gardens and aviary owners wish to import passerine eggs for the purposes of hatching to produce birds for inclusion in their collections.

Over 50% of the world's 9,600 avian species fall within the Order Passeriformes. These are the song birds or perching birds and include such well known birds as house sparrows, starlings, thrushes, magpies, crows, swallows and the many species of finches. There are 53 families that fall directly within the order Passeriformes, while another 29 families are contained in the suborder Oscines and 5 families are contained in the suborder Tyranni (1).

There have been very few evaluations of the diseases or potentially pathogenic organisms carried by bird eggs, other than those of poultry, in New Zealand. Most relevant avian disease information comes from poultry species and/or sporadic case reports and/or from local and regional surveys.

Many of the organisms considered in this risk analysis commonly infect birds without causing disease. On occasions, however, they may be associated with incidents of disease. Examples of this include avian influenza viruses, paramyxoviruses, adenoviruses, alphaviruses, bunyaviruses and *Salmonella spp.* Surveillance for many of these organisms in New Zealand is relatively insensitive so that their lack of recognition in this country does not provide a basis for confidence that they are not present. In New Zealand surveillance information on diseases in passerine species comes mainly from passive surveillance (i.e. reports of incidents of disease sufficiently pronounced to attract attention and to encourage investment in professional examinations and laboratory investigations) and it is likely that organisms causing sub-clinical disease or only occasional clinical disease may remain undiagnosed.

More information is available on organisms present in passerines in Great Britain, continental Europe and the United States. This arises, in part, from the potential for these birds to carry zoonotic organisms and it also results from the interest of the public and

scientists in ensuring the well being of their native and introduced avifauna. The recognition that passerine birds are contributing to the global phenomenon of “emerging diseases” originating in wildlife and causing disease in humans and animals has contributed to recent increases in interest in the organisms (particularly arthropod-borne viruses) carried by these birds. Prominent amongst these is West Nile Virus which had been recognised in Africa, with incursions into southern Europe, for many years before it emerged in North America in 1999 causing extensive mortalities in passerine birds and numerous deaths in humans.

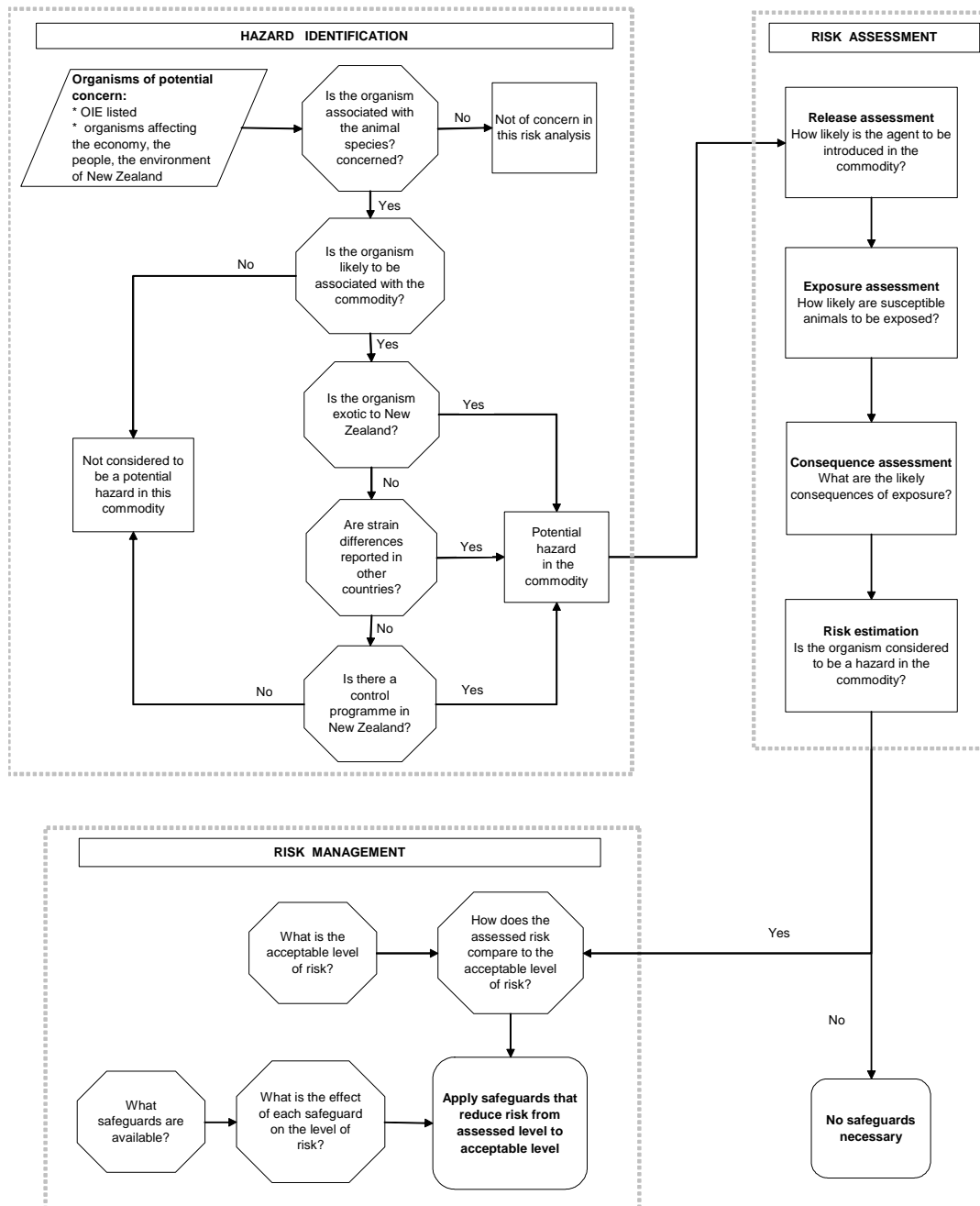
Large numbers of birds, previously exotic to New Zealand, have been imported with little or no evaluation of their carriage of organisms that might, in today’s terms, be classified as hazards. Importations of passerine birds identified by Heather and Robertson (2) are house sparrows (>100, source not stated, 1866 – 1871), chaffinches (several hundred, from South Africa, 1862 – 1880), redpolls (500, source not stated, 1862 – 1875), goldfinches (500, source not stated, 1862), greenfinches (<100, source not stated, 1862 – 1880), yellow hammers, starlings (1,000, 1862 – 1883), mynahs (several hundred, source not stated, 1870 – 1880), Australian magpies (>1,000, Australia, 1864 – 1874), black birds (1,000, source not stated, 1862 – 1875) and song thrushes (several hundred, source not stated, 1862 – 1878). All of these birds were imported prior to the recognition of most of the diseases covered in this risk analysis and prior to the recognition of the aetiology of any of them. During the 20<sup>th</sup> century, importations of large numbers of birds, including passerine species, continued from Europe and the United States into the 1960s and from Australia until 1997. An unknown number of the birds imported from Australia had originated in Europe and entered Australia under entry conditions directed at protecting Australia from the major epidemic diseases of poultry, particularly Newcastle disease and avian influenza.(3) It is likely that a high proportion of potential hazards that could reasonably be expected to have been imported with passerine species from Europe and Australia entered New Zealand with the importations that have taken place over the past 143 years.

## 2.3 Methodology

The methodology used in this risk analysis follows the guidelines in Section 1.3 of the *OIE Terrestrial Animal Health Code* (4). In New Zealand, the OIE risk analysis framework is applied as described in *Import Risk Analysis Animals and Animal Products* (5), the key elements of which are shown in Figure 1.

The hazard identification process begins with the collation of a list of organisms potentially associated with the commodity. Table 1 shows these organisms, together with some of the key information considered for each organism in determining whether or not it must be classified as a potential hazard in the commodity. This list was compiled from those contagious diseases of passerine birds identified from the several standard textbooks (6, 7) and from searches of the international scientific literature. Additional diseases were included on the basis of initial uncertainty as to whether they might infect passerines species.

**Figure 1. The risk analysis process.**



**Table 1. Organisms considered in this risk analysis**

<b>Organism / Disease</b>	<b>Present in New Zealand?</b>	<b>OIE listed? <sup>1</sup></b>	<b>Under official control or unwanted ? <sup>2</sup></b>	<b>More virulent strains overseas ? <sup>3</sup></b>
<b>Orthomyxoviridae</b>				
Avian influenza	Yes	Yes (H5 or H7 strains or any highly pathogenic strains)	Unwanted (H5 and H7 strains)	Yes
<b>Paramyxoviridae</b>				
Avian paramyxovirus 1 (APMV-1 to 9)	Yes (Some serogroups and strains)	Yes (Newcastle disease)	Notifiable (Exotic strains - Newcastle disease)	Yes
Pneumoviruses	No?	No	None	N.A.
<b>Herpesviridae</b>				
Duck virus enteritis	No	Yes	Notifiable	N.A.
Laryngotracheitis	Yes	Yes	None	Yes
Marek's disease	Yes	Yes	Other exotic organism (Exotic strains)	Yes
Other avian Herpes viruses	Yes / No	No	Pacheco's disease (Other exotic organism)	Yes
<b>Coronaviridae</b>				
Infectious bronchitis	Yes	Yes	Unwanted (exotic strains)	Yes
<b>Adenoviridae</b>				
Group I avian adenoviruses	Yes	No	None	Yes
Group II avian adenoviruses	No	No	None	N.A.
Group III avian adenoviruses (Egg Drop Syndrome)	Yes	No	None	No
<b>Poxviridae</b>				
Avipoxvirus	Yes	Yes (Fowl pox)	None	Yes
<b>Circoviridae</b>				
Gyrovirus (Chicken Infectious Anaemia)	Yes	No	None	No
Avian Circovirus	Yes	No	None	Yes
<b>Birnaviridae</b>				
Infectious Bursal Disease	No	Yes	Unwanted (Exotic strains)	N.A.



<b>Papovaviridae</b>				
Polyomavirus	Yes	No	None	No
Papillomavirus	No	No	None	N.A.
<b>Parvoviridae</b>				
Parvoviruses	No	No	Unwanted	N.A.
<b>Togaviridae</b>				
Equine encephalitides	No	No	Notifiable	N.A.
Other Alphaviruses	Yes	No	None	Yes
<b>Flaviviridae</b>				
West Nile Virus	No	No	None	N.A.
Japanese encephalitis virus	No	No	Notifiable	N.A.
Louping ill	No	No	Unwanted	N.A.
Other Flaviviruses	No	No	None	N.A.
<b>Reoviridae</b>				
Rotavirus	Yes	No	None	Yes
Orbivirus	Yes	No	None	No
Other reoviruses	Yes	No	None	Yes
<b>Bunyaviridae</b>				
Nairoviruses	No	No	None	N.A.
Other Bunyaviruses	No	No	None	N.A.
<b>Bornaviridae</b>				
Bornavirus	No	No	Unwanted	N.A.
<b>Picornaviridae</b>				
Avian encephalomyelitis	Yes	No	None	No
Duck hepatitis (DHV 1 & 3)	No	No	Unwanted	N.A.
<b>Astroviridae</b>				
Astroviruses	Uncertain	No	None	N.A.
<b>Hepadaviridae</b>				
Duck virus hepatitis	No	Yes	Unwanted	N.A.
<b>Retroviridae</b>				
Leucosis/sarcoma complex viruses	Yes	No	None	No
Lymphoproliferative disease virus	No	No	Unwanted	N.A.
Reticuloendotheliosis	Yes	No	None	No
Other retroviruses	No	No	None	No
<b>Chlamydophila</b>				
Chlamydia sp	Yes	Yes	None	Yes
<b>Bacteria associated with enteric and generalised infections in birds</b>				
<i>Salmonella</i> spp	Yes / No	Yes (Fowl typhoid, Pullorum disease)	Unwanted (Some serovars and variants within serovars)	Yes
<i>Escherichia coli</i>	Yes	No	None	No

<i>Campylobacter</i> spp.	Yes	No	None	No
Other Enterobacteriaceae	Yes / No	No	Not species infecting birds	Not species infecting birds
<b>Bacteria commonly associated with respiratory disease in birds</b>				
<i>Pasteurella multocida</i>	Yes	Yes (Fowl cholera)	None	Yes
<i>Riemerella anatipestifer</i>	Yes	No	None	No
<i>Pasteurella gallinarum</i>	No	No	None	N.A.
<i>Ornithobacterium rhinotracheale</i>	No	No	Other exotic organism	N.A.
<i>Bordetella avium</i>	No	No	Other exotic organism	N.A.
<i>Haemophilus paragallinarum</i>	No	No	Other exotic organism	N.A.
<i>Mycoplasma gallisepticum</i>	Yes	Yes	None	No
<i>Mycoplasma synoviae</i>	Yes	No	None	No
<i>Mycoplasma iowae</i>	No	No	Other exotic organism	N.A.
<b>Intracellular bacteria</b>				
<i>Mycobacterium tuberculosis</i>	Yes	No	Unwanted	No
<i>Mycobacterium avium</i>	Yes	Yes	None	No
Other Mycobacteria	Yes (Some)	No	Other exotic organism (exotic strains)	Yes
<b>Other bacteria</b>				
<i>Francisella tularensis</i>	No	No	Other exotic organism	N.A.
Megabacteria	Yes	No	None	No
Gram positive contaminants (e.g. Staphylococci / Streptococci)	Yes	No	None	No
<b>Spirochetes</b>				
<i>Borrelia anserina</i> (Avian spirochaetosis)	No	No	Other exotic organism	N.A.
<i>Borrelia burgdorferi</i> (Lyme Disease)	No	No	Other exotic organism	N.A.
<i>Brachyspira</i> spp	Yes	No	None	No
<b>Rickettsial agents</b>				
<i>Coxiella burnetii</i>	No	No	Notifiable	N.A.
<i>Cowdria ruminantium</i>	No	No	Unwanted	N.A.
<i>Aegyptianella pullorum</i>	No	No	None	N.A.
Other Rickettsia	Yes / No	No	Some general in register	Yes

<b>Fungi and yeasts</b>	Yes / No	No	Yes ( <i>Histoplasma farciminosum</i> )	Yes
<b>Internal parasites</b> (Nematodes, cestodes, protozoa)	Yes / No	No	None	Yes
<b>External parasites</b> (ticks, mites, lice)	Yes / No	No	Unwanted (Some genera)	Yes

- 1 Based on information on the OIE website at [www.oie.int/eng/maladies/en\\_classification.htm](http://www.oie.int/eng/maladies/en_classification.htm)
  - 2 Based on the information from the register of unwanted organism at [http://www.biosecurity.govt.nz/pests-diseases/registers-lists/unwanted organisms/](http://www.biosecurity.govt.nz/pests-diseases/registers-lists/unwanted_organisms/)
  - 3 More virulent exotic strains are recognised where either strain typing of New Zealand isolates allows differentiation from more pathogenic types recognised in other countries or where descriptions of the disease in New Zealand allow it to be recognised as less virulent than disease episodes in other countries. Where host specific strains are recognised overseas but not in NZ, these are treated as “more virulent” in the compilation of this table.
- N.A. Not applicable because assessment of strain variations is not relevant to this process when the organism is not recognised as present in New Zealand.

In this analysis, for each organism listed, the epidemiology is discussed, including a consideration of the following questions:

- 1) whether eggs from passerine birds could act as a vehicle for the introduction of the organism,
- 2) if the organism requires a vector, whether competent vectors might be in New Zealand,
- 3) whether the organism is exotic to New Zealand but likely to be present in exporting countries and
- 4) if it is present in New Zealand,
  - a. 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
  - b. whether more virulent strains are known to exist in other countries.

For any organism, if the answers 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.

Under this framework, which is based on international agreements on trade in agricultural products, organisms that are present in New Zealand cannot be considered as 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 than risks resulting from the presence of the organism in this country already. In

such situations, measures to limit negative impacts on the health of humans or animals in contact with the imported commodity, or subsequent progeny, should be those appropriate to good practice irrespective of the importation.

In line with the OIE risk analysis methodology, for each potential hazard the following analysis is carried out:

#### Risk Assessment

- a) Release 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.

In assessing the likelihood of exposure to wild birds, caged or aviary birds or poultry in New Zealand, an assumption is made that there is potential for contact between caged or aviary birds and those outside that environment. Such contact might be direct through the walls of enclosures, indirect through transfer of fomites, movement of rodents, insects or other animals or through escape or release of the imported birds.

It is important to note that all of the above steps may be necessary in all risk assessments. The OIE methodology makes it clear that if the likelihood of release is negligible for a certain 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 release 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 release and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

For all organisms that are classified as a hazard, the risk management step is carried out, comprising the following three sub-steps:

- a) Risk evaluation - a determination is made as to whether sanitary measures are necessary.
- b) Option evaluation - identify the options available for managing the risk, and consider risk reduction effects.

- c) Recommended measures - the recommendation of the appropriate option or combination of options that achieve a negligible likelihood of entry, spread or establishment, while minimising negative trade effects.

Further details, including the full hazard identification, and where appropriate the risk assessment and the recommended risk management measures, can be found in the chapters on the individual agents.

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## 3 ORGANISM RISK ANALYSES

### 3.1 Orthomyxoviridae

#### 3.1.1 Avian influenza

##### 3.1.1.1 Hazard identification

###### **Aetiological agent**

Avian Influenza (AI) viruses are Influenza A viruses within the family Orthomyxoviridae. These viruses are characterised by antigenic surface glycoprotein haemagglutinin (types H1 – 15) and neuraminidase (N1 – 9) (1). H and N antigens may be present in any combination (Hx, Ny). Strains of AI are commonly separated into highly pathogenic strains (HPAI) and low pathogenic strains (LPAI) on the basis of their pathogenicity in poultry. All HPAI virus isolates have been subtypes H5 or H7 but not all H5 or H7 isolates have been highly pathogenic. For statutory purposes, the main basis for differentiation on HPAI and LPAI strains has been pathogenicity in susceptible chickens (1, 2).

###### **OIE List**

Notifiable Avian Influenza (NAI) viruses are on the OIE List. NAI refers to any avian influenza virus of H5 or H7 subtypes or any AI virus with pathogenicity above limits set in the Terrestrial Animal Health Code 2005 (3).

###### **New Zealand Status**

Avian influenza H5 and H7 are listed as notifiable organisms in the unwanted organisms register.

AI viruses have been isolated from healthy wild mallard ducks in New Zealand (4, 5, 6, 7). Subtypes identified have included H4N6, H1N3 and H5N2 (4, 7). The H5N2 isolates were shown to be non-pathogenic (7).

A survey in the 1990s found no evidence of AI virus infection in 54 pigeons trapped at three locations in NZ nor in samples from 55 native birds (8). This negative finding received support from a 2003 serological survey of 560 pigeons (domestic and feral) sampled from Auckland, Wellington, Christchurch and Dunedin (9).

###### **Epidemiology**

The epidemiology of disease attributable to HPAI viruses has been reviewed and summarized by Alexander *et al.* (2). In the years from 1959 to the time of writing of Alexander's report (2003), 19 epidemics of disease in poultry had been attributed to

HPAI. All were caused by H5 or H7 viral sub-types (2) and all had the characteristics of high morbidity and high mortality affecting farmed flocks of either turkeys or chickens. Flocks reared outdoors, and, therefore, more vulnerable to exposure to wild birds, are affected more frequently than flocks maintained indoors. AI virus is introduced into an area by infected wild birds, mainly waterfowl, with gulls and sea birds playing a lesser role. The main means of spread is through large quantities of virus passed in faeces or respiratory secretions. Spread may be directly into areas occupied by poultry flocks, through contamination of water or through carriage on fomites. Because of the relatively high prevalence of AI viruses in migratory waterfowl, commercial flocks located outdoors and within migratory pathways appear to be at highest risk. Secondary spread is through transfer of infection from faeces, most commonly with people moving between farms (2). More recently, spread of AI (HPAI and LPAI) within live-bird markets has been found to be an important means of dissemination between poultry farms in both Asia (10, 11, 12) (HPAI) and the north eastern United States (13, 14) (LPAI). Market hygiene, (including control of interspecies contact) has been found to be a critical factor in controlling spread within these environments.

It appears that virulent H5 and H7 strains derive, by mutation, from low pathogenic H5 and H7 strains. These mutations arise following transfer of infection from the wild host to poultry and the ability of these mutated viruses to infect multiple tissues results in their high pathogenicity. Infectivity of HPAI in poultry appears to be lower than the infectivity of the strains from which they are derived (2). The propensity for AI strains to mutate is further illustrated by the genetic diversity of isolates within local epidemics (12).

Reports of identification of avian influenza virus from birds in the order Passeriformes include birds in quarantine in Great Britain arriving from India, Ghana, Taiwan and Holland (15); isolations from birds in Canada (16, 17); isolations from 42 of 134 passerine birds in Canada from birds sampled during spring 1980 and summer 1981 (18); isolations from starlings and serological evidence of infection of sparrows in the vicinity of an epidemic of AI in Australia (19); isolations from one dead and one sick passerine birds in Malaysia (20) and isolations from two house sparrows from the vicinity of an epidemic of HPAI in Italy (21).

The pathogenicity of AI virus strains varies depending upon the species infected. This has been illustrated by differences in responses of different species to experimental infections with an H5N1 strain of AI (22) and differences in clinical and pathological presentation of natural infections with LPAI and HPAI H7N1 in different species (23).

Horimoto and Kawaoka (24) reviewed cross-species infection with AI, particularly cross infections between ducks, pigs and humans, and the mechanisms (adaptation and genetic re-assortment) for the development of strains with high levels of virulence in humans. These processes are aided by the intensive mix of humans, pigs and ducks found in Asia.

## **Conclusion**

1. Any H5 or H7 strain of AI virus is considered to be potentially able to mutate to HPAI and is, therefore, a potential hazard in this commodity.
2. LPAI viruses may cause disease with varying morbidity and mortality. The determinants of pathogenicity of LPAI strains for different species of birds are not known. Any strain of AI virus is therefore considered to be a potential hazard in this commodity.

### **3.1.1.2 Risk assessment**

#### **Release assessment**

A number of strains of AI, including H5 and H7 strains, have been isolated from both live and dead birds imported to the United Kingdom (25, 26). Infections identified in farmed chickens and ducks have, commonly, coincided with periods of migration of wild birds (27). Waterfowl and sea birds in northern Europe have been shown to harbour LPAI viruses including types H5 and H7. The majority of the samples were collected from the Netherlands (28).

There is extensive movement of birds between Great Britain and continental Europe and migration of birds from the Arctic to areas of the UK, continental Europe and countries further south. These movements include species of waterfowl, sea birds and passerines (29, 30, 31, 32). On this basis the continuing presence, or repeated introductions of LPAI H5, H7 and other strains of AI into Great Britain and continental Europe is considered likely. Although infection rates in healthy passerine birds may be relatively low, and reports indicating that passerines play an important part in the epidemiology of AI have not been located, the likelihood of infection with AI can not be excluded.

HPAI strains are believed to develop in farmed poultry and most records of isolation of HPAI strains from other birds appear to have been as the result of overflow infections from affected poultry flocks.

AI virus has been isolated from both the internal contents (yolk and yolk plus albumen) of eggs and from egg shells from both broiler breeder flocks both in the presence of clinical disease and in infected flocks with no clinical signs (33). Although reports of transmission of infection to chicks via infected eggs have not been identified, movement of egg trays and associated fomites was a significant risk factor in the spread of AI infection during an epidemic in the Netherlands in 2003 (34). No reports of studies of infection of passerine eggs have been located. In the absence of evidence to the contrary, it is accepted that there is a non-negligible likelihood that passerine eggs can be infected with AI virus.

Therefore the release assessment for LPAI virus (including H5 and H7 strains) in passerine eggs is non-negligible. The release assessment for HPAI virus is negligible



except at times when HPAI infection is present in the area from which birds are to be imported. In that latter situation the release assessment is non-negligible.

### **Exposure assessment**

Separation of imported birds from wild and other captive birds in NZ will be minimal. AI virus is contagious being spread through faeces, on fomites and with people. Therefore the exposure assessment for AI viruses is non-negligible.

### **Consequence assessment**

There is a high likelihood that consequences of the importation of HPAI could include epidemic disease in poultry in New Zealand with high mortalities, disruption of the poultry industries and of export trade in poultry products. Both low susceptibility of birds in the wild and the imposition of control measures in the event of an incident of HPAI infection would limit the consequences of HPAI introduction on birds in the wild. However, as the current Asian H5N1 epidemic has demonstrated, spillover from poultry to wild birds can occur in some circumstances it is possible that the consequences may be severe.

Most isolations of LPAI virus have been from healthy birds. In 1961 an outbreak of serious disease South African terns was attributed to an influenza virus that would be classified as LPAI using current criteria (35) and deaths of cage birds due to influenza virus infections during transport (15, 20, 25, 26) have been reported. The factors contributing to the differences in development of disease in birds infected with LPAI strains are not known and the potential for LPAI strains to cause disease in birds (native or otherwise) in NZ can not be excluded.

Establishment of additional strains of H5 and/or H7 AI virus in NZ may increase the risk of the development of HPAI strains in NZ poultry. This could result in heavy direct losses to the poultry industry and would result in loss of access to most international markets currently supplied by the NZ poultry industry. Impacts of HPAI on native species are unknown but, based on experience with wild birds elsewhere, the likelihood of significant disease in native species is low but not negligible.

The intensive mixing of humans, pigs and ducks, seen in Asia and considered to contribute strongly to the development of new human strains of influenza virus pathogenic to humans, is very uncommon in New Zealand. The likelihood of adaptation or genetic re-assortment of AI viruses leading to the development of new strains capable of causing serious disease in humans is very small.

The epidemiology of the development of strains of AI virus pathogenic to humans is such that the likelihood of AI viruses in this commodity resulting in a hazard to human health is very low. However, as with all influenza viruses, if genetic reassortment were to occur and result in a human pandemic strain, the human health consequences would be severe.

Avian influenza viruses in this commodity are considered to be a potential hazard to New Zealand native birds, wild birds and poultry and the economy.

The consequence assessment for AI virus is non-negligible.

### **Risk estimation**

Since the release assessment, exposure assessment and consequence assessments for avian influenza viruses in passerine hatching eggs are all non-negligible, the release estimation is non-negligible and avian influenza viruses are classified as a hazard in the commodity.

#### **3.1.1.3 Risk management**

##### **Risk evaluation**

Since avian influenza viruses are considered to be hazards in the commodity, sanitary measures will need to be employed to effectively manage the risks to reduce them to negligible.

##### **Risk management objective**

To ensure that importation of the commodity does not result in release of AI virus to bird populations in New Zealand.

##### **Risk management options**

1. High to moderate levels of confidence that birds from which eggs will be collected are not infected with AI can be achieved by
  - a. Ensuring that the birds are from flocks in area(s) recognised as free from notifiable avian influenza as defined in the OIE Terrestrial Animal Health Code 2005 (3). This will provide a high to moderate level of assurance that the birds are not carrying highly pathogenic notifiable avian influenza (HPNAI) and a moderate to low level of assurance that they are free of low pathogenicity notifiable AI (LPNAI).
  - b. Moving birds intended for production of eggs into an enclosure that isolates them from other birds and testing these birds for AI. See comments in 3 below on test procedures.
  - c. Maintaining birds intended for production of eggs in isolation from other birds from the time of testing through the period of egg production.
2. Additional confidence that hatchlings to be given biosecurity clearance in New Zealand are not infected with AI can be attained by hatching the eggs and maintaining the hatchlings in quarantine and
  - a. testing material from all embryos/chicks dead-in-shell and from any hatchlings dying.

- b. testing a sample of hatchlings prior to clearance.
- 3. Test procedures available
  - a. Cloacal or choanal swabs from live birds or hatchlings may be tested by
    - i. using PCR methods that detect Group A influenza viruses (36)
    - ii. Culturing the swabs for AI virus using methods described in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (36).
  - b. For blood samples – Commercial ELISA tests are available.
  - c. Keeping the birds and/or hatchlings in contact with specific pathogen free chickens during the quarantine period and then testing the chickens for infection with AI using methods identified above.

Note: Neither reports of the magnitude of serological responses to AI in passerines, nor reports of the validation of serological tests in passerine birds have been located. Testing using PCR or viral culture is recommended.

### **Recommended sanitary measures**

- 4. birds from which eggs will be collected should come from flocks
  - a. in area(s) recognised as free from notifiable avian influenza as defined in the OIE Terrestrial Animal Health Code 2005

**AND**

  - b. with negative test results for AI on a sample of birds.
- 5. prior to the period of egg collection, birds from which eggs will be collected should be isolated from other birds and tested for AI.
- 6. eggs should be hatched in quarantine and
  - a. samples from all embryos/chicks dead-in-shell and any hatchlings that die should be tested for AI

**AND**

  - b. a sample of hatchlings should be tested for AI prior to biosecurity clearance in New Zealand.
- 7. samples from both laying birds and hatchlings are cloacal or choanal swabs.
- 8. test procedures for laying birds, hatchlings and embryos/chick dead-in-shell or dead hatchlings are **EITHER**
  - a. PCR methods that detect Group A influenza viruses

**OR**

  - b. Culturing samples or swabs for AI virus using methods described in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

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## **3.2 Paramyxoviridae**

### **3.2.1 Avian paramyxoviruses**

#### **3.2.1.1 Hazard identification**

##### **Aetiological agent**

Nine “prototype” virus strains of paramyxovirus are recognised in birds. They are in the genus Rubulavirus, are differentiated on serological grounds and identified as Avian paramyxoviruses 1 to 9 (APMV-1 to 9) (1).

Pathogenic strains of APMV-1 cause Newcastle disease (ND) and strains have, in the past, been differentiated on the basis of their ability to cause chick embryo mortality (2) which provides a guide to the severity of disease caused by the virus strains. The OIE criteria for reporting an outbreak of ND provide for differentiation of isolates of APMV-1 on the basis of either intra-cerebral pathogenicity in day-old chicks or demonstration of specific amino acids at specific locations on F1 and F2 proteins in the virus (3).

APMVs 1 to 9 show varying degrees of host specificity (see “epidemiology” section below).

##### **OIE List**

Newcastle disease is included in the OIE list of notifiable diseases.

##### **New Zealand Status**

APMV - 1 (exotic strains) (Newcastle disease) is listed as notifiable in the unwanted organisms register.

APMVs - 2, 3 and 5 are listed as “other exotic organisms” in the unwanted organisms register.

Newcastle disease has never been diagnosed in New Zealand. A non-pathogenic strain of APMV-1 is present (4). Pharo (5) reviewed New Zealand’s status with respect to Newcastle disease. APMV-1 has been isolated from mallard ducks, chickens and one parrot. All New Zealand APMV-1 isolates have been demonstrated to be avirulent.

In addition to the APMV-1 isolations, APMV-4 was identified in samples from 17 ducks. Serological tests in ducks were positive for APMV-1, 2, 3, 4, 6, 7, 8 and 9 but, because of the cross reactivity that occurs between the prototype strains (1), only APMV-1, 4 and 6 could be concluded to be present. Testing did not include APMV-5 (6).

Stanislawek *et al.* (7) interpreted serological results from caged birds, wild birds and poultry as indicative of the presence of APMV-1 in all categories and suggestive (but not

confirmatory) of the presence of APMV-2 in wild birds. These interpretations applied to both caged and wild passerines. Because of cross reactivity between APMVs, the presence of other APMVs in caged or wild birds could not be excluded.

## **Epidemiology**

Newcastle disease virus (NDV) is distributed in poultry throughout the world with clinical disease being largely controlled in more developed areas through the widespread use of vaccines (1). Transmission between birds may be through either inhalation or ingestion. Geographic spread may be aided by movement of live birds, contact between animal groups, movement of people and/or fomites and spread in aerosols. Contamination of waterways, ponds and surface water have also been proposed as means of spread of NDV (8, 9). Infection in groups of animals may present with signs varying from high morbidity and high mortality to inapparent infections depending upon viral strain and host strain or species. There is anecdotal evidence that ND causes mild transient conjunctivitis and, occasionally, fever in humans. Reports of human to human transmission have not been identified (1).

Mutation of a NDV of low virulence was proposed as the most likely source of high virulence virus that caused a disease outbreak in Australia (10, 11).

APMV-1 infection has been reported from 241 species of birds from 27 orders with differences in clinical presentation even between species within the same genus (12). Further identifications have taken place since that time and Alexander (1) proposed that the majority, if not all, birds are susceptible to infection.

APMV-2 (also called Yucaipa virus) is widespread in poultry, particularly chickens and turkeys, around the world (1). In these species it, most commonly, causes mild respiratory disease although more severe disease has been reported in turkeys (13). In wild and caged birds APMV-2 has been recorded from Europe, Asia, Africa and America with most isolations being from passerine birds (1).

APMV-3 was first identified in turkeys in the United States and, subsequently in other countries. In turkeys it causes egg production problems. There have been no reports of natural infections of chickens. APMV-3 has been isolated relatively frequently from caged and quarantined birds, mainly psittacines but also passerines. Based on both structural polypeptide analyses (14) and serotyping using monoclonal antibodies (15), APMV-3 strains infecting caged birds differ from those infecting turkeys.

APMV-4 viruses have been isolated only from ducks and geese and have not been associated with disease (1, 6).

APMV-5 has been reported only from budgerigars in a single unique epizootic in pet budgerigars in Japan between 1974 and 1976 (16)



APMV-6 has been isolated from turkeys, in which it may cause mild disease and from ducks and geese from which disease association has not been reported (1).

APMV-7 has been reported from pigeons, doves, turkeys and ostriches. It has been associated with mild respiratory disease in turkeys (1) but searches for reports of pathogenicity in other species have not been successful.

APMV-8 and 9 have been reported from ducks and geese (APMV-8) (1) but reports suggesting pathogenicity have not been located.

### **Conclusion**

APMV-1 is considered to be a potential hazard in this commodity.

APMV-2, 3, 5, 7, 8 and 9 are considered to be potential hazards in this commodity.

APMV-4 and 6 are present in New Zealand and are not considered to be potential hazards in this commodity.

### **3.2.1.2 Risk assessment**

#### **Release assessment**

Passerine birds are common amongst the wild and caged birds identified as infected by APMV-1. Reports include

1. 25% mortality in Gouldian finches in Germany (17),
2. 50% mortality in canaries in Germany (17),
3. Isolation from a dyspnoeic small cubafinch from a flock that had sporadic mortalities in Austria (18),
4. Isolation from a tree sparrow in Germany (19),
5. Isolation of a lentogenic strain from a migrating starling with neurological disease in Israel (20),
6. Isolation from a White spotted Munia (21) and
7. Isolations from starlings and sparrows in Pakistan (22).

Reports of isolation of APMV-2 from passerine species include

1. From tracheal swabs of captured birds in Germany (23),
2. From faeces of wild birds of passerine species in Senegal destined for export to Europe (24),
3. From cloacal swabs of common wrens in Czechoslovakia (25),
4. From cloacal swabs from one caged finch and one caged wren in Costa Rica (26). Both birds were in good health.

Serological evidence of APMV-2 was found in Passerine species (sparrows) during surveys of wild and domestic birds in southern Spain (27).

Reports of disease associated with APMV-2 in passerine birds have not been located and the virus is, generally considered to be non-pathogenic in non-poultry species. Goodman *et al.* (28), however, found decreased activity in recently experimentally infected finches and suggested that the behavioural changes could result in increased susceptibility of wild birds to disease. Alexander (16) considered that passerine birds were the primary hosts for APMV-2 and that psittacines became infected when in close proximity.

APMV-3 has been isolated from caged, but not feral, birds. Isolations have been from imported psittacine and passerine birds (1) and passerine species may carry infection sub-clinically for months (29). Reports of infection have included those

1. From two exotic finches in breeder aviaries, both from the same importation, in Germany (30),
2. From red headed tits that died in a quarantine facility in Hong Kong after being captured in China (31),
3. From imported caged passerine birds in Israel. The birds had become ill following importation (32).
4. From an incident of high mortality in a breeding colony of ornamental finches in Sweden that had recently received new birds from a breeder in Denmark (33).

Alexander (16) suggested that psittacine birds were the primary hosts for APMV-3 (caged bird strains) and that passerines became infected when in close proximity.

No reports of APMV-4 or 6 to 9 in wild or caged passerine birds have been located.

There is only one report of APMV-5 and that was from a single episode of disease in caged budgerigars in Japan.

Alexander (1) considered that whether true vertical transmission of NDV occurred was controversial, at least in part because birds infected with pathogenic strains commonly cease laying and, also because infection of eggs commonly results in death of the embryo. Lethality of NDV in embryonated eggs, is however, used as measure of virulence of virus isolates (3), so the comments by Alexander are interpreted as referring to transmission of virulent NDV during outbreaks of disease. Chen and Wang (34), on the basis of epidemiological evidence and results from experimental infection of chicken embryos, concluded that egg borne transmission of NDV was possible. McFerran *et al.* (35) found that Yucaipa virus and Bangor virus strains (both members of APMV-2) isolated from finches grew in eggs and that some embryos survived. While there may be doubt whether true transovarial vertical transmission of APMV occurs, there are fewer doubts that APMV can penetrate eggs shells, either cracked or intact, after laying. While there is no information available on the role of the egg in transmission of APMV in passerines, it is concluded that the likelihood of such a means of spread is non-negligible.

The release assessment for APMV-1, 2 and 3 (cage bird strains) is non-negligible.

The release assessment for APMV- 3 (turkey strains), and 4 to 9 is negligible.

## **Exposure assessment**

Although little is known of the epidemiology of APMVs other than APMV-1, all can be expected to behave as contagious organisms. Should infected eggs be imported and lead to infected birds that are given biosecurity clearance, spread to other susceptible species to which they are exposed is likely.

Exposure assessments for APMV-1, 2 and 3 (cage bird strains) in passerine eggs are non-negligible..

## **Consequence assessment**

The potential consequences of introduction of new strains of APMV-1 to New Zealand vary greatly. The current lentogenic strain is reported to spread relatively slowly in poultry and introduction of a strain that spread rapidly could disrupt current sero-surveillance (Christensen, N.H. 2005. Review of Import Risk Analysis: Birds of the Order Passeriformes from the European Union, Draft 14 May 2005). Otherwise such an introduction would be of no consequence unless it subsequently mutated to a more pathogenic form. The introduction of a velogenic strain would have serious consequences for the poultry industry and it could result in substantial mortalities in wild and/or caged birds. Although there are anecdotal reports of APMV-1 causing disease in humans, these reports have not been confined to velogenic strains. Given the presence of a lentogenic strain of APMV-1 in New Zealand and the mild and transient nature of the disease reported anecdotally as being caused by APMV-1 in humans (1), any consequence to human health is considered negligible.

The introduction of APMV-2 could have negative consequences for the poultry industry, especially the turkey industry, with mild respiratory disease and some decreases in egg production (1). Clinical disease in passerine, or other wild or caged birds is unlikely, however, negative behavioural effects in stressed birds can not be excluded.

The introduction of APMV-3 strains with imported passerine eggs could result in disease in both passerines and psittacines. Although reports in the literature are from caged birds and birds in quarantine, the possibility of infection and disease in native birds, especially birds under stress, cannot be excluded.

The consequences of APMV-1 and 2 in the commodity would be restricted to the New Zealand poultry industry.

The consequences of APMV-3 (cage bird strains) in the commodity would be expected in both passerine and psittacine birds.

These organisms would not affect the environment or industries other than poultry. Any consequences to human health are considered minor, if not negligible.

Therefore, the consequence assessments for APMV-1 and 2 and 3 are non-negligible.

## **Risk estimation**

Since the release assessment, exposure assessment and consequence assessments for APMV- 1, 2 and 3 (cage bird strains) in passerine hatching eggs are all non-negligible, the release estimation for these viruses is non-negligible and they are classified as hazards in the commodity.

### **3.2.1.3 Risk management**

#### **Risk evaluation**

Since APMV- 1, 2 and 3 (cage bird strains) are considered to be hazards in the commodity, sanitary measures will need to be employed to effectively manage the risk to reduce them to negligible.

#### **Risk management objective**

To ensure that importation of the commodity does not result in release of APMV- 1, 2 or 3 (cage bird strains) to bird populations in New Zealand.

#### **Risk management options**

1. High levels of confidence that birds from which eggs will be collected are not infected with APMV-1, -2 or -3 (cage bird strain) can be achieved by
  - a. Ensuring that the birds are from flocks in area(s) recognised as free from notifiable Newcastle disease as defined in the OIE Terrestrial Animal Health Code 2005 (36). This will provide a high level of assurance that the birds are not carrying velogenic APMV-1.
  - b. Testing a sample of birds from each source flock for APMV. See comments below on test procedures.
  - c. Maintaining birds in pre-export quarantine prior to, and during, pre-export testing.
2. Additional confidence that hatchlings to be given biosecurity clearance in New Zealand are not infected with APMV-1, -2 or -3 (cage bird strain) can be attained by
  - a. Testing material from all eggs failing to hatch and from any hatchlings dying.
  - b. Testing a sample of hatchlings prior to clearance.
3. Test procedures available
  - a. Culture for APMV using methods described in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (3).

Alternatives for the identification of any isolates of APMV are

- i. Test any APMV isolate for serotype, particularly for APMVs -1, -2 and -3 (cage bird strains), paying particular attention to the antigens and antisera used to avoid erroneous identification. See reference 3.

**OR**

- ii. Assume that any APMV isolated is either APMV -1, -2 or -3 (cage bird strain). This assumption is justified on the basis of the epidemiology and releases assessments in this risk analysis.

- b. Serological tests – haemagglutination, haemagglutination inhibition tests and ELISAs are used in the diagnosis of Newcastle disease (3). Validation of tests has, mainly, focussed on APMV-1 in poultry and Alexander (1) comments on the need for care in reagent selection.

Note: Use of sentinel specific pathogen free chickens in contact with passerines may allow detection of APMV-1 and 2. This procedure may not allow the detection of APMV-3 as, although one-day-old chickens have been shown to be susceptible to experimental infection with one isolate (37) this was not a cage-bird strain and there are no reports of natural infection of chickens with APMV-3.

### **Recommended sanitary measures**

1. birds from which eggs will be collected should come from flocks
  - a. in area(s) recognised as free from Newcastle disease as defined in the OIE Terrestrial Animal Health Code 2005

**AND**

  - b. with negative test results for APMV on a sample of birds.
2. prior to the period of egg collection, birds from which eggs will be collected should be isolated from other birds and tested for APMV.
3. eggs should be hatched in quarantine and
  - a. samples from all embryos/chicks dead-in-shell and all hatchlings that die should be tested for APMV

**AND**

  - b. a sample of hatchlings should be tested for APMV prior to biosecurity clearance in New Zealand.
4. samples from both laying birds and hatchlings are cloacal or choanal swabs.
5. test procedure for laying birds, hatchlings, embryos/chicks dead-in shell and dead hatchlings is culture for APMV using methods described in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (3). It should be assumed that any APMV isolated is either APMV -1, -2 or -3 (cage bird strain).

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### **3.2.2 Pneumovirus**

#### **3.2.2.1 Hazard identification**

##### **Aetiological agent**

Family Paramyxoviridae, subfamily Pneumovirinae, genus *Metapneumovirus*. Avian pneumovirus is a virus within the genus *Metapneumovirus*. Avian pneumovirus has proved difficult to culture and, for that reason, much of the research involving identification of infected birds has used PCR technology. Serological tests are also available.

##### **OIE List**

Not listed.

##### **New Zealand Status**

Turkey rhinotracheitis virus is listed in the unwanted organisms register.

No evidence of turkey rhinotracheitis or of swollen head syndrome has been reported in New Zealand.

##### **Epidemiology**

Four types of avian pneumovirus (APV) have been identified. Subgroups A and B are the common types in Europe. United States isolates have been shown to have significantly different genetic make-up from the European subgroups and have been classified as subgroup C. Two strains isolated in France were classified as subgroup D (1). APV is the cause of Turkey rhinotracheitis and a cause of (or precursor to) swollen head syndrome of chickens and guinea fowl. The major importance of the virus is as a cause of disease in turkeys. Direct contact is believed to be the main means of transmission between birds (2)

In Minnesota, Shin *et al.* (2000) (3) detected APV viral RNA in sparrows, geese, swallows and starlings that had been captured in the vicinity of infected turkey farms. Attempted virus isolation was unsuccessful. Two groups (a and b) of sentinel ducks caged in ponds neighbouring infected turkey farms were PCR positive at weeks 1 and 2 and weeks 8 and 9 respectively. Virus isolation was successful from one duck in group a. Serological testing of group b ducks provided positive titres from week 4. These findings were consistent with the two groups of birds having become infected during the first and seventh weeks of the trial.

Using PCR technology RNA of Avian pneumovirus has been detected in Canadian geese (3, 4), Blue-winged teal (4), mallard ducks, English sparrows, barn swallows and European starlings (3). In these studies, virus was isolated from only wild Canadian

geese(4) and sentinel ducks confined in the vicinity of an infected turkey flock (3). No attempt was made to culture the virus from the passerine species. Antibody to APV was demonstrated in sentinel ducks but passerine species were not tested (3). In chickens experimentally infected with APV, Shin *et al.* (2000) (5) were able to isolate virus for up to 6 days; APV RNA remained detectable for up to 15 days by which time birds had established positive titres to the virus. The authors proposed that RNA detectable by PRC beyond the time that virus could be isolated was probably due to the immune response resulting in a lack of viable virus. This explanation is consistent with the demonstration of the ability to detect APV RNA by PCR following the destruction of virus infectivity using autoclave or microwave treatments (6).

Spread of APV within turkey establishments is thought to be, largely, through direct contact. It is suspected that fomites and movement of people may contribute to local spread but the means of dissemination over longer distances are not known (2). Geographic spread within continents has been slow (3, 7, 8) even though Minnesota (the centre of infection in turkey flocks) is in the centre of major bird migratory routes. Despite the demonstration of infection of ducks held in the vicinity of infected turkey establishments (3) and evidence of APV RNA in other wild birds (3,8) the role of birds in the dissemination of infection remains speculative. Although APV infection in turkeys has been shown to persist for several weeks in turkeys (9), Shin *et al.* (5) found that active infection could only be demonstrated for a short time after infection of chickens. The inability to identify infectious virus in other wild birds (including passerines) with APV RNA and APV antibodies is also consistent with an hypothesis that many infections are terminated shortly after the development of an immune response.

In Europe, APV has been isolated from farm-reared pheasants (10), and an APV with greatest similarity to the United States group C virus has been identified from Muscovy ducks (11).

Although there is some speculation that APV may be transmitted vertically, supporting evidence is scarce and inconclusive. Jones *et al.* (12) found that when immunologically naïve turkeys were infected with APV intra-nasally pathology developed in the oviduct and eggs with abnormal shells developed. There was widespread replication of virus in the epithelium of the reproductive tract for only a short time (detected on days 7 and 9). Virus was not identified in the ovary. They commented that if egg transmission does occur it is temporary, at a very low rate and of little importance in the spread of infection. They further concluded that there was no evidence to-date (1988) that the virus is transmitted through eggs. However, Shin *et al.* (13) suggested, on the basis of the detection of APV RNA in very young turkeys and their later sero-conversion, that vertical transmission may have led to infection of the poults. Experimental infections of chickens with APV have resulted in oviduct pathology and egg abnormalities (14, 15) but neither infection of eggs nor vertical transmission have been demonstrated.

No reports of disease of passerine birds associated with APV have been located.

## Conclusion

Based on the known presence of APV in Europe and evidence that APV genetic material can be identified in passerine birds in the United States, APV is considered to be a potential hazard in the commodity.

### 3.2.2.2 Risk assessment.

#### Release assessment

There is no evidence that APV subgroups A or B infect passerine birds. Nor are there reports of APV infecting passerine species in Europe. Whether APV genetic material detected in passerine birds in the United States (5) represented viable virus infection is unknown. If passerine species do become infected with APV, it is most likely that infection will be transitory.

The paucity of evidence for vertical transmission in turkeys and chickens, the species in which APV is most common, causes most disease and has been most intensively researched, raises doubts over the likelihood that vertical transmission will occur in passerine species. Based on the evidence of Jones et al. (12) from turkeys, any infection of eggs is likely to be restricted to a very short period soon after infection.

The release assessment for APV in passerine eggs imported from the EU is negligible.

#### Risk estimation

Since the release assessment for APV in passerine eggs is negligible, it is not considered to be a hazard in the commodity.

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### **3.3 Herpesviridae**

#### **3.3.1 Duck virus enteritis**

##### **3.3.1.1 Hazard identification**

###### **Aetiological agent**

Duck virus enteritis (DVE) is caused by a herpes virus.

###### **OIE List**

DVE is included in the OIE list of notifiable diseases.

###### **New Zealand Status**

DVE is listed in the unwanted organisms register as notifiable.  
DVE has not been diagnosed in NZ.

###### **Epidemiology**

DVE is a contagious disease of ducks and other waterfowl. One means of transmission of DVE is vertically from hen to egg and hence to hatchlings. No records of duck virus enteritis in Passeriformes species have been found.

###### **Conclusion**

Based on the restricted host range and the absence of reports of DVE in passerines, DVE is not considered to be a potential hazard in the commodity.

### **3.3.2 Infectious laryngotracheitis**

#### **3.3.2.1 Hazard identification**

##### **Aetiological agent**

Infectious laryngotracheitis (ILT) is caused by Gallid herpesvirus 1, a virus within the subfamily Alphaherpesviridae, family Herpesviridae.

##### **OIE List**

ILT is included in the OIE list of notifiable diseases.

##### **New Zealand Status**

ILT is endemic in NZ and is not listed in the register of unwanted organisms.

##### **Epidemiology**

ILT is primarily a disease of chickens and there have been reports of the disease in pheasants (1)

Experimental challenge of peafowl (*Pavo cristatus*), various species of pheasant (*Phasianus colchicus*, *Lophura swinhoii*, *Lophophorus impejanus*), guinea-fowl (*Numida meleagris*), canaries (*Serinus canaria*), budgerigars (*Melopsittacus undulatus*) and Japanese quail (*Coturnix coturni japonica*) found only the peafowl and pheasants to be susceptible. Canaries, the only passerine species included in this trial, proved refractory (2).

No reports of vertical transmission of ILT have been located.

No reports of natural infections of passerine species have been located.

##### **Conclusion**

Based on the narrow host range and lack of reports of ILT in passerines, ILT (Gallid herpesvirus 1) is not considered to be a potential hazard in the commodity.

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### **3.3.3 Marek's disease**

#### **3.3.3.1 Hazard identification**

##### **Aetiological agent**

Marek's disease (MD) is caused by a cell associated herpes virus (MDV).

##### **OIE List**

MD is included in the OIE list of notifiable diseases.

##### **New Zealand Status**

MD is endemic in New Zealand. "Avian herpesvirus (exotic strains) – Marek's disease exotic strains" is listed in the register of unwanted organisms.

Strains of Marek's disease virus and of Turkey herpesvirus are used in vaccines to provide protection against Marek's disease.

##### **Epidemiology**

MD is a transmissible lymphoproliferative disease of chickens present in poultry producing areas around the world. Natural infections have been diagnosed in Japanese quail and turkeys. Sparrows appear to be refractory to MD virus (1). Literature searches have failed to identify reports of MD in passerine birds.

No reports of vertical transmission of Marek's disease virus have been located.

##### **Conclusion**

Based on the narrow host range, the evidence that sparrows are refractory to MD, the lack of reports of MD from passerines, and the absence of reports of vertical transmission, the organism is not considered to be a potential hazard in the commodity

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### **3.3.4 Other avian herpesviruses**

#### **3.3.4.1 Hazard identification**

##### **Aetiological agent**

Herpesviruses are recognised in a number of avian species, and are known to cause a range of diseases including Pacheco's disease and Amazon tracheitis in psittacines, hepatitis in pigeons and others. All avian herpesviruses that have been characterised using REA or PCR methods have been alphaherpesviruses. The viruses tend to be relatively host specific but cross-species and cross-order infections do occur.

##### **OIE List**

Other avian herpesviruses are not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

A probable case of herpesvirus infection of pigeons, characterised by focal hepatocellular necrosis and intranuclear inclusion bodies, has been reported in NZ (1) and antibodies to pigeon herpesvirus are widespread (2).

Two incidents of Pacheco's disease were diagnosed in 1977 on the basis of pathology and viral isolation and characterisation (3). In 1997 cases were encountered in birds in quarantine with some birds from the consignment being illegally removed and not recovered (4). A serological survey, carried out in 2002, resulted in one positive virus Pacheco's virus neutralisation test result in testing carried out in the United States (5). In 1997, material from formalin fixed, paraffin embedded tissues from the 1977 cases was tested for evidence of Pacheco's disease virus using *in situ* hybridation with negative results (Personal communication Loth, L. 7 October 2005). Several months after the initial sampling, the bird that returned the positive serological test in the 2002 survey was resampled (blood and cloacal swab) (6) and both serological and cultural tests carried out at the Ministry of Agriculture and Forestry laboratory were negative (5). The negative results from these repeat tests led to suggestions that New Zealand should be considered free of Pacheco's disease (4, 5). This suggestion, however, has not been universally accepted.

##### **Epidemiology**

Herpesviruses are generally host-specific and spread from host to host by direct contact. Once an animal is infected it will commonly remain infected for life. The virus genome of alphaherpesviruses may become incorporated into neurones of cranial and spinal ganglia. Subsequently the virus can become reactivated, particularly under periods of stress, and be excreted. At times of reactivation clinical signs may reappear (7, 8).



Pacheco's disease (9), is recognised as a cause of disease and death in psittacine species in many countries. Other reported diseases with herpesvirus involved in their aetiology include internal papillomas of parrots in Germany (10), mortalities in cranes in China (11) and Austria (12), haemorrhagic enteritis in storks in Spain (13), oesophagitis in psittacines in Canada (14), coagulative necrosis and death in eagles in Spain (15) and pneumonic disease in parakeets in north America (16) and Europe (17) (termed "Amazon tracheitis" because of the apparent susceptibility of birds in the *Amazona* genus).

Pigeon herpesvirus-1 (PHV-1) causes a disease in pigeons which is widely distributed and commonly presents with signs of respiratory distress and with pathology characterized by multifocal-hepatocellular necrosis with intra-nuclear inclusion bodies. This disease has been reported from Europe (18, 19, 20, 21, 22, 23, 24) the United Kingdom (25, 26), North America (27), Australia (28) and New Zealand (1). Another disease of pigeons presenting with signs of central nervous system disturbance and pathology of encephalomyelitis was first reported in 1979 from Iraq (29) and subsequently from Egypt (30), Poland (31), Saudi Arabia (32) and the Canary Islands (33). This disease was, initially, attributed to a herpesvirus which was named pigeon herpes encephalomyelitis virus (PHEV). Subsequently cultures of the virus have been shown to contain a pigeon paramyxovirus-1 and the disease attributed to either pigeon PMV-1 (34) or, alternatively, a combination of pigeon PMV-1 and PHV-1 (35).

A herpesvirus has been identified as the cause of mortalities in Gouldian finches in Europe (36, 37) and North America (38). Characterisation of the virus from the North American incident, using PCR, indicated that it was an alphaherpesvirus (38). Differences in species susceptibility to the virus causing disease in Gouldian finches were illustrated by the 100% mortality of the finches while no birds of other species in the same room showed any signs of ill-health (37). An epidemic of conjunctivitis and respiratory disease affecting Gouldian finches, and other finch species of Australian origin, in Belgium was attributed to a herpesvirus which, on the basis of histological and ultrastructural characteristics was considered to be a cytomegalovirus (39) however reports of genetic characterisation of the virus have not been identified. The clinical and pathological findings in these cases, together with the host range being restricted to finches of Australian origin leaves open the possibility that all of these incidents had a common aetiology.

Other reports of herpesvirus infection in passeriformes include an inclusion body conjunctivitis in a Red-cheeked Cordon-blue (*Uraeginthus bengalus*) (40), and virus isolations from Bengalese finch (*Lonchura striata*), Northern cardinal (*Cardinalis cardinalis*), canaries (*Serinus canaria*), Zebra finch (*Taeniopygia guttata*), Bronze mannikin (*Spermestes cucullatus*) (41) and a Superb starling (*Lamprolaima superbus*) (42) some of which had central nervous disorders or respiratory disease.

Genetic typing of avian herpesviruses has shown close relationships between passerine and psittacine isolates. Gunther et al (43) compared 15 avian isolates, including five from passerines and two from psittacine on the bases of serotype and REA patterns. The passerine and psittacine isolates were distinct from isolates from other avian orders and

were categorised within serotype 4 (4.1 or 4.2) and restriction pattern group IV (IV a, b, c or d). The authors were able to differentiate the four passerine isolates into types (each from different sources) and the two psittacine isolates into two (again from different sources). They considered that the divergence in REA patterns was greater than that between strains of ILTV tested by other authors and proposed names for four species of Herpesviridae on that basis. Tomaszewski et al. (44), on the other hand, characterised a herpes isolated from a superb starling using PCR and concluded that virus was a psittacine virus PsHV genotype 1. These authors also examined the data from Gunther et al (43) and considered that the viruses of passerine origin examined by those authors also fell within the PsHV genotype 1 category.

Following later standards for the classification and phylogenetic assessment of herpesviruses, Wellehan et al. (45) described the characterisation of a herpesvirus from Gouldian finches in Canada without reference to the work by Gunther et al. The virus of Gouldian finch origin was considered to be an alphaherpesvirus (Passerid HV1) distinct from other herpesviruses but most closely related to Gallid HV1 and Psittacid HV1.

Based on the above, there is a reasonable likelihood that passerine specific herpesviruses exist and that some psittacine herpesviruses can infect passerines. In the absence of genetic characterisation, whether the viruses infecting Gouldian finches in Europe are cytomegalovirus or alphaherpesviruses similar to that identified from Australian finches in Canada is unknown.

There have been relatively few studies on the epidemiology of avian herpesviruses other than in poultry. Serological evidence of infection in clinically normal birds has been found in 47 % of carrier pigeons sampled in Belgium (46) and 60% of pigeons sampled in Germany (47). Vindevogel and others (48, 49, 50, 51) have reported that up to 100% of adult pigeons may be infected with PHV. Infection within a group may persist for over 12 months with some birds continuing to excrete virus. Birds with latent infection excrete virus during the reproductive period and infect their young prior to weaning although the young appear partially protected by passive immunity acquired from the yolk sac. Seven of ten squabs were asymptomatic carriers of the virus after weaning with no detectable antibody. Immune suppression, with cyclophosphamide, of birds with latent infections resulted in re-excretion of virus.

Literature searches revealed one report of vertical transmission of an avian herpesvirus. Burgess and Yuill (52) demonstrated that clinically healthy ducks infected with strains of duck virus enteritis virus (DVEV) laid eggs with decreased hatchability and that this was attributable to DVEV. Some hatchlings died within two weeks while survivors beyond that time carried infections of DVEV and excreted virus. The significance of these findings in the epidemiology of the disease remained unknown because the quantity of virus shed by surviving hatchlings was low and the authors were uncertain whether exposure to such levels of virus would result in infection of other birds. No other reports of vertical transmission of avian herpesviruses have been located.

The observations on the epidemiology of PHV in pigeons are consistent with the behaviour of herpesviruses in other species. In passerine birds, infection with herpesviruses will be more common than clinical disease, latent infections must be expected, some birds with latent infections will be seronegative, recrudescence of infections with excretion of virus will occur at times of stress or when immunity is suppressed and transfer of infection to chicks will take place.

## **Conclusion**

Based on their restricted host ranges neither Pacheco's disease virus nor Pigeon herpesvirus are considered to be potential hazards in the commodity.

Based on the presence of herpesviruses in passerine birds in Europe and the evidence that at least one avian herpesvirus can be transmitted through eggs, passerine herpesviruses are considered to be a potential hazard in the commodity.

### **3.3.4.2 Risk assessment**

#### **Release assessment**

Herpesvirus infections of birds from the order Passeriformes have been diagnosed in Europe. Information on the strains of herpesvirus that may infect passerine birds is incomplete as is information on the prevalence of latent infection. Evidence indicates that at least one psittacine herpes virus can infect passerine species.

Based on one report of vertical transmission of an avian Herpesvirus (DVEV) (Burgess and Yuill (52), the likelihood that infection of eggs of passerine species is considered non-negligible.

The release assessment is non-negligible.

#### **Exposure assessment**

Burgess and Yuill (52), the only authors who have reported vertical transmission of avian Herpesvirus, considered that the quantities of virus excreted by the hatchlings from infected eggs were so small that transmission to other birds was doubtful.

This observation, from an experimental infection, is consistent with the absence of reports of suggesting that vertical transmission of Herpesvirus plays a role in the epidemiology of the organism.

The exposure assessment is negligible.

## Risk estimation

Based on the the scarcity of reports on vertical transmission and the negligible exposure assessment, Herpesviruses are not considered a hazard in the commodity.

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## **3.4 Coronaviridae**

### **3.4.1.1 Hazard identification**

#### **Aetiological agent**

Coronaviridae are large enveloped RNA viruses.

Recognised avian diseases caused by Coronaviruses are

1. Infectious bronchitis of chickens,
2. Turkey coronavirus enteritis,
3. Poult enteritis-mortality syndrome and
4. a respiratory / renal disease of pheasants.

#### **OIE List**

Infectious bronchitis is included in the OIE list of notifiable diseases.

#### **New Zealand Status**

Infectious bronchitis virus (IBV) is endemic in New Zealand.

Infectious bronchitis (exotic strains) is listed on the register of unwanted organisms.

Coronavirus infections of turkeys or pheasants have not been reported in NZ.

#### **Epidemiology**

Infectious Bronchitis is a coronavirus disease of chickens. Chickens are the only species recognised as being naturally infected with IBV and in which it causes disease. Very similar viruses have been isolated from pheasants. IBV did not cause disease in pheasants, turkeys or starlings when administered experimentally (1).

Turkey coronavirus enteritis is an acute highly contagious disease of turkeys. It has been recognised in the United States, Canada and Australia. Turkeys are the only species recognised as being naturally infected with this virus (2).

Poult enteritis-mortality syndrome is a contagious disease of young turkeys in which it is thought that coronavirus plays a role. It is thought that it is multifactorial disease which may involve a combination of viral, bacterial and protozoal agents (3).

Coronavirus isolates from pheasants are genetically similar to IBV but appear to have a different host range (1).

Literature searches have failed to identify reports of coronavirus infection in passerine birds.



## **Conclusion**

Based on the host specificity of recognised strains of avian coronaviruses and the lack of reports of coronaviruses in passerine species, corona viruses are not considered to be potential hazards in the commodity.

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## 3.5 Adenoviridae

### 3.5.1 Group I adenoviruses

McFerran (1), reviewing avian adenoviruses, highlighted the widespread distribution of adenoviruses in healthy birds, their role as opportunistic pathogens and the place of a small number of these viruses as primary pathogens. The avian adenoviruses have been placed in three groups based on serological differences. Their known distribution extends from chickens and turkeys to long-tailed ducks in Alaska (2) to southern giant petrels in Patagonia (3).

#### 3.5.1.1 Hazard identification

##### **Aetiological agent**

Group I adenoviruses are in the genus aviadenovirus. A number of subtypes are identified based on the usual species infected and serological differences. These include (4)

Usual species infected	Sub-group name	Numerical identifier
Chickens	FAV	1 to 12
Geese	GAV	1 to 3
Ducks	DAV	2
Turkeys	TAV	1 and 2
Pigeons	PiAV	-

##### **OIE List**

Group 1 adenoviruses are not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Not listed in the unwanted organisms register.

A number of the Group I FAV viruses are endemic in New Zealand. FAV 1, 4, 5 and 8 were isolated from domestic hens, some with a variety of clinical conditions but most clinically healthy (5) and types 1, 8 and 12 from commercial broiler flocks with signs of inclusion body hepatitis (IBH) (6). Serological reactions to avian adenoviruses are found routinely in flock surveillance programmes (7).

An adenovirus was isolated from broiler chickens in New Zealand in which hepatic inclusion bodies accompanied marked liver pathology in 12 flocks. A diagnosis of inclusion body hepatitis (IBH) was made. The possible contribution to a period of poor egg production in the breeder flock at the time of production of the eggs for the affected flocks was raised.(8). Positive serology to adenovirus is common in both domestic and feral pigeons throughout New Zealand (9).

## Epidemiology

Adenoviruses are widespread in chicken populations and adenoviruses of the chicken serotypes have been recovered from a range of other species (10). Antibodies to adenoviruses are sufficiently common to have lead Hess (11) to comment that serology is of little value as a diagnostic tool. In chickens, vertical transmission through eggs is common. Horizontal transmission is also important. Spread by fomites can take place and venereal transmission in semen is also possible, associated with renal infection and excretion.(10) Latent infections are known to establish in chickens and be transmitted between generations (12).

A syndrome of IBH in chickens is commonly attributed to Group I adenovirus. McFerran *et al.* (13) reported the isolation of adenoviruses types 2, 3, 4, 5 and 8 from outbreaks of IBH in chickens but cautioned the interpretation of these findings pointing out that adenoviruses could be isolated from virtually all broiler flocks at the time that maternal antibody had diminished. Similar reservations over the role of adenoviruses in incidents in which hepatic inclusion bodies are found in chickens have been expressed by others (8, 14).

Hepatic inclusion bodies associated with adenoviruses (or adenovirus-like particles) have also been reported from psittacines(15, 16, 17), American kestrels (18), pigeons (19) and geese(20). Other reported sites of inclusion bodies associated with adenoviruses include the intestines (21) and renal tubules of psittacines (22). McFerran (23) reported the isolation of adenoviruses (types 2, 5 and 8) from a range of avian species with a variety of clinical diseases and Pennycott (24) reported the isolation of an adenovirus, along with a reovirus-like organism, *Escherichia coli* and *Clostridium perfringens* from episodes of high mortality in budgerigars in Scotland. Authors commonly expressed reservations over the role of the adenoviruses as primary pathogens.

One report of the finding of an adenovirus in passerine birds was located. That infection, of Gouldian finches in a single aviary in California, was considered likely to be consequent to infection of the birds with a circovirus. The circovirus infections were associated with lymphoid depletion in both the bursa of Fabricius and the thymus and concurrent infections with *E. coli* and *Klebsiella oxytoca* were also present. The adenovirus infection was located in the renal tubules and was associated with a mild nephritis (25). For the purpose of this risk analysis, this infection will be considered as a Group I adenovirus.

## Conclusion

Group I avian adenoviruses are considered to be potential hazards in the commodity.

### **3.5.1.2 Risk assessment**

#### **Release assessment**

The release assessment for Group I adenoviruses in passerine eggs from Europe is based on the general ubiquitous nature of these viruses and the evidence that transmission through eggs take place.

The release assessment is non-negligible.

#### **Exposure assessment**

Hatchlings from imported eggs may be infected. Both horizontal and vertical transmission may lead to spread of the virus.

The exposure assessment is non-negligible.

#### **Consequence assessment**

There is only one report of an adenovirus associated with disease in Passeriformes. That case was in California, was as a co-infection with a circovirus and was associated with only a mild nephritis.

The consequence assessment for adenoviruses in passerine eggs imported to New Zealand from Europe is negligible.

#### **Risk estimation**

Based on the absence of reports of Group I adenoviruses causing ill health in passerine species in Europe, these viruses do not present a hazard to New Zealand bird species (endemic, native or introduced), the New Zealand poultry industry agriculture, the economy or the environment in the importation of birds within the commodity definition addressed by the risk analysis.

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### **3.5.2 Group II adenoviruses – HE, MSD, AAS**

#### **3.5.2.1 Hazard identification**

##### **Aetiological agent**

Group II adenoviruses fall within the proposed genus of Siadenoviruses. The unique feature of the members of this group is their genetic coding for sialidase (1). The viruses in this group cause haemorrhagic enteritis (HE) of turkeys, marble spleen disease (MSD) of pheasants, and avian adenovirus splenomegaly (AAS) of chickens.

##### **OIE List**

Group II adenoviruses are not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Not listed on the unwanted organisms register.

Literature searches have not revealed any New Zealand record of the diagnosis of any of the diseases caused by Group II adenoviruses.

##### **Epidemiology**

The host range of each of the diseases associated with Group II adenoviruses is limited. Haemorrhagic enteritis is a disease of turkeys from four weeks old. Marble spleen disease affects pheasants between three and eight months old raised in captivity and AAS occurs in broiler chickens. Disease has been diagnosed in guinea fowl and there is one report of a suspect infection in psittacine birds (2). A survey of 618 wild birds of 42 species in the south eastern United States for antibodies to the Haemorrhagic enteritis / Marble spleen disease group of viruses produced negative results. This survey included 207 passerines from eight families with all passerines sampled being from Florida (3).

##### **Conclusion**

As group II adenoviruses are recognised as avian pathogens and are not present in New Zealand they are considered to be potential hazards in the commodity.

#### **3.5.2.2 Risk assessment**

##### **Release assessment**

The host range of Group II adenoviruses is limited. No reports of Group II adenoviruses in non-captive birds have been located, reports of natural infections in species outside the gallinaceous group are restricted to one, from psittacines (4). Searches for reports of infection in passerine species have been unsuccessful.

On the basis of the restricted range of hosts from which Group II adenovirus have been reported and the lack of reports from passerine species, the likelihood of Group II adenoviruses being present in passerine eggs from Europe is negligible.

The release assessment is negligible.

### **Risk estimation**

With the proposed importation of passerine eggs from Europe, Group II adenoviruses do not present a hazard to New Zealand native, endemic or other wild birds. Nor do they present a hazard to poultry, agriculture, human health or the environment.

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### **3.5.3 Group III adenoviruses – EDS**

#### **3.5.3.1 Hazard identification**

##### **Aetiological agent**

The aetiological agent for egg drop syndrome (EDS) is the sole member of Group III adenovirus.

##### **OIE List**

EDS is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

EDS is not included in the register of unwanted organisms.

##### **Epidemiology**

Egg drop syndrome is a contagious disease of chickens, spreading through both lateral and vertical transmission and causing decreased egg production. The virus is also recognised in ducks, geese and quail (1).

EDS was first diagnosed in New Zealand in 1981 (2). Although serological prevalence remained low through the 1980s and Howell (3, 4) predicted that the virus was not likely to persist in commercial flocks, infection, as evidenced by serological titres, has remained (5, 6).

No reports of natural infections of passerines with EDS have been located.

##### **Conclusion**

Based on its restricted host range, and its presence in New Zealand, EDS is not considered to be a potential hazard in the commodity.

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## 3.6 Poxviridae

### 3.6.1 Avipoxvirus

#### 3.6.1.1 Hazard identification

##### Aetiological agent

Order – mononegavirales; Family – Poxviridae; Genus – Avipoxvirus.  
Poxviruses are enveloped DNA viruses.

Recognised species within the Avipoxvirus genus are (1):

1. Fowlpox virus (three species),
2. Pigeonpox virus
3. Psittacinpox virus
4. Quailpox virus
5. Turkeypox virus
6. Starlingpox virus - \*
7. Sparrowpox virus - \*
8. Canarypox virus - \*
9. Juncopox virus - \*
10. Mynahpox virus - \*

Tentative species are (1):

11. Crowpox virus
12. Peacockpox virus
13. Penguinpox virus

Note: in the above list, the viruses marked \* have passerine species as their primary host.

##### OIE List

Fowlpox is included in the OIE list of notifiable diseases.

##### New Zealand Status

Not listed in the unwanted organisms register.

Fowlpox is widespread, especially in the northern parts of New Zealand. Pigeonpox and fowlpox vaccines have been used routinely in endemic areas in the past. Both cutaneous and “wet” (laryngeal) pox are seen in chickens (2). Poxvirus infections have also been diagnosed in turkeys, pigeons and canaries<sup>+</sup> (3). Gartrell *et al.* (4) listed pipit (*Anthus novaeseelandiae*<sup>+</sup>), shore plover (*Thinornis novaeseelandiae*), variable oyster catcher (*Haematopus unicolor*), weka (*Gallirallus australis*), NZ wood pigeon (*Hemiphaga novaeseelandiae*), thrush (*Turdus philomelos*<sup>+</sup>), silver eye (*Zosterops lateralis*<sup>+</sup>), black robin (*Petroica traverse*<sup>+</sup>) and North Island robin (*Petroica Australia*<sup>+</sup>) as species in

which avian pox had been diagnosed. Pox infection of an Oyster catcher (*Haematopus leucocephalus*) has also been reported (5). The status of New Zealand with respect to psittacinepox virus is uncertain following the finding of psittacinepox in a quarantine aviary and illegal removal of birds from it (4). The six species marked <sup>+</sup> are in the Order Passeriformes.

## Epidemiology

Poxviruses are stable in dry environments and can be transmitted by aerosols, direct contacts, fomites or by biting insects. Latent infections occur and these may be reactivated during times of stress (6).

Bolte *et al.* (7) reviewed the avian species reported as infected with avipoxviruses and included 232 species from 23 orders in their list. That list included 99 species within the Passeriformes Order but only three of the passerine species identified with avian pox in NZ. The true number of species that are vulnerable to infection with avipoxviruses must be very large.

As with other poxviruses, avipoxviruses are generally considered to be host specific or to have a narrow host range. Reports in the literature, however, provide examples where avipoxviruses appear to have host ranges of varying scope. Some examples that illustrate this mixed picture include:

- Infection of caged Rothschild's mynahs (*Leucospar rothchildii*) by virus from starlings (*Sturnus vulgaris*) gaining access to the aviary while over a hundred birds of different species remained unaffected (8) (infection of birds in different genera within the Family *Sturnidae* but not birds in other families – this incident was consistent with observations by Williamson (9) that Starlingpox is specific to the *Sturnidae* family),
- Failures of attempts to infect chickens and canaries (*Phylloscopus canariensis*) with poxvirus from diseased Amazon parrots (*Amazona aestiva*) (10) (failure to infect birds in another genus or Family),
- Non-pathogenicity, to fowl, turkey, duck and pigeon, of poxvirus from diseased Red Siskins (*Carduelis cucullatus*) while a minor lesion developed in a bird of another Siskin species (11) (infection of a bird in another species within the same genus but not of birds in other orders) and
- An outbreak of pox in an aviary restricted to canaries (*Serinus canaria*) and house sparrows (*Passer domesticus*) although 10 other passerine species were present (12) (restriction of infection to two species and genera with the order Passeriformes.)
- Avipoxvirus infection affecting five species of pheasants and free-ranging Indian red junglefowl (*Gallus gallus murghi*) in a zoo at the same time (13) (infection of birds from several genera within the family Phasianidae),

- Infection of chickens and bobwhite quail with poxvirus from mynahs (*Gracula religiosa*) imported from Malaysia (14) (Note – Infection was dependent upon route of inoculation. Cutaneous inoculation resulted in lesions whereas swabbing of virus onto conjunctivae and oral mucosa did not.) (Infection of birds from the Order Galliformes with virus from a passerine bird),
- Infection establishing in *Amazona ochrocephala* (Yellow crowned parrot), *Aratinga holochlora* (Perico mexicano) and chickens following inoculation with a virus isolated from *Amazona albifrons* (White fronted parrot) (15). (Infection of birds of different species within the same genus and of birds in a different genus.) and
- Simultaneous cases of pox infection in house finches, (*Carpodacus mexicanus*), mourning doves (*Zenaida macroura*), house sparrows (*Passer domesticus*), robin (*Turdus migratorius*) and a golden eagle (*Aquila chrysaetos*) (16). (Infection of birds in different families with the same order and of a bird in a different Order.)

These observations are consistent with those of Kirmse (17), who recognised examples of cross-species and cross-Order infections and also commented that cross immunity was highly variable.

With over 100 species in the Order Passeriformes recorded as having been infected with Avipoxvirus, and five Avipoxvirus species recognised as being sufficiently host related to passerine birds to be named after them, there remains a great deal to learn about the range of avipoxviruses that infect passerines and their host ranges.

## Conclusion

Avipoxviruses are considered to be a potential hazard in the commodity.

### 3.6.1.2 Risk assessment

#### Release assessment

No reports suggesting vertical transmission of avian poxviruses have been located.

The release assessment for avipoxviruses in passerine eggs imported from Europe is negligible.

#### Risk estimation

It is concluded that Avipoxviruses are not a hazard in the commodity.

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## **3.7 Circoviridae**

### **3.7.1 General**

#### **3.7.1.1 Hazard identification**

##### **Aetiological agent**

Circoviruses are small, non-enveloped viruses that replicate in the nucleus of cells. They are stable in the environment and maintain infectivity through temperatures up to 60 degrees for 30 minutes (1).

The family Circoviridae contains two recognised genera;

- Gryovirus - the only member of which is the Chicken anaemia virus and
- Circovirus – which includes Porcine circovirus and Beak and Feather disease virus (2).

In addition, unidentified Circovirus-like viruses have been identified in birds>

##### **OIE List**

No members of the Circoviridae are included in the OIE list of notifiable diseases.

##### **NZ status**

No avian members of the Circoviridae are included in the register of unwanted organisms.



### **3.7.2 Chicken infectious anaemia**

#### **3.7.2.1 Hazard identification**

##### **Epidemiology**

Chicken infectious anaemia virus (CIAV) is established and widespread in New Zealand (3, 4). It infects only chickens.

##### **Conclusion**

On the basis of its presence in New Zealand and its restricted host range, CIAV is not considered to be a potential hazard in the commodity.

### **3.7.3 Psittacine beak and feather disease**

#### **3.7.3.1 Hazard identification**

##### **New Zealand Status**

Ritchie *et al.* (5) identified psittacine beak and feather disease virus (PBFDV) in 21 of 25 captive psittacine birds sampled in New Zealand. These positive results came from eight of the ten species included in the study. Genotype clustering of viruses within related psittacine species was found and these clustering patterns were similar to those seen in similar species in Australia. This has led to the suggestion that PBFDV was introduced into New Zealand in psittacines imported from Australia. In 2003, 5 of 71 captive psittacines sampled by Jakob-Hoff (6) were positive for PBFDV, while all 76 wild psittacines sampled were negative by PCR.

##### **Conclusion**

On the basis that PBFDV is present in New Zealand it is concluded that PBFDV is not a potential hazard in the commodity.

### **3.7.4 Circovirus-like viruses**

#### **3.7.4.1 Hazard identification**

##### **Aetiological agent**

Pigeon circovirus (PiCV) has been accepted as a tentative member of the Circovirus genus (7) and Todd (8) listed reports of circovirus-like viruses being identified in Senegal doves, canaries, finches, ostrich, goose and a gull between 1994 and 1999. Since Todd's review Circovirus-like agents have been identified in pheasants (9) and mulard ducks (10).

##### **New Zealand Status**

A circovirus-like virus was identified in a southern black-backed gull that was found terminally ill with *Aspergillus spp.* infection and severe airsacculitis in the Manawatu region (11).

##### **Epidemiology**

Available evidence indicates that specific genotypes of circoviruses have restricted host ranges. Ritchie *et al.* (5) identified clustering of genotypes of PBFD viruses from taxonomically related psittacine species. Different viral groupings were associated with lorikeets, cockatoos and a budgerigar. The genome of PiCV is sufficiently distinct from that of PBFDV for it to have been given tentative status as a virus in the Circovirus genus (7). Reports of Circovirus in Senegal doves (12), canaries (13, 14), mulard ducks (10) and geese (15) have each claimed that these viruses are novel. Shivaprasad (16) reported circovirus infection associated with lesions of lymphoid depletion of the bursa of Fabricius and the thymus in a Gouldian finch from an aviary in which 50% of the birds of that species died while other species of finch in the aviary remained healthy. Todd (8) expressed the opinion that "other mammalian and avian species are likely to be infected with novel circoviruses that are presently unrecognized" and went on to suggest that "now that the characteristic lesions are becoming better known to avian pathologists, it is likely that reports describing circovirus infections of additional avian species will be forthcoming". In the five years since Todd's review was published, these forecasts have proven to be correct.

Consistent pathological findings in Circovirus-associated disease in birds include lesions of lymphoid tissue including the bursa of Fabricius. The pathogenesis of disease appears to include immune suppression allowing other pathogens or opportunist pathogens to infect the bird(s) and cause disease. Diagnoses of Circovirus-like infections are largely dependent upon recognition of inclusion bodies in lymphoid tissues and investigations using electron microscopy. Investigations of the prevalence of PBFDV and PiCV, for which PCR tests are available, have revealed that infections of psittacine and columbid species, respectively, are mainly in healthy birds.

Studies of the epidemiology of Circovirus and Circovirus-like viruses has been hampered by difficulties in growing the virus in tissue culture. PBFDV, however, is thought to be transmitted both horizontally and vertically (1) but specific information on the means of transmission of the Circovirus-like viruses is not available. It is assumed that circovirus-like viruses can be transmitted by the same routes as those proposed for PBFDV.

## Conclusion

Based on the following factors

- Both recognised avian Circoviridae (CIAV and PBFDV) are established in New Zealand,
- The finding of a Circovirus-like infection in a southern black-backed gull in New Zealand raises the possibility that other birds may also be infected,
- Thousand of passerine birds have entered New Zealand without biosecurity measures that would have restricted the entry of Circovirus-like viruses,
- In species in which investigations have taken place, Circovirus-like viruses are common in healthy birds,
- Very few passerine birds are subject to laboratory diagnostic procedures in New Zealand and procedures likely to identify infection with Circovirus-like viruses will rarely be used

it is likely that Circovirus-like infections are present in birds in New Zealand, either as a result of long-term presence, or as the result of the introduction with imported birds.

It is concluded that Circovirus-like viruses are not a potential hazard in the commodity.

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## **3.8 Birnaviridae**

### **3.8.1 Infectious bursal disease**

#### **3.8.1.1 Hazard identification**

##### **Aetiological agent**

Infectious bursal disease virus (IBDV) is the only species within the Avibirnavirus genus within the Birnaviridae. Sixteen strains are recognised by the International Committee on Taxonomy of Viruses (<http://www.ncbi.nlm.nih.gov/ICTVdb/Ictv/index.htm>). Strains are commonly differentiated into two based on serological grouping.

##### **OIE List**

IBDV is included in the OIE list of notifiable diseases.

##### **New Zealand Status**

IBDV (Exotic strains) is listed in the unwanted organisms register as a notifiable organism.

Serological surveys had indicated that NZ poultry were free of IBDV until its presence was detected in 1993 (1). The virus was less contagious and less persistent in the environment than classical strains (2). Subsequently, IBDV isolated in NZ were characterised as type 1 and, in challenge trials in SPF chickens, did not cause clinical disease (3). Implementation of a national control programme by the poultry industry has resulted in a situation where commercial poultry flocks have been sero-negative since 1999 and national freedom has been viewed as a realistic goal (4). The serological status of non-commercial poultry or of caged or wild birds is unknown.

##### **Epidemiology**

IBDV is, generally, considered to be a disease of chickens in which species it is highly contagious and a cause of significant disease. The virus persists for several weeks in vacated chicken houses. In susceptible chicken flocks morbidity may be 100% while mortality varies between nil and 30%. Following establishment of infection in a flock, disease is less marked and may be non-clinical. Apart from its role as a primary cause of disease, IBDV infection results in immunosuppression and increased susceptibility to other infectious diseases (5).

IBDV has been isolated from chickens, turkeys, ducks, ostriches (5) and penguins (6). Serological evidence of infection has been found in sparrows, ducks and geese in China (7), from rooks, wild pheasants and several rare species of birds in Ireland (8), several species of wild birds including carrion crow and jungle crow (9), and accipitrid birds (the family including hawks, eagles, buzzards and Old World vultures, harriers and kites)(10).

In Japan, 4 of 212 carrion crow were positive to IBDV serotype 1 and 10 to serotype 2. Reports of isolation and characterisation of IBDV from passerines have not been located.

Attempts to infect Guinea fowl (11, 12), pheasants, partridges and quail (12) have not resulted in clinical signs of disease or lesions discoverable post-mortem.

## **Conclusion**

IBDV is considered to be a potential hazard in the commodity.

### **3.8.1.2 Risk assessment**

#### **Release assessment**

Isolation of IBDV from passerine birds has not been reported but positive serology from sparrows (7), rooks (8) and crows (9) suggests that infections of passerines may occur. There is no information on the persistence of infection in non-poultry species.

There is no evidence that IBDV can be transmitted vertically through eggs.

The release assessment is negligible.

#### **Risk estimation**

IBDV is not a hazard in the commodity.

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## **3.9 Papovaviridae**

### **3.9.1 Polyomavirus**

#### **3.9.1.1 Hazard identification**

##### **Aetiological agent**

The genus Polyomavirus is within the Family Polyomaviridae. The only Polyomavirus of birds recognised by the International Committee on Taxonomy of Viruses is Budgerigar fledgling disease virus (BFDV).

##### **OIE List**

Avian polyomavirus is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Not listed in the unwanted organisms register.

A survey by Jakob-Hoff (1) provided positive serological evidence of the presence of avian polyomavirus in two umbrella cockatoos, one sun conure, and one sulphur crested cockatoo. These findings confirm the speculation by epidemiologists (2) and the suspicions of veterinary pathologists (3) and an avian microbiologist (4) that avian polyomavirus is present in New Zealand.

##### **Epidemiology**

Avian Polyomavirus has been reported from many parts of the world, including Europe, and is considered by many to be ubiquitous within avian populations. BFD affects a wide range of psittacine species with young birds being most susceptible. Morbidity and mortality in affected groups can be up to 100%. Horizontal transmission is the main means of spread but vertical transmission may also take place (5). Disease and mortality attributed to Polyomavirus, has also been reported from a number of passerine species (6, 7, 8, 9, 10). Lafferty *et al.* (8), using PCR technology found that the virus in a Green aracaris (*Pteroglossus viridis* – Order Piciformes (Woodpeckers)) was a variant of previously characterised Polyomavirus(es). Serological testing of remaining birds in the aviary produced positive results from birds in four orders including both psittacine and passerine species. Analysis of the genotypes of 20 isolates of Polyomavirus from various locations, times and bird species lead Phalen *et al.* (11) to conclude that species-specific types had not developed.

## Conclusion

Based on the evidence for the presence of Polyomavirus in New Zealand and the apparent lack of species specificity, avian Polyomavirus is not considered to be a potential hazard in the commodity.

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## **3.9.2 Papillomavirus**

### **3.9.2.1 Hazard identification**

#### **Aetiological agent**

Papillomavirus is a genus within the family Papillomaviridae.

#### **OIE List**

Avian papillomavirus is not included in the OIE list of notifiable diseases.

#### **New Zealand Status**

Not listed in the unwanted organisms register.

No reports of Papillomavirus in birds in New Zealand have been located.

#### **Epidemiology**

Papillomaviruses are, generally, host specific and tissue specific. Lesions in different tissues of the same species are caused by different viral types (1, 2). Cloacal papillomatosis is a condition of “new world” psittacines (3), proventricular papillomas are seen in *Amazona* spp. and macaws (3) and cutaneous papillomas are most common in African grey parrots (4). There are doubts that cloacal papillomatosis is caused by a Papillomavirus (5, 6, 7). Papillomaviruses may be latent with no signs of infection until activated during a period of stress. Virus is shed with cells desquamating from the surface of papillomas (1).

Amongst birds of the Order Passeriformes, papillomatosis has been reported most frequently from chaffinches (*Fringilla coelebs*). Literak *et al.* (8) identified 12 reports from this species. Countries of origin were Great Britain, Germany, Netherlands, Sweden and the Czech Republic and all reports involved lesions on the feet and/or legs and/or claws. Most reports were of single cases although one was of an incident in which 10 of 20 birds in an aviary were affected. Lesions can persist and grow for at least two years. Papillomatosis has also been reported from canaries (*Serinus canaries*) (2, 9) and greenfinches (*Carduelis chloris*) (10). Lina *et al.* (11) reported infection of both chaffinches (*Fringilla coelebs*) and bramblings (*F. montifringilla*) in one incident whereas all other reports of apparent Papillomavirus infection in passerines traced have involved only one species although other birds (including passerines) may have been sharing the aviary.

Very few reports of characterization of Papillomaviruses from birds has been located. Moreno-Lopez *et al.* (12) found that individual viruses from chaffinches in Sweden and Holland were closely related but that there was little genetic homology with a bovine Papillomavirus. A Papillomavirus isolated from an African grey parrot (*Psittacus*

*erithacus timneh*) was found to be distinct from 17 mammalian and one chaffinch viruses tested (13).

## **Conclusion**

Papillomaviruses of passerines are considered to be a potential hazard in the commodity.

### **3.9.2.2 Risk assessment**

#### **Release assessment**

Papillomaviruses have been reported from only very few passerine species and then, with the exception of chaffinches, on only very few occasions and with low prevalence of affected birds. Although the existence of Papillomaviruses in other species can not be excluded, it seems that such viruses must be either infrequent or of very low pathogenicity.

No reports suggesting or demonstrating vertical transmission of avian Papillomaviruses through eggs have been discovered.

The release assessment for Papillomaviruses in passerine eggs is negligible.

#### **Risk estimation**

Based on a negligible release assessment it is concluded that avian Papillomaviruses are not a hazard in the commodity.

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### **3.10 Parvoviridae and other non-specific enteritis-associated agents**

#### **3.10.1 Parvoviruses**

##### **3.10.1.1 Hazard identification**

###### **Aetiological agent**

Parvovirus is a genus within the Parvoviridae. They are DNA viruses replicating within the nuclei of dividing cells.

###### **OIE List**

Goose parvovirus is not included in the OIE list of notifiable diseases.

###### **New Zealand Status**

Goose parvovirus is listed in the unwanted organisms register.  
No reports of parvovirus infections of birds in New Zealand have been located..

###### **Epidemiology**

Goose parvovirus infection (Derzsy's disease) is not recognised as infecting species other than geese, Muscovy ducks and some hybrid breeds (1).

Both chicken anaemia and psittacine beak and feather disease have been associated with parvovirus-like organisms but their causative organisms are now classified as circoviruses. (See section 3.7)

The only reference located suggesting the presence of a Parvovirus-like organism in passerine birds is that by Helfer et al (2) who reported an incident of myocarditis-encephalopathy in canaries accompanied by parvo-virus like intranuclear inclusions in brain and heart. This report was from California.

Goose Parvovirus is transmitted vertically through eggs.

###### **Conclusion**

Parvoviruses are considered to be a potential hazard in the commodity.

### **3.10.1.2 Risk assessment**

#### **Release assessment**

Based on there being only one report of a suspect parvovirus-like infection in passerines and that being from California, the likelihood of parvovirus being present in the commodity is considered negligible.

The release assessment is negligible.

#### **Risk estimation**

Based on the negligible release assessment, it is concluded that Paroviruses are not a hazard in the commodity.

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### **3.10.2 Enteritis associated viruses (see also astrovirus and rotaviruses)**

#### **3.10.2.1 Hazard identification**

##### **Aetiological agent**

Searches of electronic databases and text books for reports of enteritic diseases of passerine species caused by viruses other than astrovirus, rotavirus or parvovirus failed to produce positive results. Such viruses, therefore, are not a hazard in the commodity.



## **3.11 Togaviridae**

### **3.11.1 Equine encephalitis (Eastern, Western and Venezuelan equine encephalitis)**

#### **3.11.1.1 Hazard identification**

##### **Aetiological agent**

The aetiological agents for these encephalitides are Alphaviruses within the family Togaviridae.

##### **OIE List**

Equine encephalitis are included in the OIE list of notifiable diseases.

##### **New Zealand Status**

These organisms are listed in the unwanted organisms register as notifiable. They have not been diagnosed in NZ.

##### **Epidemiology**

Eastern, Western and Venezuelan equine encephalitides are caused by arthropod borne viruses which also infect birds including passerine species. These diseases (and viruses) are found only in the Americas (1).

##### **Conclusion**

Based on the limited geographic distribution of these viruses, they are not considered to be potential hazards in the commodity.

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### **3.11.2 Other Alphaviruses**

#### **3.11.2.1 Hazard identification**

##### **Aetiological agent**

Alphaviruses are in a genus within the family Togaviridae. They are arboviruses. (That is, insects or other arthropods act as vectors.)

##### **OIE List**

Avian alphaviruses are not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

Not listed in the unwanted organisms register.  
Whataroa virus has been reported from NZ.

##### **Epidemiology**

No reports of Alphaviruses causing disease in birds have been encountered. The viruses circulate between vertebrate hosts and insects (most reports refer to mosquitoes).

Alphaviruses identified as infecting birds are:

Buggy Creek virus and Fort Morgan virus – These viruses infect cliff swallows and sparrows in central USA. No reports of disease associated with these viruses have been located.

Getah virus – This virus is reported from east Asia (China, Japan, Malaysia, Indonesia) and India. It is reported as infecting Columbiformes birds.

Chikungunya virus – reported from Africa and reported as infecting birds.

Mayaro virus – Reports of this virus are limited to coming from South America where birds are reported to be amongst the species infected.

Highlands J virus – Reported from the eastern United States infecting blue jays, scrub jays, turkeys and chukar partridges and other avian species including passerines.

Ross river virus – This virus is distributed within Australia and in islands to its north. Reports of its being found in passeriformes were not located.

Simliki Forest virus – Reports from China, Senegal and Romania indicate that this virus does infect birds. There is serological evidence that this virus is present in central and

southern Europe and it has been identified as infecting water fowl. No reports of evidence of it infecting passerine species have been found.

Sindbis virus – Although Sindbis virus is widely distributed, with reports of its recognition from Europe, Africa, Asia and Australasia, it is inappropriate to regard the virus as an homogenous population for risk analysis purposes. Examinations of the antigenic and genetic characteristics of Sindbis viruses from different geographic locations have allowed them to be placed into two main groups. Those from Palearctic – African locations and those from Asia and Australia. Further genetic, and resulting phenotypic, changes have taken place in Sindbis viruses within those major geographic groupings. Results of these analyses show that Sindbis viruses from particular zoogeographic regions are restricted to that region even though birds and other major vertebrate hosts are not (1, 2). The authors commented that the genetic change might be due to genetic drift or to adaptation but they were unable to determine which. Later analyses have placed isolates of Ockelbo virus (from Sweden and Russia) as a sub-strain of Sindbis virus (3). Based on RNA analyses Whataroa virus is within the Sindbis group but showing greater divergence than other members of the group (4, 5) and has not been reported as being associated with disease in humans. On these bases, Sindbis virus from locations other than New Zealand are regarded as exotic organisms and should be subject to hazard analysis on that basis. The organisms considered in this analysis are those known as Sindbis virus and Ockelbo virus and are endemic within Europe.

Evidence of infection (either isolation of virus or demonstration of antibodies) of birds in Europe with Sindbis virus has been reported from house sparrows and a number of other passeriformes species in Czechoslovakia (6, 7, 8), house sparrows and tree sparrows in Poland (9), and 17 passerine species in Sweden (10). Serological evidence of Sindbis virus infection has been found in 10 avian species, including seven passerine species, in the UK (11). Although Sindbis virus has been identified in other species (12), the great majority of infections recognised have been in Passeriformes. Lundstrom (13) demonstrated that Sindbis virus infections, with viraemias, could be established in Anseriformes, Galliformes and Passeriformes birds. Lundstrom *et al.* (10) concluded that thrushes are the main amplifying hosts for Sindbis virus in Sweden.

Sindbis virus is an arbovirus. The vectors proposed for transmission between birds in Sweden are *Culex pipiens pipiens*, *Cx. torrentium*, *Culiseta morsitans* and *Cs. ochroptera*. *Aedes communis* and other *Aedes* spp. are suspected as the vectors between birds and man (14). *Cx. univittatus* was considered the main vector for both bird to bird and bird to man transmission of Sindbis virus in South Africa (15).

Sindbis viruses have been associated with human disease in northern Europe, Egypt and South Africa (15). L'vov *et al* (16) concluded that Ockelbo disease, Pogosta disease and Karelian diseases in Sweden, Finland and Russia, respectively, are synonyms for the same disease from which Sindbis virus has now been isolated (17). In northern Europe, this disease presents as periodic epidemics affecting hundreds (or thousands) of individuals with rash, arthralgia and moderate fever (18, 19) the effects of which may be prolonged in excess of 30 months (20).

Whataroa virus has been identified in southern Westland in the South Island of NZ. It infects a number of bird species with serological prevalence varying between years and with time of year(21). Over the period of the study by Miles (22), the highest serological prevalences were in Thrushes (*Turdus philomelos*), hedge sparrows (*Prunella modularis*), Chaffinches (*Fringilla coelebs*), Bell birds (*Anthornis melanura*) and Blackbird (*Turdus merula*). The main mosquito vectors were thought to be *Culiseta tonnoiri* and *Culex pervigilans*. These two mosquitoes, together with *Opifex fuscus*, *Aedes notoscriptus* and *Ae australis* have been shown in laboratory testing, to be capable of acting as hosts to Whataroa virus (23). Whataroa virus is not reported as being associated with disease in animals or humans.

## **Conclusion**

Alphaviruses infecting birds are considered to be potential hazards in the commodity.

### **3.11.2.2 Risk assessment**

#### **Release assessment**

Neither Buggy Creek virus nor Fort Morgan virus nor Getah virus nor Chikungunya virus nor Mayaro virus nor Highlands J virus nor Ross river virus have been identified in Europe.

Simliki Forest virus has been identified in Roumania and may be present elsewhere in Europe but it has not been identified in passerine species.

Sindbis virus is present in passerine species in Great Britain and elsewhere in Europe.

Vertical transmission of Alphaviruses in avian species has not been reported.

On the above bases, the release assessment for alphaviruses in passerine eggs is negligible.

#### **Risk estimation**

It is concluded that Avian alphaviruses are not a hazard in the commodity.

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## **3.12 Flaviviridae**

### **3.12.1 West Nile virus**

#### **3.12.1.1 Hazard identification**

##### **Aetiological agent**

The aetiological agent for West Nile virus (WNV) is a Flavivirus in the Flaviviridae family. It is an arthropod borne virus.

##### **OIE List**

WNV is not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

WNV is not listed in the unwanted organisms register.  
WNV is not recognised in New Zealand.

##### **Epidemiology**

WNV is an arthropod borne virus particularly infecting wet-land birds and transmitted by mosquitoes, especially *Culex* spp. but also species from other genera. Ticks (especially species that feed on birds) have also been found to be infected. The disease was first recognised in Uganda in 1937 and the virus was isolated in Egypt in 1950. At that time the distribution covered much of Africa, the middle east and western areas of Asia, including the western areas of India, and there were occasional incursions into southern Europe (1).

WNV has been identified in a number of mammalian species including humans in which the virus can cause mortality (1). WNV has also been found in hippoboscids on infected birds (2). Reports on the duration of survival of the virus in the flies and the role of the flies as vectors have not been discovered. Disease outbreaks in horses have been reported from Italy in 1998 (3) and France in 2000 (4). Lesser effects on horses have been reported from other locations. Since 1960, diseases due to WNV have become more common and more severe in southern Europe and around the Mediterranean basin. Disease incidents, particularly affecting horses, with a number of deaths, have occurred in Israel, Italy, Morocco, and France since 1996. In 1998, WNV caused significant mortalities in migrating birds in Israel (5).

WNV became evident in the United States in 1999 and has continued to spread as a serious epidemic in humans with 9862 clinical cases and 264 deaths diagnosed during 2003 (6). This epidemic has been marked by significant mortalities in birds, especially American Crows (*Corvus brachyrhynchos*) (7, 8). Domestic geese, Canadian geese,

chickens, rock dove and sparrows were amongst other avian species showing serological evidence of infection (9).

Although the rapidity of spread and the numbers of deaths in both humans and birds in the US epidemic differ from the behaviour of WNV usually seen in Eurasia and Africa, Giladi *et al.* (10) found a very high degree of homology between strains originating from Israel during the 1997 –1998 epidemic, which involved high mortality rate in birds, and virus from the New York epidemic in 1999. This homology was considered supportive of the hypothesis advanced earlier, and based on epidemiological similarities and genomic sequencing (11, 12), that the New York incursion had originated from Israel.

Serological evidence of WNV has been reported from a large number of bird species, including several members of the passeriformes family, in Great Britain (13). Although the virus was not isolated, WNV-specific RNA was identified in sera and brains from a small number of tissues tested. The authors proposed that WNV was introduced to UK resident birds by migrant birds. The finding of antibodies in juvenile birds indicated that virus was being actively transmitted between birds within the UK resident bird population. Low mosquito population density, viral strain differences and “herd immunity” within the UK bird population were suggested as possible reasons why disease associated with WNV has not been observed in Great Britain.

## **Conclusion**

WNV is considered to be a potential hazard in the commodity.

### **3.12.1.2 Risk assessment**

#### **Release assessment**

Neither reports of vertical transmission of WNV in birds, nor suggestions that such transmission might play a part in the epidemiology of the organism, have been located.

The likelihood of such transmission is negligible.

#### **Risk estimation**

Based on the negligible release assessment it is concluded that WNV is not a hazard in the commodity.

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## **3.12.2 Japanese encephalitis virus**

### **3.12.2.1 Hazard identification**

#### **Aetiological agent**

The aetiological agent for Japanese encephalitis (JE) is a Flavivirus in the Flaviviridae family. It is an arthropod-borne virus.

#### **OIE List**

JE is included in the OIE list of notifiable diseases.

#### **New Zealand Status**

JE is listed as notifiable on the unwanted organisms register.

JE is not present in New Zealand.

#### **Epidemiology**

JE is an important zoonosis, also affecting horses and, to a lesser extent, pigs. Herons and egrets are recognised as carrying the virus and acting as reservoirs for the virus. *Culex* mosquitoes play a major role in virus transmission (1). JE virus, or antibodies to it, have been identified in a number of passeriformes species (2, 3, 4).

JE is endemic in much of Asia, particularly southeast Asia (5)

No reports suggesting vertical transmission of JE virus in birds have been located.

#### **Conclusion**

Based on the restricted geographic distribution and the lack of evidence of vertical transmission in birds JE is not considered to be a potential hazard in the commodity.

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### **3.12.3 Louping Ill**

#### **3.12.3.1 Hazard identification**

##### **Aetiological agent**

The Louping Ill is caused by a member of the Flavivirus group within the family Flaviviridae.

##### **OIE List**

Louping Ill virus is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Louping Ill virus is listed on the unwanted organisms register. The disease does not occur in NZ.

##### **Epidemiology**

Louping Ill is a disease of sheep (with occasional infections of humans) which is transmitted by the tick *Ixodes ricinus*. The virus does infect other species including cattle, horses and deer (1). Red grouse are commonly affected with high mortality rates (2, 3). Other species of grouse can also be infected (4, 5).

Searches of the literature failed to find reports of Louping Ill virus infection in bird species other than grouse.

##### **Conclusion**

Based on the host preference of Louping Ill virus, it is not considered to be a potential hazard in the commodity.

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### **3.12.4 Other flaviviruses**

#### **3.12.4.1 Hazard identification**

##### **Aetiological agent**

Birds are known to be infected by a number of Flaviviruses in the family Flaviviridae.

##### **OIE List**

Avian flaviviruses are not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Not listed in the unwanted organisms register.

Not recognised in New Zealand.

##### **Epidemiology**

Literature searches have identified the following Flaviviruses as infecting birds

Dengue virus – Recorded as infecting ducks and poultry. The only presence of Dengue virus in Europe is in travellers returning from infected areas of the world and carrying the infection.

Edge Hill virus and Kokobera virus – Recorded as infecting poultry. All reports of these viruses are from Australia.

Kyasanur Forest virus – Reported as infecting doves in China and birds in India. Based on reports in the literature, the distribution of this virus is limited and well distanced from Europe.

Meaban virus and Tyuleniy virus – Infect sea birds around France and have also been reported from Wales.

Saboya virus – Recorded as infecting birds in Guinea. The distribution of this virus appears to be restricted to Africa.

Wesselsbron virus – Recorded as infecting Ostriches. This virus has a range restricted to southern Africa.

Iguape virus – Birds are thought to play a role in the transmission of this virus. The only reports of this virus are from Brazil.

Usutu virus – Until 2001 this virus was thought to be restricted to Africa. In that year there were a considerable number of bird deaths in Austria. The mortality rate

appeared highest in Eurasian Blackbirds (*Turdus merula*) around Vienna. Deaths also occurred in Barn Swallows (*Hirundo rustica*) 200 km west of Vienna and in Barn Owls in a zoo. Both of the first two species are in the Passeriformes family. Usutu virus was diagnosed as the cause of these deaths and it was postulated that the virus may have been introduced to Europe by migrating swallows (1). Subsequently, evidence of Usutu virus has been reported from birds, including several members of the Passeriformes family, in the United Kingdom. The authors of this report concluded that, while Usutu virus was probably introduced to the UK by migrating birds, it appeared to be being transferred between birds within the country (2).

## **Conclusion**

Flaviviruses are considered to be potential hazards in the commodity.

### **3.12.4.2 Risk assessment**

#### **Release assessment**

No reports suggesting a transmission route for flaviviruses through avian eggs have been located. On that basis, the likelihood of importation of flaviviruses through passerine eggs is negligible.

Flaviviruses listed in this section, other than Usutu virus can also be excluded from being likely to be in imported passerine eggs on the basis of their geographic distribution, the species of birds infected and/or the absence of reports of pathogenicity.

The release assessment for flaviviruses in passerine eggs imported from Europe is negligible.

#### **Risk estimation**

Based on the negligible release assessment, it is concluded that flaviviruses are not a hazard in the commodity.

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## **3.13 Reoviridae**

### **3.13.1 Rotavirus**

#### **3.13.1.1 Hazard identification**

##### **Aetiological agent**

Rotavirus is a genus within the Reoviridae family. Members of this genus cause diarrhoea in intensively reared animals worldwide (1). Rotaviruses have been differentiated on the basis of a group antigen. The vast majority of both mammalian and avian rotaviruses fall within the conventional group A, whereas Groups B, C and E rotaviruses are found in mammals and groups D, F and G in birds (2).

##### **OIE List**

Avian rotaviruses are not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

Not listed in the unwanted organisms register.

Species from which rotaviruses have been reported in New Zealand include cattle, foals, dogs, cats (3), pigs (4, 5), chickens (6) rabbits (7), deer (8) and humans (9). The common nature of sub-clinical infections of rotaviruses (2) means that rotaviruses may be more widely distributed than reports indicate.

##### **Epidemiology**

Avian rotavirus infection has been described in turkeys, chickens, pheasants, partridges, ducks, guinea fowl, pigeons and lovebirds. Virus is excreted in faeces, contaminates the environment and leads to horizontal transmission. Both infection and disease are most common in young birds. Infection, in the absence of disease, is common. Strain variations in pathogenicity do occur (2). Viral strains are generally, but not exclusively, host specific (2). The common nature of sub-clinical infections of rotaviruses (2) means that rotaviruses may be more widely distributed than reports indicate.

Egg transmission of rotavirus in turkeys was postulated on the basis of detection of infection in three-day old poults (10). Supporting evidence has not been forthcoming in the 18 years since that report and the development of clinical rotaviral infections in calves in the very early days of life (11) suggests that eggs transmission is not required as an explanation for the early development of disease in chickens.

Searches of the literature have not identified any reports of rotavirus in passeriformes.



## **Conclusion**

Rotaviruses are considered to be potential hazards in the commodity.

### **3.13.1.2 Risk assessment**

#### **Release assessment**

The lack of reports of rotavirus in passerine birds does not exclude the likelihood that sub-clinical infections may occur, however, there is no evidence that avian rotavirus infection is transmitted through eggs.

The combined lack of reports of rotaviruses in passerine birds and the lack of evidence that rotaviruses are transmitted through eggs means that the likelihood of infection arising from the importation of passerine eggs is negligible.

The release assessment for rotaviruses in passerine eggs imported from Europe is negligible.

#### **Risk estimation**

Based on the negligible release assessment, rotaviruses are not considered a hazard in the commodity.

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### **3.13.2 Orbivirus**

#### **3.13.2.1 Hazard identification**

##### **Aetiological agent**

Orbiviruses are arthropod borne viruses in the family Reoviridae. This genus includes viruses that cause important diseases of mammalian species (African horse sickness, Bluetongue, Epizootic haemorrhagic disease of deer) but no reports of the viruses causing these diseases being found in birds have been located.

##### **OIE List**

Avian orbiviruses are not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

None of the Orbiviruses known to infect birds are included in the unwanted organisms register.

An Orbivirus has been found in ticks collected from penguins on Macquarie Island (1).

##### **Epidemiology**

Orbiviruses are widespread in sea-birds and their associated tick populations with reports from many locations. Moss *et al.*(2) commented on the distribution of one sub-group as being from the arctic to the sub-Antarctic. Reports do not associate these viruses with mortality or disease but in wild marine environments such associations might not be observed unless the incidents are spectacular.

Hirai *et al.* (3) reported the isolation of viruses classified as Orbiviruses from a cockatiel and a budgerigar in the United States. Experimental infection of chickens, quail and budgerigars with one of these isolates resulted in a period of diarrhoea and faecal shedding of the organism in budgerigars. The authors considered that the viruses were only mildly pathogenic in the budgerigars.

No reports suggesting egg borne transmission of Orbiviruses in birds have been located.

No reports of Orbiviruses in passeriformes have been located.

##### **Conclusion**

Based on low pathogenicity in budgerigars, a lack of reports of Orbiviruses in passerine birds and a lack of reports suggesting egg borne transmission, Orbiviruses are not considered to be potential hazards in the commodity.

## References

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### **3.13.3 Other reoviruses**

#### **3.13.3.1 Hazard identification**

##### **Aetiological agent**

Searches of electronic databases for diseases of passerine birds caused by reoviruses other than rotaviruses and orbiviruses were unsuccessful.

Based on this absence of information, Reoviruses are not considered a hazard in the commodity.

## 3.14 Bunyaviridae

### 3.14.1 Nairoviruses

#### 3.14.1.1 Hazard identification

##### Aetiological agent

Nairoviruses are within the family Bunyaviridae which consists, mainly, of arthropod borne organisms. Members of the genus include Nairobi sheep disease virus, Dugbe virus, Sedlec virus and Crimea-Congo haemorrhagic fever virus (CCHFV).

##### OIE List

None of these viruses are included in the OIE list.

##### New Zealand Status

Nairobi sheep disease is listed on the unwanted organisms register as a notifiable organism.

None of these viruses are recognised as being present in New Zealand.

##### Epidemiology

Nairobi sheep disease is a tick transmitted disease affecting sheep and cattle in Africa. Literature searches have not identified records of the virus in birds.

Dugbe virus causes fever in humans and has been reported to be present in cattle, sheep, goats, rodents and birds in parts of Africa (1).

Sedlec virus has been isolated from a number of Reed Warblers (*Acrocephalus scirpaceus*) in the Czech Republic (2, 3, 4). The surveys carried out between 1984 and 1993 included a large number of bird species but this virus was found in samples from only the one species. The virus was found to be pathogenic for mice when inoculated intra-cerebrally but not when inoculated intra-peritoneally. The authors did not relate the virus to any natural disease incident. The main range of Reed warblers is across northern Europe with some populations in southern Europe. This species does have migratory behaviour (5) but information on the extent has not been found.

CCHFV causes Crimea-Congo Haemorrhagic fever in humans. The virus infects a wide range of domestic and wild animals including a small number of bird species. The main vectors for human infection are *Hyalomma* ticks, while a range of genera including *Hyalomma*, *Haemaphysalis*, *Amblyomma* and *Dermacentor* maintain infection in domestic and wild animals (6). The distribution of CCHFV is broad, from eastern Europe to much of Africa and Asia (7), including as far east as Greece and the Balkan peninsula

(8). Bird species in which either CCHFV, or anti-CCHFV antibodies, have been identified include ostriches (*Struthio camelus*) (9), red-beaked hornbills (*Tockus erythrorhynchus*), glossy starlings (*Lamprotornis sp.*) and grey-breasted helmet guinea fowl (*Numida meleagris*) (10). Of these, glossy starlings are members of the passeriformes order.

## **Conclusion**

Based on host range Nairobi sheep disease virus, is not considered to be a potential hazard in the commodity.

Based on geographic distribution, Dugbe virus is not considered to be a potential hazard in the commodity.

Based of lack of pathogenicity, Seldec virus is not considered to be a potential hazard in the commodity.

Based on the geographic distribution of CCHFV virus to include a member state of the EU (Greece) and two prospective member states (Bulgaria and Romania) (11, 12) this virus is considered to be a potential hazard in the commodity.

### **3.14.1.2 Risk assessment**

#### **Release assessment**

The only passerine species in which reports of CCHFV infection has been discovered is glossy starlings (*Lamprotornis sp.*). Reports of infection of passerine birds with other Nairoviruses are restricted to infection of Reed Warblers (*Acrocephalus scirpaceus*) with Sedlec virus. From this information it seems that the host range of Nairoviruses is restricted. It seems likely that the passerine host range of CCHFV is restricted to one, or a very small number of, species. No reports suggesting vertical transmission of Nairoviruses in birds have been located.

On the basis of the above evidence, the likelihood that Nairoviruses might be carried in passerine eggs imported from Europe is negligible.

The release assessment for Nairoviruses in passerine eggs imported from Europe is negligible.

#### **Risk estimation**

Based on the negligible release assessment, it is concluded that Nairoviruses are not a hazard in the commodity.

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### **3.14.2 Other Bunyaviruses**

#### **3.14.2.1 Hazard identification**

##### **Aetiological agent**

Other Bunyaviridae for which evidence of infection in birds has been reported (virus identification or positive serology) are Issyk-Kul virus from Russia / Tajikistan (1), Tahyna virus and Batai (or Calovo) virus from Poland (2) and the Czech Republic (3).

##### **OIE List**

Avian Bunyaviruses are not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

These organisms are not listed in the unwanted organisms register, nor have they been identified in NZ.

##### **Epidemiology**

Issyk-Kul virus is an arbovirus which has been identified as causing disease in humans and infecting seven species of birds in south Tajikistan (1). It has also been shown, experimentally, to be capable of infecting other mammalian species (4). The source of reports of Issyk-Kul virus is limited to the area of Tajikistan.

Tahyna virus is an arbovirus recognised as causing disease in humans (1). The distribution of the virus is restricted to Europe and reports of its findings in surveys have been summarized by Gratz (5). Positive survey findings come from the Russian Federation, the Czech Republic, Croatia, Romania, Austria, Serbia, Slovakia, Hungary, France, Germany and Spain. Species infected include humans, cattle, sheep and a number of other mammals. The majority of findings in mosquitoes have been in *Aedes* spp. Infected birds have included cormorants, ducks, swallows, martins, and sparrows.

Batai (or Calovo) virus is an arbovirus, the presence of which has been reported throughout Scandinavia and in northern Russia. It is present in eastern and central Europe and has also been found in Portugal. The same virus (or similar) has been identified in parts of Asia and Africa. Virus has been recovered from *Anopheles* and *Culex* spp. of mosquitoes (6). No reports have been found linking Batai virus to disease in humans or other species. Antibodies to Batai (Calovo) virus were found in house sparrows (*Passer domesticus*) (2, 3) and tree sparrows (*Passer montanus*) (2), both of which are passerine species, in central Europe. Birds included in surveys were not selected on the basis of ill health. No evidence is presented linking Batai virus to disease in birds.

## **Conclusion**

Issyk-Kul virus and Tahyna virus are considered to be potential hazards in the commodity.

Based on lack of evidence that Batai virus causes disease it is not considered to be a potential hazard in the commodity.

### **3.14.2.2 Risk assessment**

#### **Release assessment**

Reports of Issyk-Kul virus come only from the region of Tajikistan. The likelihood of infection in birds from areas covered by this commodity definition is negligible. The release assessment for Issyk-Kul virus is negligible.

Tahyna virus has been reported from birds, including passerine species, in large areas of Europe including Germany and Portugal.

No reports suggesting vertical transmission of Bunyaviruses have been located.

On the basis of Bunyaviruses being transmitted through insects and the lack of any suggestion that these viruses might be transmitted through birds eggs, the likelihood of the entry of these viruses to New Zealand through the importation of passerine eggs is negligible.

The release assessment for Bunyaviruses in passerine eggs imported from Europe is negligible.

#### **Risk estimation**

It is considered that Bunyaviruses are not a hazard in the commodity.

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## **3.15 Bornaviridae**

### **3.15.1 Borna virus**

#### **3.15.1.1 Hazard identification**

##### **Aetiological agent**

Bornavirus is the sole member of the family Bornaviridae. It is a spherical, enveloped RNA virus.

##### **OIE List**

Borna disease virus is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Borna disease virus (BDV) is listed in the unwanted organisms register.

A virus antigenically related to Borna virus (1) and sharing a number of physical characteristics (2) causes a fatal neuropathological disease (wobbly possum syndrome) of possums (*Trichosurus vulpecula*) in New Zealand (3). Testing using PCR technology suggested that the virus is Borna-like, rather than Borna virus (Personal communication. J. O'Keefe. Phone call 13 May 2005) but information on characterisation of the virus is not available. For the purposes of this risk analysis it is assumed that this virus is distinct from Borna virus and that Borna virus is not present in New Zealand.

##### **Epidemiology**

Reviews of Borna Disease have been provided by Hatalski *et al.* (1997) (4), Richt *et al.* (1991) (5), Staeheli *et al.* (2000) (6) and Richt *et al.* (2001) (7). Borna disease virus is recognised as a cause of disease of horses and sheep (in parts of central Europe) and infection has also been found in donkeys, goats, sheep, llamas, ostriches, cats, rabbits, llamas, pigmy hippopotamus, sloth, vari monkeys, cattle and ostriches. Associations between BDV and neuropsychiatric disorders in humans have been proposed although this is a topic of some debate. The full geographic distribution of the virus is not certain although natural infections have been reported in northern and central Europe, North America, and parts of Asia. Reports from western Europe include, as host species, horses, foxes, cattle, dogs, sheep and roe deer in France (8) and cats in the United Kingdom (9). Beyond those species reported to have been naturally infected with BDV, the virus has been transmitted experimentally to a number of species including chickens, pigeons, rodents and non-human primates. Hatalski *et al.* (4) commented, on the basis of the wide range of species known to be infected naturally and the wider range that had been infected experimentally, "the host range is likely to include all warm-blooded animals". Disease in infected animals appears related to both the infected species and the infecting

viral strain. The mechanisms of spread are not understood but it has been suggested that infection may take place through inhalation. Insect vectors have also been proposed but the virus has not been identified in species other than “warm blooded”.

In birds, natural infections with BDV occur in young ostriches and the mortality rate can be very high (10). Evidence of viral infection (based on PCR technology) with distinct strains of BDV has also been reported from Mallard ducks (*Anas platyrhynchos*) and Jackdaws (*Corvus monedula*) in Sweden (11) and the authors proposed that these findings might be evidence for birds, including Jackdaws (passerine species), serving a role as reservoirs for BDV. Other reports of evidence of BDV in passerines have not been located.

## **Conclusion**

It is considered that BDV is a potential hazard in the commodity.

### **3.15.1.2 Risk assessment**

#### **Release assessment**

Based on there being only one report of possible BDV infection in a passerine species and the lack of discovery of any suggestion that BDV might be transmitted through the eggs of birds, the likelihood that BDV might be imported in passerine eggs from Europe is negligible.

The release assessment for BDV in passerine eggs is negligible.

#### **Risk estimation**

Based on the negligible release assessment, it is concluded that Borna Disease Virus is not a hazard in the commodity.

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## **3.16 Picornaviridae**

### **3.16.1 Avian encephalomyelitis**

#### **3.16.1.1 Hazard identification**

##### **Aetiological agent**

Avian encephalomyelitis (AE) virus is a picornavirus, tentatively placed in the *Hepatovirus* genus (1).

##### **OIE List**

AE is not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

AE is not included in the unwanted organisms register.

AE was recognised in New Zealand prior to 1972 when satisfactory use of a vaccine was reported (2). Further confirmation of its presence comes from McCausland (3) and Howell and Bell (4). Vaccination contributes to a high percentage of chickens being serologically positive (5).

##### **Epidemiology**

AE is widely distributed in chickens around the world. Serological evidence also suggests that turkeys are commonly infected (1).

Strain variations relate to laboratory adapted strains and strains selected for use in vaccines.

AE is vertically transmitted through poultry eggs (1). No reports of AE in passeriformes species have been located.

##### **Conclusion**

It is considered that AE is not a potential hazard in the commodity.

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### **3.16.2 Duck hepatitis (DHV 1 & 3)**

#### **3.16.2.1 Hazard identification**

##### **Aetiological agent**

Duck hepatitis 1 and 3 are caused by different picornaviruses which share no common antigens in virus neutralization or fluorescent antibody tests.

##### **OIE List**

Duck virus hepatitis is included in the OIE list.

##### **New Zealand Status**

Duck hepatitis virus is listed in the unwanted organisms register. Neither DHV 1 nor 3 have been diagnosed in New Zealand.

##### **Epidemiology**

Both DHV 1 and 3 affect only young ducklings in natural infections.

DHV 3 was recognised in ducklings on Long Island, New York in the 1960s (1) and does not appear to have been diagnosed from field outbreaks since.

Chickens and turkeys do not appear to be susceptible to field infections with DHV 1 although there are reports of successful experimental infections (1).

There are no reports of infections of passerine species with DHV 1 or 3, nor are there reports of natural infections of species other than ducks.

##### **Conclusion**

Based on host specificity and the lack of reports of evidence of the virus in passerine species, neither DHV 1 nor 3 are considered to be potential hazards in the commodity.

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## **3.17 Astroviridae**

### **3.17.1 Astroviruses - duck hepatitis type 2 (DVH 2), turkey astrovirus 1 and 2, avian nephritis virus.**

#### **3.17.1.1 Hazard identification**

##### **Aetiological agent**

Astroviruses are single stranded RNA viruses (1).

##### **OIE List**

Duck hepatitis is included on the OIE list. Other organisms addressed in this section are not listed by OIE.

##### **New Zealand Status**

Duck hepatitis type 2 and turkey astrovirus are listed in the unwanted organisms register.

No avian Astroviruses have been identified in New Zealand although Howell (2) reported that antibodies to avian nephritis virus had been identified in pooled flock sera tested overseas and that renal pathology consistent with the disease had been seen.

##### **Epidemiology**

A number of agents previously named as picornavirus, picornavirus-like, Enterovirus or Enterovirus-like are now classified, or thought likely to become classified, as Astroviruses (3). Amongst these are avian nephritis virus (4) and duck hepatitis virus (5,6, 7) (both previously classified as picornaviruses) and turkey Enterovirus-like agent (8).

Astroviruses isolated from different species are antigenically distinct and are species specific (1). Although Gough *et al.* (6) commented on an association of outbreaks of duck hepatitis type 2 with contact with wild birds there have been no reports of Astroviruses in wild bird populations to support such a causal link.

Astroviruses were found in 9 of 22 (41%) clinically normal turkey flocks in the USA while 37 of 43 (86%) flock with signs of enteritis were found to be infected (9). In both England (10) and Ireland (11), clinically normal chicken flocks, including specific pathogen free flocks, and some turkey flocks were found to have serological evidence of avian nephritis virus (10). Positive serology was not found in ducks. These findings are consistent with the distribution of Astroviruses within their relevant host species being much wider than the distribution of disease. The possibility of distribution through contaminated poultry vaccines was raised but not supported by serological results following vaccination of groups of chickens with 23 batches of different commercial

vaccines (1). Passage of virus in faeces is thought to be the main means of spread (1) although an enterovirus-like agent has been reported from the meconium of dead-in-shell chicken embryos (11) suggesting that vertical transmission through eggs may occur with that virus.

## **Conclusion**

Astroviruses are considered to be a potential hazard in the commodity.

### **3.17.1.2 Risk assessment**

#### **Release assessment**

No reports of Astroviruses in passeriformes have been located. Either diseases caused by Astroviruses in passeriformes are sufficiently uncommon to have avoided detection, Astroviruses in wild or aviary birds do not cause disease or Astroviruses have not established a host relationship with passeriformes species.

Based on the absence of reports of astroviruses in a passerine species, and the apparent host specificity of astroviruses, the likelihood that astroviruses might be imported in passerine eggs from Europe is negligible.

The release assessment for BDV in passerine eggs is negligible.

#### **Risk estimation**

Based on negligible release assessment, it is concluded that Astroviruses are not a hazard in the commodity.

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## **3.18 Hepadnaviridae**

### **3.18.1 Hepadnavirus (duck virus hepatitis)**

#### **3.18.1.1 Hazard identification**

##### **Aetiological agent**

Hepadnaviruses are DNA viruses of which the human Hepatitis B virus is a member.

##### **OIE List**

Duck virus hepatitis is included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Duck virus hepatitis is listed in the register of unwanted organisms. This organism has not been identified in New Zealand.

##### **Epidemiology**

Avian Hepadnaviruses are relatively host specific. While hepatitis B-like virus in birds was first recognised in Peking ducks (1), strains have now been recognised in grey herons (*Ardea cinerea*) (2), snow geese (*Anser caerulescens*) (3) and white storks (*Ciconia ciconia*) (4). Prassolov *et al.* (5) reported a hepatitis B virus from cranes with a broader host range but still restricted to members of the order Anseriformes.

No reports have been located of Hepadnaviruses in passeriformes. No reports of Hepadnaviruses in birds other than Anseriformes have been located.

##### **Conclusion**

Based on host specificity and the absence of reports of Hepadnaviruses in passerine birds, it is considered that these organisms are not potential hazards in the commodity.

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## **3.19 Retroviridae**

### **3.19.1 Avian leucosis-sarcoma group.**

#### **3.19.1.1 Hazard identification**

##### **Aetiological agent**

The avian leucosis-sarcoma group of viruses (ALSVs) are within the Genus Alpharetrovirus (1). Taxonomically the group includes Avian leucosis virus, Rous sarcoma virus, Avian carcinoma Mill Hill virus, avian myeloblastosis virus, Avian myelocytomatosis virus and Avian sarcoma virus (2).

Endogenous avian retroviruses of the ALSV group, incorporated into the host genome, are recognised in at least 26 species from three families and 14 genera of the Galliformes Order (3). These viruses are not recognised as having either pathogenic or developmental effects (4) and are not given further consideration in this RA. Consideration of their phylogeny, along with that of their hosts, does, however, indicate longstanding relationships through the evolutionary history of both hosts and retroviruses (3, 4).

##### **OIE List**

Avian leucosis is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Not listed in the unwanted organisms register.

Avian leucosis is endemic in New Zealand poultry (5, 6). Information on subgroup identification of ALSVs in New Zealand has not been discovered except that Group J, which is recognised as causing significant losses in meat chickens (1) was diagnosed in New Zealand in 1996 (7).

Vickers (8) reported investigations of psittacine erythroblastosis in New Zealand parakeets. Testing of these birds for ALSV provided negative results. “Avian leucosis” has been reported from a mallard duck (9) and a haemopoietic neoplasm resembling myeloblastosis has been reported from an incident of unthriftiness and deaths in several budgerigars, the progeny of imported parents (10). Although this latter report stated that the disease was caused by avian leucosis virus no evidence of confirmation of the aetiology was provided. Lymphoid tumours, one of which was described as “similar to lymphoid leucosis in poultry”, have been described from two ostriches and one emu (11).

##### **Epidemiology**

All recognised Alpharetroviruses have birds as their hosts. They cause a range of transmissible neoplasms, some of which are malignant.(1) Serotyping of the ALSVs



places them into subgroups A to J with A, B, C, D, E and J infecting chickens and F, G, H and I infecting pheasants, partridge and quail (1).

Searches of electronic data bases and of available relevant texts have identified one incident of multiple cases of lymphoid leucosis in passerines. That was in canaries (*Serinus canarius*) in Brasil (12).

A number of case reports of passerine birds with pathology similar to those associated with the avian leucosis-sarcoma group of viruses in poultry were located. These were

- Two cases of lymphosarcoma in canaries (*Serinus canarius*) (13, 14),
- a fibroma in a canary (*Serinus* spp.) (15) and
- a pancreatic adenocarcinoma in a mynah (*Sturnidae*) (16),
- an hepatic adenocarcinoma in a crow (*Corvus splendens splendens*) (17),
- a squamous cell carcinoma in a rook (*Corvus frugilegus*) (18),
- a multicentric lymphoma in a European starling (*Sturnus vulgaris*) (19) and
- lymphoid leukosis in a white-throated jay thrush (*Garrulax albogularis*) (20) and a finch (20)
- myeloid leucosis in a red cheeked Cordonbleu (*Uraeginthus begalus*) (21) and
- stem-cell leukosis in a canary (*Serinus canarius*) (20).

Varejka and Tomsik ( 22) identified Rous sarcoma virus-neutralising activity in one pool of sera from three sparrows captured at a poultry farm near Brno. In total, sera from 41 sparrows were tested in pools of three to five samples. The authors suggested that one young bird may have contributed the neutralising activity. The neutralising activity was low and the result was interpreted with caution.

The report by Martins et al (12) must be considered suggestive of causation by an ALSV type organism. Whether the report by Varejka and Tomisk (22) is evidence of infection of passerines with an ALSV is uncertain. The sporadic nature of the diseases referred to in all other reports of ALSV-like pathology could be consistent with a non-contagious aetiology or with aetiology by a contagious agent that rarely manifests itself in disease.

## Conclusion

Whether a retrovirus (or retroviruses) of the leucosis-sarcoma group infect passerine birds and cause disease is unknown. If such infection does occur, disease occurrence is uncommon and the only report of multiple cases of disease was in Brasil.

It is concluded that retroviruses of the leucosis-sarcoma group are not a potential hazard in the commodity.

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### **3.19.2 Lymphoproliferative disease virus**

#### **3.19.2.1 Hazard identification**

##### **Aetiological agent**

This term is used to refer to a virus within ALSV group but is not accepted as a valid name by the International Committee on Taxonomy of Viruses (1)

##### **OIE List**

Lymphoproliferative disease is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Lymphoproliferative disease is listed in the unwanted organisms register. This disease has not been diagnosed in NZ

##### **Epidemiology**

Lymphoproliferative disease is a rare disease of turkeys in Europe and Israel (2)  
The only reference located referring to “lymphoproliferative disease” in passerine species is in the section of Diseases of Cage and Aviary Birds dealing with neoplasms (3, page 486). While the articles that form the basis of this reference are not readily available, their titles refer to lymphosarcoma; a pathological (rather than aetiological) condition that bears no specific relationship to the viral condition of Lymphoproliferative disease.

##### **Conclusion**

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

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### **3.19.3 Avian reticuloendotheliosis virus group**

#### **3.19.3.1 Hazard identification**

##### **Aetiological agent**

This virus group within the genus Gammaretrovirus includes chick syncytial virus, reticuloendotheliosis virus and Trager duck spleen necrosis virus (1). The Gammaretroviruses include viruses infecting mice (Murine leukaemia virus), cats, Gibbon apes, guinea pigs, reptiles and other vertebrate species. Martin et al. (2) were successful in extracting genomic DNA of murine leukaemia virus-related retroviruses from 23 of approximately 100 vertebrate taxa. Virus DNA was not identified from 30 species of fish and other basal chordates. Ten different DNA sequences were obtained from eight species of birds, including four passerines (Wren, Redwing, Bowerbird and Rook). Phylogenetic analysis of the viral DNA showed a close parallel with the phylogeny of the host species suggesting that host/virus relationships are long established and that much of the retroviral transmission is vertical.

##### **OIE List**

REV is not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

REV is not listed in the unwanted organisms register.

Positive REV serology was reported by Howell et al (3) to be relatively widespread in chickens. Whether disease is associated with this virus is uncertain (4).

##### **Epidemiology**

The main route for infection of birds with REV appears to be transovarial vertical transmission. Horizontal transmission appears to play some role in the epidemiology of the organism and this may be assisted by mechanical transfer by insects (5). Witter and Faldy (5) identified chickens, turkeys, ducks, geese, pheasants, Japanese quail, peafowl and prairie chickens as species recognised as being infected naturally, however the host range of Gamma retroviruses has been extended by the work of Martin et al.(2) and it seems probable that REV-like Gammaretroviruses infect many species of terrestrial vertebrates, including passerine species. Disease associated with REV uncommon except in situations where poultry have been vaccinated with REV-contaminated vaccines (5).

REV causes a range of pathological syndromes including runting disease syndrome, chicken bursal lymphoma, chicken non-bursal lymphoma, turkey lymphoma, lymphoma of other species and acute reticulum cell neoplasia (5).

## Conclusion

Based on the past history of the importation of large numbers of passerine birds from Europe and Australia without reference to their retrovirus status, it is likely that retroviruses are well established in New Zealand. The absence of reports, both from New Zealand and internationally, of pathology in passerine species suggestive of REV infection is consistent with the viruses having little, if any, pathological effect in this avian order.

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

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### **3.19.4 Other retroviruses**

#### **3.19.4.1 Hazard identification**

##### **Aetiological agent**

Searches of the scientific literature for retroviruses in passeriformes yielded no references.

##### **Conclusion**

Retroviruses are not a hazard in the importation of passerine species from Europe.

## 3.20 Chlamydophila

### 3.20.1 Chlamydophila spp. (ornithosis)

#### 3.20.1.1 Hazard identification

##### Aetiological agent

The aetiological agent for avian chlamydiosis (psittacosis, ornithosis) is *Chlamydophila psittaci*. This follows reclassification of chlamydial organisms in line with the recommendations of Everett *et al.* (1). These organisms are small obligate intracellular bacteria. Eight serovars, distinguished using monoclonal antibodies and with differences in their predominant host ranges, are recognised (1).

##### OIE List

Avian Chlamydiosis is included on the OIE list.

##### New Zealand Status

Avian chlamydiosis is not listed in the unwanted organisms register.

Chlamydiosis is endemic in New Zealand birds with reports of infection in diseased psittacine birds soon after importation (2) and, later, in diseased resident caged exotic and native psittacine species and pigeons (3, 4). Serological (CFT), cultural (Cloacal swabs and tissues) and antigen detection testing (ELISA) of 54 clinically normal feral pigeons from three distant sites revealed infection at all sites (5). Infection was found in healthy New Zealand Keas shortly after importation into the United Kingdom (6). Following a diagnosis of psittacosis in an adult Takahe (*Porphyrio mantelli*) on Mana Island evidence of infection (positive antigen detection EIA tests results) was found in 73 of 121 faecal samples from captive and wild endangered and threatened native birds. In a follow-up investigation on Kapiti Island, using serological (CFT), cultural (cloacal swabs), and antigen detection testing (ELISA) on 62 native psittacines (some captive and some wild), only inconclusive evidence of chlamydial infection was in two Kaka and three Weka from Kapiti Island (5).

*Chlamydophila psittaci* infection is endemic in psittacine and pigeon populations in New Zealand. The infection status of other native birds is unclear.

##### Epidemiology

The documented avian host range of *Chlamydophila* spp. has been reviewed by Kaleta and Taday (7). Reports from 469 avian species were identified in their review. Sources included nine species of poultry and 460 species of wild and pet bird. Chlamydiosis was recorded from 45% of 342 Psittaciformes species (parrots), 28% of 92 Lariformes (gulls



and terns), 21% of 157 Anatiformes (waterfowl), varying percentages of species in 26 other orders and 2% of the approximately 4000 recognised species in the order Passeriformes. Although Chlamydiosis was reported from only 2% of passerine species, that constituted 89 affected species from four sub-orders and 30 families. The authors recognised that the extent of reporting is influenced by the extent to which birds within species and orders are subject to investigation.

Six serovars (A to E) of *Chlamydophila psittaci* are recognised as infecting birds. Five of these have a wide geographic distribution while F is represented by only one isolate (8). There is strong host specificity in the infection of different orders of birds by the various serovars. A has been identified as most commonly infecting Psittaciformes, B most commonly infects pigeons and doves (Columbiformes), C – waterfowl (Anseriformes), D – turkeys (Phasianiformes) and E – pigeons and ratites (8, 9, 10, 11, 12). Genotyping of isolates of serovars A and B has shown close correlation between the results from the two methods of characterization (13).

Although the relationships between *Chlamydophila psittaci* serovars and avian order are strong these relationships are not exclusive.

- Reports of psittacine infections serotyped other than type A are uncommon, and other than A or B rare (A budgerigar infection typed as serotype C (9)).
- Reports of Columbiformes infections serotyped as other than A or B have been rare ( One isolated types as group E (11).

All reports of typing of passerine infections have been either type A or B (9, 10, 11)

Avian chlamydiosis is a zoonosis with serovars C and D presenting particular hazards to people working with ducks or turkeys or in poultry processing plants. Other serovars may infect humans (12).

Given the patterns of host preference of *C. psittaci* serotypes, the evidence of widespread infections of pigeons and psittacines in New Zealand is consistent with the presence of serotypes A (Psittaciformes) and B (Columbiformes) in the NZ avian population.

## **Conclusion**

Based on the evidence that they are present in New Zealand, it is considered that *C. psittaci* serotypes A and B are not hazards in the commodity.

It is considered that Serovars C, D, E, and F of *Chlamydophila psittaci* are potential hazards in the commodity.

### 3.20.1.2 Risk assessment

#### Release assessment

In experimental studies by Davis et al. (14) turkey hens were inoculated with *C. psittaci* and dead-in-shell embryos checked for presence of the organism with negative results. They also found that, following inoculation of turkey eggs with *C. psittaci*, the organism could not be recovered from eggs beyond the ninth day of incubation. Most authors referring to the topic suggest that transovarial transmission of *C. psittaci* in birds is uncommon (15, 16, 17) but evidence, published by others, that transmission by this route could occur in chickens, ducks, parakeets, sea gulls and snow geese was accepted by Vanrompay et al. (16) and by the Scientific Committee on Animal Health and Animal Welfare of the European Commission (18) in their reviews.

All *Chlamydophila psittaci* isolates from passerines that have been subject to serotyping have been either type A or type B or untypable. Andersen *et al.* (10) included four canary and one gouldian finch isolates in their study and found two of them to be serovar A and three serovar B. Analysis of the data presented by Sudler *et al.* (12) (para. 1, page 240) indicates that either the two strains from passerines included in their study were serovar A or one was serovar A and the other was untypable.

Given

- the pattern of infection of birds within individual orders of birds being infected with particular serovars and the finding that most, if not all, isolates from passerine species are of types A or B and
- the widely stated view that egg borne transmission is uncommon

the likelihood of imported passerine eggs being infected with *Chlamydophila psittaci* serovars C, D, E or F is negligible.

The release assessment for *Chlamydophila psittaci* serovars C, D, E or F is negligible.

#### Risk estimation

Based on the negative release assessment it is concluded that *Chlamydophila psittaci* is not a hazard in the commodity.

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### 3.21 Bacteria associated with enteric and generalized infection in birds

#### 3.21.1 Salmonellae - general

##### 3.21.1.1 Hazard identification

###### Aetiological agent

As members of the Enterobacteriaceae, Salmonellae are motile Gram-negative rods that ferment glucose and other sugars and are oxidase negative.

The *Salmonella* genus contains over 2,400 serotypes. Nomenclature now places most Salmonellae of veterinary relevance in the sub-species *Salmonella enterica* subspecies *enterica*. Over 2,300 serotypes fall within this subspecies. The commonly used names (e.g. *Salmonella* Typhimurium) identify serotypes within the *Salmonella enterica enterica* sub-species. Some of these serotypes are further partitioned on the basis of phage type. Most salmonella species are considered to be relatively non-host specific (1). Nomenclature of *Arizona* spp. or *Salmonella arizonae* has changed over the years but *Salmonella enterica arizonae* and *Salmonella enterica diarizonae* are now considered subspecies within *Salmonella enterica*. *Salmonella enterica arizonae* contains over 300 serotypes (2).

The Salmonellae of major interest in this risk analysis are those designated as unwanted organisms (*S. Gallinarum*, *S. Pullorum*, *S. Abortusovis*, *S. arizonae*, *S. Dublin*, *S. Typhimurium* DT 104, *S. Typhimurium* DT 44, *S. Enteritidis* pt 4 and *Salmonella* spp. (exotic, affecting animals)

###### OIE List

*Salmonella* serotypes other than *S. Gallinarum*-*Pullorum* are not included in the OIE list of notifiable diseases.

### **3.21.2 *Salmonella enterica* subsp. *enterica* serovar Gallinarum-Pullorum**

#### **3.21.2.1 Hazard identification**

##### **Aetiological agent**

This name now covers the organisms previously known as *Salmonella* Gallinarum and *Salmonella* Pullorum. This is a highly host adapted, non-motile salmonella in sero-group D (1). Because of changes in nomenclature and because of the existence of chick and turkey host-adapted strains, the literature is dominated by references to *S. Gallinarum* and *S. Pullorum*.

##### **New Zealand Status**

Both *S. Gallinarum* and *S. Pullorum* are listed in the unwanted organisms register.

*S. Gallinarum* has not been diagnosed in NZ and *S. Pullorum* was last diagnosed in 1985 following an extensive eradication programme operated within the commercial poultry industries.

##### **Epidemiology**

The natural host for *Salmonella enterica* subsp. *enterica* serovar Gallinarum-Pullorum (*S. Gallinarum*-Pullorum) is chickens. The organism occurs in most countries. In episodes of infection within flocks both morbidity and mortality can be highly variable and the age group most affected depends upon the pattern of infection within the flock. Transovarian infection does take place and resulting chicks may die in incubators. Clinical signs in adult birds may vary from none to severe with high mortality. Disease outbreaks have been reported from turkeys and a small number of other species. Transmission can occur both horizontally and vertically with carrier birds playing an important role in spreading the disease (1).

There are few reports of the isolation of *S. Gallinarum*-Pullorum from wild birds or from caged or aviary birds. Truche (2), in 1923, stated that a number of species, including sparrows, were susceptible to infection. The only other report of recovery of *S. Gallinarum*-Pullorum from passerine birds is the unreferenced comment by Snoeyenbos (3) that *S. Pullorum* had been identified in sparrows, canaries and European bullfinches. The origins of these reports have not been located. Literature searches have identified a large number of investigations of salmonella infections in wild, caged and aviary birds, including sampling of birds in the vicinity of poultry flocks infected with *S. Gallinarum* - Pullorum. While the isolation of many different *Salmonella* species from passerine birds has been reported, none of these reports include the identification of *S. Gallinarum*-Pullorum. Snoeyenbos (3) commented that natural infections with *S. Pullorum*, of species other than chickens or turkeys, have usually resulted from direct or indirect exposure to infected chickens.

The effective eradication of *S. Pullorum* from New Zealand through the implementation of a programme directed solely at commercial poultry supports the proposal by Snoeyenbos (3) that mammals or birds other than chickens and turkeys are of little or no importance in the epidemiology of the disease.

## **Conclusion**

Based on host preference, *S. Gallinarum-Pullorum* is not considered to be a potential hazard in the commodity.

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### 3.21.3 Salmonella Abortusovis, S. arizonae and S. Dublin

#### 3.21.3.1 Hazard identification

##### Aetiological agent

*Salmonella* Abortusovis, *S. arizonae* and *S. Dublin* are covered in this section.

##### New Zealand Status

*Salmonella* Abortusovis, *S. arizonae* and *S. Dublin*, are listed in the register of unwanted organisms.

##### Epidemiology

S. Abortusovis is strongly host adapted to sheep. Reports of natural infection in species other than sheep and goats have not been located.

S. Dublin is host adapted to cattle with limited infections occurring in other species. This is reflected in the data on *Salmonella* serotypes involved in “livestock incidents” in the United Kingdom during 2002 with 80.7% of such incidents in cattle being attributed to *S. Dublin*, 21.2% of incidents in sheep, 0.9% of those in pigs and a smaller proportion (not reported) in chickens (1). There is a small number of reports of *S. Dublin* in poultry but reports of the organism in passerine birds have not been located.

S. arizonae – Nomenclature applied to *S. arizonae* (*Arizona* spp.) has undergone changes which have resulted in *Salmonella enterica* subspecies III being partitioned to IIIa (*S. enterica arizonae*) and IIIb (*S. enterica diarizonae*) (2,3). Serological typing designation has also changed with moves from the use of *Arizona* antisera to *Salmonella* antisera. Designations used here will be those based on *Salmonella* antisera. Serotypes within these subspecies have not been named.

Three major epidemiological groups are identifiable within the subspecies III.

1. *S. arizonae* serotypes 18:Z<sub>4</sub> Z<sub>32</sub> and 18:Z<sub>4</sub>,Z<sub>23</sub> cause serious disease in turkeys. Chickens are affected infrequently and together with humans, sheep and dogs, are the only other species from which reports of this serotype have been identified (4, 5, 6),
2. *S. diarizonae* serotypes 61:k:1,5, (7) and 61:l,v: 5, (7) are common in sheep and there have been a small number of isolations of each from humans, snakes and/or other reptiles (4, 6),
3. Snakes, turtles other reptiles and amphibians (all “cold blooded” species) are infected by a wide range of serotypes of *S. arizonae* (4, 6). Weiss (4) reported the identification of 51 serotypes from snakes with 17 of these also being reported from humans. Of the 72 serotypes identified from humans 17 were also identified in snakes, three from sheep and one from cattle.

There are very few reports of isolations of *S. arizonae* from avian species other than commercial turkeys and chickens. *S. arizonae* has been reported from two individual cases of diseased caged psittacines in the United States (8) and Spain (9). The latter case followed shortly after the introduction to the premises of Iguanas which also became diseased and were found to be infected with the same organism. It has also been reported from wild sandhill cranes in the United States (10). Tizard (11) did not refer to *S. arizonae* in his review of salmonellosis in wild birds and independent searches of the scientific literature have revealed only one report of *S. arizonae* from a passerine bird. That was a report from India in which *S. arizonae* was isolated from four doves and a crow (13). The serotype was the same as that previously isolated from snakes and rats in the same area (13). The same serotype was also reported from snakes and humans in the United States (4), humans in the United Kingdom (most of whom had travelled to eastern Europe or Africa), Indonesian frogs legs and bean sprouts (6).

## Conclusion

*Salmonella Abortusovis* - On the basis of host specificity and lack of records of infection in birds, this species is not considered to be a potential hazard in the commodity.

*Salmonella Dublin* – On the basis of host preference and the lack of reports of infection in passerine birds, this species is not considered to be a potential hazard in the commodity.

*S. arizonae* – On the basis of species preference of major serotypes, the paucity of reports of infection in birds other than commercial poultry and the lack or reports of infection in passerine birds in Great Britain and Europe, *S. arizonae* is not considered to be a potential hazard in the commodity.

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### **3.21.4 *Salmonella* Typhimurium DT 104 carrying resistance to several antibiotics**

#### **3.21.4.1 Hazard identification**

##### **Aetiological agent**

The *Salmonella* addressed in this section is *S. Typhimurium* DT (phage type) 104, especially those strains carrying resistance to several antibiotics.

##### **New Zealand Status**

*S. Typhimurium* DT 104 is listed in the register of unwanted organisms.

*S. Typhimurium* DT 104 is isolated from humans and non-human sources in New Zealand relatively infrequently. Four isolates, one from each of four Health Districts, were identified in humans during 2003 (1). Thirty nine human isolates of antibiotic-resistant *S. Typhimurium* DT 104 were recorded between 1992 and 2001 with 37 of those being multi-resistant (2). A small number of multi-resistant isolates of *S. Typhimurium* DT 104 have also been obtained from non-human sources (2).

##### **Epidemiology**

*S. Typhimurium* DT 104 has a broad host range including cattle, sheep, goats, pigs, poultry, humans, dogs, cats, horses, and a number of other species (3, 4, 5, 6). In Britain, the earliest reports of *S. Typhimurium* DT 104 were from humans in the early 1960s (5). The first isolations of the multi-resistant strain, ACSSuT, were from a migratory gull and an imported parrot in 1984, with further isolations from imported exotic birds during 1985 and 1986 (7). In Britain, these isolations were followed by an epidemic of multi-resistant DT 104 ACSSuT (and variant strains) involving cattle, sheep, pigs, poultry and other species that peaked in 1996 and has since declined (7, 8). The pattern of infections in humans has been similar to that in cattle with a peak in 1997 and a decline continuing until the latest data available, from 2002 (9). Cattle are considered to be the reservoir host. The initial identification of multi-resistant *S. Typhimurium* DT104 in Britain, and the volume of data from there, may be the result of the thoroughness of its *Salmonella* surveillance system rather than a true reflection of the role of that country in the origin or epidemiology of the organism (10).

During the 1990s an epidemic of multi-resistant *S. Typhimurium* affected many countries. In the Pacific Northwest of the United States the commencement of the epidemic, which affected cattle and humans, was recognised in 1990 (11), in parallel with that in Great Britain. Threlfall (5), in his review, identified reports of human infections with multi-resistant *S. Typhimurium* DT104 through much of Europe, in the Middle East, South Africa, Trinidad and in the Philippines.

Although infection of poultry (chickens and turkeys) with DT104 is widely recognised, reports of its presence in other birds are uncommon and only one report of this organism being recovered from passerines has been found. Besser *et al.* (12) identified multi-resistant *S. Typhimurium* DT104 from one starling and one pine siskin in the Pacific Northwest of the United States between 1986 and 1991. Reports of *S. Typhimurium* from passerine birds are common but very few reports include the results of phage typing. Refsum *et al.* (13) identified DT40, U277, DT99 and DT110 from 12 isolates from passerines in Norway. The absence of DT104 may be due to the low level of presence of that organism in Norway at the time (14). Hudson *et al.* (15) examined 22 isolates from “non-domestic” birds in the south-east USA and identified DT104 from two pet birds, neither of which were from the order Passeriformes. *S. Typhimurium* DT160 was the cause of death of a large number of sparrows in New Zealand (16) and in their report on investigations into deaths of finches in Great Britain, Pennycott *et al* (17) found all *Salmonella* isolates to be *S. Typhimurium* DT40.

Swedish studies showed that as many as 5-11% of travellers could be carrying *Salmonella*, and more than half of these could be asymptomatic (18, 19). Travellers arriving in New Zealand are not screened for *Salmonella* infections (20).

## Conclusion

On the bases that

- the epidemic of *S. Typhimurium* DT104 is in decline and the prevalence in the reservoir host in Britain and Europe is now low,
- there is only one report of DT104 from passerines (and that in the United States) and
- dogs, cats and horses (all identified as hosts of *S. Typhimurium* DT 104) have entered New Zealand throughout the period of the global epidemic without constraints relating to this organism and
- there are many uncontrolled pathways by which salmonellae can enter New Zealand, including humans travelling to this country

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

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### **3.21.5 Salmonella Enteritidis phage type 4**

#### **3.21.5.1 Hazard identification**

##### **Aetiological agent**

The Salmonella addressed in this section is *S. Enteritidis* phage type 4 (3).

##### **New Zealand Status**

*S. Enteritidis* phage type 4 is the second most common *S. Enteritidis* phage type isolated from humans in New Zealand (1) and 22 isolates from 12 Health Districts were recorded in 2003 (2). The majority of these infections appear to arise during international travel (1). Isolations from non-human species in New Zealand have been reported but are infrequent (3).

##### **Epidemiology**

The earlier stages (1979 to 1987) of an international pandemic of human disease attributed to *Salmonella Enteritidis* were described by Rodrigue *et al.* (4). The affected countries were in North and South America, Europe and possibly southern Africa. Human cases were attributed to consumption of eggs or poultry from infected chickens in which infection was asymptomatic. Data was based on reporting to WHO. Data on the pandemic, as it affected Great Britain, shows a slow increase in isolations of *S. Enteritidis* from domestic fowls from the early 1980s with a rapid increase from 1987 to a peak in 1990, followed by an initially rapid and subsequently slower decline to very low levels in 2000. Human cases rose rapidly from 1985 to peak around 1989. A lengthy plateau followed, with a marked decline from 1999, though not to the same extent as the decline in data from poultry. The decline in *S. Enteritidis* incidents in poultry followed introduction of control measures and codes of practice in the industry. These measures appear to have been effective *for S. Enteritidis*. The data does not show a comparable decline in incidents of other *Salmonellae* in poultry but it is suggested that this might be because of increased monitoring (5) This pandemic has been associated with *S. Enteritidis* PT 4 in Great Britain and continental Europe (6) but with PT 13, PT 8 and PT4, in declining order, in North America (7). The on-farm epidemiology of *S. Enteritidis* and the basis for the development of the pandemic have been reviewed by Guard-Petter (8). In a retrospective view of the epidemic of *S. Enteritidis*, Baumler *et al.* (9) hypothesised that the control of *S. Gallinarum*-Pullorum, which shares an immunodominant surface antigen with *S. Enteritidis*, during the mid-1900s, removed the protective effect of that organism and left an ecological niche to be filled by *S. Enteritidis*. Mice are considered to be the likely reservoir for *S. Enteritidis* (10, 11, 12) and the decline in flock immunity following control of *S. Gallinarum*-Pullorum allowed widespread infection of poultry.

The one report of *S. Enteritidis* in passerine birds located was from Greece where three of 182 canaries necropsied during the 1990s were found to be infected with this serotype.

Thirty of the birds were infected with *S. Typhimurium*. The phage type of *S. Enteritidis* is not recorded in the abstract available (13).

Swedish studies showed that as many as 5-11% of travellers could be carrying *Salmonella*, and more than half of these could be asymptomatic (14, 15). Travellers arriving in New Zealand are not screened for *Salmonella* infections (16).

## Conclusion

On the bases that

- only one report of *S. Enteritidis* from passerines has been located (Phage type not known) and the accumulated picture from other reports indicate that the prevalence of this serotype in passerines is very low,
- no suggestions, or evidence, that passerine birds play any significant role in the epidemiology of *S. Enteritidis* infections in poultry or humans have been located,
- the reservoir of the organism appears to be mice,
- the epidemic of the organism in poultry is in decline,
- the low rate of isolation of *S. Enteritidis* PT4 from non-human species in New Zealand is comparable with the situation in other countries and
- there are many uncontrolled pathways by which salmonellae can enter New Zealand, including humans travelling to this country,

*S. Enteritidis* PT 4 is not considered to be a potential hazard in the commodity.

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### **3.21.6 Salmonella Typhimurium phage type 44**

#### **3.21.6.1 Hazard identification**

##### **Aetiological agent**

The Salmonella addressed in this is *S. Typhimurium* phage type 44 (1).

##### **New Zealand Status**

*S. Typhimurium* pt 44 is included in the register of unwanted organisms. No reports of *S. Typhimurium* pt 44 in New Zealand have been located.

##### **Epidemiology**

Few reports of the isolation of *S. Typhimurium* pt 44 have been found. Searches of data from national salmonella surveillance programmes available on the internet revealed reports of *S. Typhimurium* pt 44 from Australia but not from any other country. Powling et al. (2), in 1994, reported an increase in the number of isolates of *S. Typhimurium* pt 44 from humans with antibiotic resistance patterns similar to those seen in isolates from cattle. In the reports discovered (3, 4, 5, 6, 7, 8, 9), all isolations have been from humans or cattle, although a case control study following an incident in which 11 people became infected following dining at a restaurant in South Australia, suggested that the source could have been either pork or apple sauce (8). Isolations have come from most states in Australia but the numbers of cases per year in both cattle and humans are small. No reports of *S. Typhimurium* pt 44 in birds have been located.

##### **Conclusion**

On the bases that no reports of *S. Typhimurium* pt 44 other than from Australia and other than from humans and cattle, this organism is not considered to be a hazard in the commodity.

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### **3.21.7 Other Salmonellae**

#### **3.21.7.1 Hazard identification**

##### **Aetiological agent**

The Salmonellae addressed in this section are those serotypes and phage types not covered in sections 3.21.1 to 3.21.5.

##### **New Zealand Status**

In New Zealand, over the period 1999 to mid 2005, typing of Salmonella isolates from humans yielded over 140 Salmonella serotypes/phage types. During the same period typing of isolates from animals, animal feeds and their environment yielded over 80 serotypes/phage types. The frequency with which specific types were isolated each year varied greatly and many of the serotypes/phage types were isolated from human or non-human sources on only one occasion. Each year, three to five serovars or phage types not previously identified in New Zealand were reported. Most were from humans, most of whom were travellers or immigrants (1). As many Salmonella infections are asymptomatic, the full range of serovars and phage types present in New Zealand and the extent of introductions to the country is unknown. Except in connection with specific incidents of note, records of Salmonella isolates do not differentiate those from passerine birds.

An epidemic of *S. Typhimurium* DT160, commencing in the winter of 2000, resulted in the death of a large number of sparrows and lesser numbers of finches and blackbirds (2). In birds, this epidemic was restricted to a small range of passerine species. It did however, extend to infect humans, livestock, cats and rabbits. This organism had not been identified in New Zealand prior to isolation from a human in 1998. Introduction to New Zealand with a human carrier was considered a possibility but there was insufficient evidence to draw any firm conclusion.

##### **Epidemiology**

The epidemiology of different Salmonella serotypes follows broadly similar patterns. Spread within and between susceptible species is mainly via the faecal-oral route, with infection passed by infected animals able to survive for varying periods of time in different environmental niches. Host specificity or host preference varies between Salmonella serotypes. Some are highly host specific, while others are less so. It has been thought that some serotypes, especially *S. Typhimurium*, have very little host preference. This view is being revised with the recognition that genetic determinants are contributing to substantial variations in the breadth of host range for many strains (3, 4, 5).

Very few reports are available on Salmonellae in cage birds. Those that have been identified (6, 7) are in German and available only as abstracts. Tizard (8) has recently reviewed Salmonellosis of wild birds. Salmonellae infecting passerine birds and

identified in that review are shown in Table 2. The passerine species from which *Salmonellae* are most commonly isolated are sparrows and finches.

**Table 2. *Salmonellae* infecting passerine birds (8)**

<b>Serotype</b>	<b>Definitive type</b>	<b>Reported from Passerines in the EU.</b>	<b>Identified in New Zealand (1)</b>
Typhimurium	DT 14	Yes	No
	DT24	No	No
	U165 / DT40	Yes	Yes, from humans
	<b>DT41</b>	Yes	Yes, Mostly from environmental samples. Also from humans, bovines and imported spices
	DT56	yes	No
	U17 / DT 80	Yes	No
	DT99	yes	Yes, from pigeons
	DT110	yes	Yes, from poultry feed
	U239 / DT129	Yes	No
	U218 / DT160	Yes	Yes, from passerines, humans and a range of mammalian and avian sources. )
	U19 / DT161	Yes	No
	U277	yes	No
Dublin		yes	Yes, from humans.
Paratyphi B		yes	Yes, from human travellers
Schleissheim		Yes (only isolate of this serovar from outside Turkey.)	No
Saintpaul		No	Yes, from humans, birds, cattle and other mammals, stock and poultry feeds and environmental samples.
Bareilly		No	Yes, from humans (including travellers), and imported poultry feed.
Weltevreden		No	Yes, from imported foods and human travellers.
Paratyphi B		yes	Yes, from human travellers

\* U designated isolates have been translated to Definitive Type (DT) based on the proposal by Anderson et al. (11).

*S. panama* (9) and *S. bovis-morbificans* (10) have also been identified from passerine birds. These organisms have been reported from New Zealand (1).

From the review by Tizard (8) it is evident that the prevalence of *Salmonella* infections in passerine birds commonly reflects environmental contamination. Birds on farms, particularly in the presence of *Salmonella* infections in livestock, have higher carrier rates than birds in most other locations. The prevalence in scattered populations is generally very low but epidemics of disease occur commonly in areas where birds gather in large numbers, attracted by feeding tables or, as was the case with some locations in the New Zealand epidemic (2), feeding opportunities around grain silos. The two *Salmonellae* most commonly associated with epidemics of disease in passerines are *S. Typhimurium* DT40 and DT160, both of which are present in New Zealand. Rabsch et al. (5) proposed that the epidemiological patterns of infection with *S. typhimurium* DT40 were consistent within its being highly host adapted to a narrow range of passerines birds. The epidemiological patterns observed for other *Salmonellae* reported from passerines do not match the criteria proposed by Rabsch et al.

## **Conclusion**

Based on the serovars and phage types of *Salmonellae* identified from passerine birds in the EU and not identified in New Zealand the following are concluded to be potential hazards in the commodity. *Salmonella* Typhimurium DT14, DT56, DT80, DT129, DT161 and U277.

### **3.21.7.2 Risk assessment**

#### **Release assessment**

Vertical transmission of salmonellae infecting poultry can take place either through internal infection in eggs, or through external contamination of egg shells during laying. *S. Gallinarum-Pullorum*, *S. Enteritidis* in chickens and *S. arizonae* in turkeys are recognised as being transmitted in this way, although transovarial transmission is considered the most important route (12, 13). These organisms have been discussed in sections 3.21.1, 3.21.2 and 3.21.4 above.

Transovarial transmission requires that the organism infects the ovary and/or oviduct of the bird (14). That such infections are restricted to only specific *Salmonellae* was illustrated by artificial infection of chickens with six *Salmonella* serovars with *S. Enteritidis* being the only serovar resulting in infection of tissues of the reproductive tract (Okamura et al 2001). The *Salmonella* serovars / DTs infecting the reproductive tracts of chickens and turkeys are highly host adapted and De Buck et al (14) observed that tropism for the reproductive tract is shown by *S. Abortusequi* and *S. Abortusovis*, both of which are highly host adapted.

Vertical transmission resulting from contamination of the outside of eggs may occur but this route appears less common than the transovarial route although it may result from

contamination by a wider range of *Salmonella* serovars present in the environment. Infection of eggs with *Salmonellae* on or through the shell is uncommon and most infection of chicks in hatcheries arises from environmental following the hatching of infected eggs (16). No reports suggesting vertical transmission of *Salmonellae* in passerine birds have been located. Although Cizek et al (18) stated that they did not consider that vertical transmission plays a significant role in the epidemiology of *Salmonellae* in passerines, the possibility of such transmission can not be ruled out.

## **Conclusion**

Based on the evidence that vertical transmission of *Salmonellae* occurs in chickens and turkeys, the lack of evidence that such transmission does not occur in passerine birds, it is concluded that *Salmonellae* serovars / DTs infecting passerines and not present in New Zealand are potential hazards in the commodity.

(*Salmonella* Typhimurium DT14, DT56, DT80, DT129, DT161 and U277 are organisms fitting the definition above.)

### **3.21.7.3 Risk Assessment**

Vertical transmission via eggs is only one way by which *Salmonellae* pathogenic to passerine birds might enter New Zealand. The recent review of pathways for entry of *Salmonellae* into New Zealand (17) documented the large number of uncontrolled (or only lightly controlled) means by which the organisms can, and do, enter the country.

Non-passerine-host-adapted *Salmonellae* are able to enter New Zealand in humans and this has been proposed as a pathway by which *S. typhimurium* DT160 may have arrived (2). Non-host adapted *Salmonellae* also enter New Zealand in poultry feeds and stock feeds without regulatory control and they may gain access with the importation of live animals, seeds or other goods (17).

Although passerine-host-adapted *Salmonellae* are not listed in the potential pathogens, pathways for entry of such organisms include grains contaminated by feeding passerines either prior to or after harvest and contaminated poultry feeds and animal feedstuffs (17).

MacDiarmid (17) comments on the need for consistency in the application of sanitary measures to different products. Given the uncontrolled entry of humans, some of whom are certainly infected with *Salmonellae* and the importation of many tons of grains, poultry feeds, stock foods and pet foods, some of which have been demonstrated to be infected with *Salmonellae*, it is considered that it would be inconsistent to apply sanitary measures targeted at *Salmonellae* to passerine eggs which have only a low likelihood of being infected.

Sanitary measures targetted at “other *Salmonellae*” are not recommended.

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### 3.21.8 *Escherichia coli*

#### 3.21.8.1 Hazard identification

##### Aetiological agent

*Escherichia coli* (*E. coli*) is a member of the Enterobacteriaceae. It is a lactose fermenter and, usually, motile. Although *E. coli* is a normal inhabitant of the intestinal tract of many species of animal (1). Strains of *E. coli* are, commonly, differentiated on the basis of somatic, flagellar and capsular antigens. Although *E. coli* is a normal gut organism, particular strains (or strains with particular virulence factors) may be associated with disease (1).

Strains considered in this risk analysis are

1. Avian pathogenic *E. coli* (APEC) and
2. Verotoxin (or shigatoxin) producing *E. coli* (VTEC).

Avian Pathogenic *E. coli* – In a review of factors associated with the pathogenicity of APEC, Barnes *et al.* (2) comment that there is no single factor clearly associated with pathogenicity or virulence. Various studies have identified associations between virulence and certain O antigens, K1 and K80 capsular antigens, adonitol fermentation, antibiotic resistance, fimbria, motility, genes for enterotoxins and others. Differentiation of *E. coli* strains as APEC and non-APEC does not appear feasible. Pennycott *et al.* (3) identified *E. coli* O86 as one of the commonest causes of deaths of wild finches in areas of Britain where high mortality rates had been reported.

Verotoxin producing *E. coli* – *E. coli* genotypes and phenotypes and virulence factors associated with verotoxin production have been reviewed by Paton and Paton (4), Law (5) and Keskimäki (6). *E. coli* O157:H7 is the most widely recognised VTEC and other strains with O157 antigen have been commonly associated with verotoxin production and disease. Verotoxin production has, however, been identified in over 100 other serotypes. Genetic typing has allowed identification of *E. coli* strains carrying genes for verotoxin (Shiga toxin) denoted stx<sub>1</sub> and stx<sub>2</sub>. These genes may be present alone or in combination and variants, especially of stx<sub>2</sub>, exist. The presence of either, or both, of these genes does not, of itself mean that the organisms will be virulent. Other proposed virulence factors include enterohaemolysin, an outer membrane protein called intimin, an extracellular serine protease and others.

##### OIE List

*E. coli* is not included in the OIE list.

##### New Zealand Status

*E. coli* is not included in the register of unwanted organisms under the Biosecurity Act.

“Acute gastroenteritis” is a notifiable disease under schedule 1 of the Health Act. The appended note states “ This category includes acute gastroenteritis in 2 or more linked persons (common food or water source); an affected person in a high-risk occupation (e.g., food handling); or single cases of botulism, chemical poisoning or verotoxin-producing infections, particularly *E. coli* 0157.”

Avian Pathogenic *E. coli*.– Although records of the characterisation of APEC have not been located, *E.coli*-induced diseases of poultry have been reported (7)

Verotoxin producing *E. coli*.– VTEC are present in New Zealand and are considered one of the more important causes of enteric disease in humans (8). Brooks *et al.* (9) isolated VTEC from beef, mutton, lamb, pork, chicken and sausage mixtures sampled from retail outlets in Dunedin, New Zealand. Based on the serotypes of the VTEC organisms identified in New Zealand and those associated with serious disease outbreaks in international reports, Lake *et al.* (10) concluded that most of the types of VTEC identified from meat products in New Zealand cause disease infrequently or not at all.

## **Epidemiology**

Although *E. coli* is a normal inhabitant of the intestinal tract of many animals it is not constant in the gut of passerine birds. Morishita *et al.*(11) isolated *E. coli* from healthy passerine birds in Ohio and suggested that isolation rates might be related to the environment in which the various species fed and contamination of the feeds they consumed. *E. coli* is shed in faeces and infects other animals through direct or indirect contact (2) and can be transmitted through eggs laid by hens recovering from infection with pathogenic strains (12).

Avian Pathogenic *E. coli*.– Host susceptibility/resistance may be a greater determinant of the occurrence of *E.coli*-associated disease in birds than the virulence factors of the associated *E. coli* strain (2). Factors associated with *E.coli* disease include infections with a wide range of viruses, bacteria and parasites, exposure to a range of toxins, physiological status and environmental stressors (2).

Verotoxin producing *E. coli*.– In his review of STEC (VTEC) Keskimäki (6) identified references to the organism(s) being found in the faecal flora of a range of species including cattle, sheep, dogs, pigs, cats, dogs horses and wild birds. The most important source of infection of humans appears to be cattle with the main route of infection being via ingestion of meat products contaminated in abattoirs. Non-O157 strains are more common than O157 strains and the range of foodstuffs from which VTEC organisms has been found includes sausages, beef, minced beef, lamb, milk, cheese, pork and milk filters (13).

The major risk factors, associated with development of disease, relate to exposure to contaminated meat (especially under cooked minced beef), farming environments and other locations where exposure to material contaminated with animal faeces is likely (e.g. drinking, or swimming in, unchlorinated water) (14, 15, 16).

*E. coli* with stx genes have been identified from chickens and turkeys (17) although production of verotoxin is considered uncommon. Investigations of the genotypes of *E. coli* isolates from gulls, pigeons and chickens in Finland (18) found an absence of stx genes, a presence of eae genes (for intimin production) and an absence of other genes characteristic of human pathogenic strains. The authors concluded that birds could not be regarded as important carriers of human pathogenic *E. coli* in Finland.

## Conclusion

Avian Pathogenic *E. coli*.– On the basis of avian Pathogenic *E. coli* being present in New Zealand it not considered that these organisms are a hazard in the commodity.

Verotoxin producing *E. coli*.– On the bases that VTEC are present in New Zealand and that there is no evidence that passerine species play any part in the epidemiology of the disease, it is considered that VTEC are not a hazard in the commodity.

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### **3.21.9 Campylobacter spp.**

#### **3.21.9.1 Hazard identification**

##### **Aetiological agent**

*Campylobacter jejuni* and *Campylobacter coli* are the organisms considered to be of interest in this risk analysis. Both are thermophilic members of the genus *Campylobacter*. *C. jejuni* is the organism of greatest interest but the epidemiology of both is similar. Where differences between the two organisms warrant, those differences will be highlighted.

##### **OIE List**

*Campylobacter* species are not included in the OIE list.

##### **New Zealand Status**

*Campylobacter* species are not listed in the unwanted organisms register. Campylobacteriosis of humans is a notifiable disease under the provisions of the Health Act 1956.

Campylobacteriosis is the most commonly reported notifiable disease of humans in New Zealand. During 2003 there were 14,786 cases notified of which 78% had laboratory confirmation. Of the 8320 cases for which hospitalisation status was recorded, 633 (7.6%) required hospitalisation. Epidemiological data reported suggested that food from retail premises, contact with farm animals, consumption of untreated water and contact with recreational water may have been sources of infection.(1) Poultry products are commonly claimed a major source of human *Campylobacter* infections and a risk profile for *C. jejuni* and *C. coli* in poultry, compiling information from a number of sources, has been prepared (2). That report indicates that the prevalence of *Campylobacter* infection of live broiler chickens in New Zealand is at the lower end of rates reported from overseas (14 to 100% varying within and between sampled populations in different countries). Data from a number of studies in NZ have shown *C. jejuni* contamination rates in raw poultry products between 14 and 80%. Contamination of cooked products is uncommon. Studies in which isolates of *C. jejuni* from poultry products and human infections were compared has shown substantial overlaps in the types from the two sources but this was not the case in a study directed at a rural area. This suggests that sources of human infections with *C. jejuni* may differ between urban and rural areas.

NZ studies contributing to risk categorisation of food/hazard combinations were reviewed (2) and have allowed conclusions that up to 65% of human campylobacteriosis cases have their origins in food, around 2.3% of the population may be infected with *Campylobacter* spp. each year with 1.5% being infected via food sources. Consumption of poultry represents the greatest risk. This report assesses that approximately 0.3% of

human infections result in hospitalisation, placing infections, generally, in a low severity category.

Use of antibiotics in animals in New Zealand is under regulatory control. Of particular concern is the control of the use of antibacterials, as mass prophylactic medicinals or for growth promotion purposes, to minimise the risks of development of antibiotic resistance in zoonotic organisms (3, 4, 5). There is no routine surveillance of antibiotic resistance amongst in *Campylobacter* spp. from either animals or humans (6). The only data available is from a study of 202 isolates from the Auckland area in 1998 which found three (1.5%) of the isolates resistant to erythromycin and five (2.5%) resistant to fluoroquinolone (7).

*C. jejuni* and *C. coli* are endemic in New Zealand animals. *C. jejuni* has been reported from faecal samples from normal and scouring foals (8), from normal hoggets and in association with *Yersinia enterocolitica* in cases of diarrhoea (9), from clinically normal dairy cattle (10), aborted lamb fetuses (11), puppies with diarrhoea (12), an ostrich with inflammatory disease of the brain, heart and liver (13), healthy and diarrhoeic pigs (14) and cases of abortion in goats (15). Reports of *C. coli* infections in animals have been found from clinically normal dairy cows (10), aborted lambs (11) and from pigs (14).

## **Epidemiology**

The epidemiology of thermophilic *Campylobacter* species has been summarised by Shane and Stern (16). Infection of birds with *C. jejuni* or *C. coli* is usually asymptomatic. Pathology associated with infection has been recorded in very young chicks. Birds, especially poultry, form a major reservoir of thermophilic *Campylobacter* spp. Infection has been recorded from many species of birds, including a large number of passerine species. Transfer of infection both within and between species is mainly through the faecal-oral route although infection of carrion-eating birds (and of humans) has shown less direct transfer of organisms, commensal in the intestinal tract, can be important avenues for infection. Although the evidence is not conclusive, it is likely that *Campylobacter* infection can be transmitted vertically through eggs.

Infection rates in passerine birds have been shown to exceed 60% in some species in some locations (17, 18). Other reports (19, 20, 21, 22) have been of lower prevalence rates. Passerine birds have been considered to be sources of infection for humans through contamination of milk in bottles (23, 24) and infection of birds in private homes, pet shops, quarantine stations, animal shelters and breeding flocks has been demonstrated (25). Infection of starlings and sparrows in the vicinity of poultry sheds has been reported (21) but the status of the poultry flocks or of wild birds more distant from the poultry shed was not reported for comparison.

The epidemiological picture of human *Campylobacteriosis*, and animal infections, in Great Britain and Europe is similar to that in New Zealand. One feature that has been reported elsewhere, but which is not clear from the New Zealand reports located, is the

presence of asymptomatic infections in humans (compared with symptomatic infections) (26, 27).

The development of strains of *Campylobacter* spp. resistant to antibiotics, especially fluoroquinolones is of concern in Europe and the United States. The Committee for Veterinary Medicinal Products of European Agency for the Evaluation of Medicinal Products (28) has concluded that the use of fluoroquinolones in poultry leads to the development of resistance in *Campylobacter* spp. and that poultry meat is the major source of such resistant strains in human infections. Krause and Ullmanz (29) reported increases in *Campylobacter* strains resistant to fluoroquinolones from 0% in 1980/82 to 12.1 – 30.3% in 2001.

## **Conclusion**

It is concluded that neither *C. jejuni* nor *C. coli* are hazards, except that Fluoroquinolone resistant strains of *C. jejuni* and/or *C. coli* are potential hazards in the commodity.

### **3.21.9.2 Risk assessment**

#### **Release assessment**

Reports of fluoroquinolone resistant strains of *Campylobacter* spp. in passerine birds (or other wild birds) have not been located, nor have reports of investigations that might have revealed such strains. The association of wild passerine birds with poultry sheds and the finding of *Campylobacter* spp. in such wild birds (21) leaves open the possibility of passerine birds acquiring infection with fluoroquinolone resistant strains of *Campylobacter* spp. It is also feasible that birds in captive collections could be directly exposed to fluoroquinolone and that resistant strains of **Campylobacter** could result. Transfer of resistant strains of *Campylobacter* spp to eggs has a low but non-negligible likelihood.

The release assessment is non-negligible.

#### **Exposure assessment**

Passerine eggs imported from Europe, carrying fluoroquinolone resistant strains of *Campylobacter* spp. may result in birds that transmit infection to other birds with which they share accommodation, birds or animals they may come in contact with through aviary walls or to associated humans. Release of the infected birds could lead to direct dissemination to a range of species, including poultry. One can also envisage more general spread from the infected passerines to other species by indirect routes (e.g. through other avian or mammalian species).

The exposure assessment is non-negligible.

## Consequence assessment

The likelihood and consequences of the dissemination of fluoroquinolone resistant strains of *Campylobacter* spp. originating from imported passerine eggs is small compared with the likelihood of the development of resistant strains through the use of the relevant antibiotics for therapeutic purposes in humans or other species. It must also be compared with the likelihood of such resistant strains reaching New Zealand in either diseased or asymptomatic human carriers. The reality of this potential is illustrated by the report of Sharma *et al.* (30) who found antibiotic resistance to be significantly more common in *Campylobacter* isolates from human patients who were thought to have acquired the infection overseas than amongst infections acquired locally in the Hunter Valley region of Australia.

The consequence assessment is negligible.

## Risk estimation

Based on the negligible consequence assessment it is concluded that Fluoroquinolone resistant strains of *Campylobacter* spp. are not a hazard in the commodity.

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### **3.21.10 Other Enterobacteriaceae**

#### **3.21.10.1 Hazard identification**

##### **Aetiological agent**

Other members of the Enterobacteriaceae considered are:

- *Proteus* spp.
- *Edwardsiella* spp.
- *Klebsiella* spp.
- *Serratia* spp.
- *Morganella* spp.
- *Enterobacter* spp. and
- *Yersinia* spp.

##### **OIE List**

None of these organisms are included in the OIE list.

##### **New Zealand Status**

*Edwardsiella ictaluri*, *Yersinia pestis* and *Yersinia ruckeri* (exotic strains) are listed in the unwanted organisms register.

##### **Epidemiology**

*Edwardsiella ictaluri* inhabits of the gut of catfish and some other species. It is an important cause of disease in some fish populations (1).

*Yersinia pestis* has rodents as its reservoir hosts and causes bubonic and pneumonic plague in humans and feline plague in cats (2).

*Yersinia ruckeri* is the cause of redmouth disease in fish, particularly rainbow trout (3).

*Klebsiella* spp. – These organisms are normal inhabitants of the bowel and respiratory tract of humans and other animals (4) including passerine birds (5). They are also commonly found in soil and water (4). *Klebsiella pneumoniae* infection has been associated with disease in humans (4) and with mastitis and metritis in cattle, metritis in sows and pneumonia in foals and primates (6). In New Zealand *Klebsiella* spp. have been reported from cases of bovine mastitis (7) and from omphalitis/peritonitis in Ostrich (8).

*Proteus* spp. – Organisms of this genus are widely distributed in nature, particularly associated with faecal material, sewage and decomposing material of animal origin (9).

*Proteus* spp. have been shown to be inhabitants of the hind-gut of clinically normal passerine birds (10). The international literature includes reports of *Proteus* spp. being associated with a wide range of clinical conditions in animals including otitis in dogs (11), urinary tract infections (12) and mastitis in cattle (13). In New Zealand *Proteus mirabilis* has been reported from urinary infection in a dog with a urethral defect (14), cases of bovine mastitis (15), and milk from goats with high somatic cell counts (16).

*Edwardsiella* spp. – Organisms of this genus are found almost exclusively in fish and the associated aquatic environments (4). Internationally *E. tarda* has been isolated from fish and from amphibians and reptiles, predominantly from aquatic environments. There is a small number of reports of infections in marine mammals (17, 18, 19), a single report of the organism being located in rock-hopper penguins (20) and one from pigs (21). No reports of *Edwardsiella* spp in passerine birds have been located. Reports of *Edwardsiella* spp. being isolated in New Zealand have not been found.

*Serratia* spp. – *Serratia* spp. are found in soil and water with some isolations coming from animals. *S. marcescens* is implicated as an opportunist pathogen in hospital patients at times (4). *S. marcescens* has been diagnosed as the cause of mastitis in cattle (6, 22). It has also been isolated from other cases of diseased animals including abscesses in ewes (23), a case of equine abortion (24), horses with respiratory disease (25) and a case of equine myocarditis (26). *Serratia* spp. have been shown to be inhabitants of the hind-gut of clinically normal passerine birds (10). From New Zealand, there are reports of the isolation of *Serratia marcescens* from a case of chronic bronchopneumonia in a cat (27), *Serratia* sp. from 11 cases of bovine mastitis (7) and *Serratia marcescens* from juvenile budgerigars with poor liveability and poor hatchability (Christensen, N. 2005. Review of draft Import Risk Analysis).

*Morganella* spp. – *M. morganii* is common in the intestinal tract and faeces of humans, other mammalian species and reptiles (4, 28). This organism has also been isolated from faeces of healthy birds (29) including a passerine species (*Prunella* sp.) (30), and from tissues of birds with respiratory diseases (31, 32). *M. morganii* has been associated with urinary tract infections, perinatal infections and joint infections in humans (33). *M. morganii* is a histamine producer and a significant contributor to scromboid poisoning (34). In animals, *M. morganii* has been shown to be capable of inducing diarrhoea in colostrum deprived calves (35), and associated with septicaemia in alligators (36), “hole disease” in soft-shelled turtles (37) and pneumonia in a captive turtle (38). No reports of *M. morganii* causing disease in birds have been located. In New Zealand, *M. morganii* has been reported from a foal that died at two weeks old (39).

*Enterobacter* spp. – *E. spp.* have been isolated from soil and water and from a wide variety of animals including humans (4, 28). *Enterobacter* spp. are a significant cause of nosocomial blood stream infections in hospitalised people (40) and *E. sakazakii* can cause serious disease in infants (41). Internationally, *Enterobacter* have been associated with genital infections of mares (42, 43) and stallions (*E. aerogenes*) (43), equine abortions (*E. agglomerans*) (44), udder infections in cows (*E. spp.*) (45), an enteric disorder in a calf (*E. agglomerans*) (46), meningoencephalitis in a calf (*E. cloacae*) (47)

and an inflammatory condition in the skin of sheep (*E. cloacae*) (48). *Enterobacter* spp. have been isolated from the faeces of healthy passerine birds (5) and from dead canaries (49) and finches (50). In New Zealand, *E. spp.* has been reported from cases of bovine mastitis (7) and *E. aerogenes* from the lungs of two cats, each dying after short febrile illness (51).

*Yersinia* spp. (other than *Y. pestis* and *Y. ruckeri*) – *Y. enterocolitica* and *Y. pseudotuberculosis* are pathogens affecting humans, other mammals and a range of other animals. Other *Yersinia* species (*frederiksenii*, *intermedia*, *kristensenii*, *rohdei*, *bercovieri*, *philomera*) are commensals or saprophytes (52) although Quan (53) recognises this latter group as occasional opportunist pathogens. Approximately 50 serotypes of *Y. enterocolitica* are recognised but only a small proportion of them are pathogenic. The serotypes that predominate in human illness are O:3, O:8, O:9 and O:5,27 (54). O:8 is common in North America whereas O:9 is common in Europe (53). Six serotypes (I to VI) of *Y. pseudotuberculosis* are recognised, each containing pathogenic strains (54).

In New Zealand, *Y. enterocolitica* serotypes O:2,3, O:3, O:5, O:6,30, O:5,27 and O:9 have been isolated, variously, from either humans, pigs, dogs or other domestic animals (55, 56, 57). *Y. enterocolitica* biotype 4 has been reported from pigs (57), biotype 5 from sheep (57), biotypes 1, 2, 3 and 5 from deer (58) and biotype 1a and untypable strains from birds (59). *Y. pseudotuberculosis* serotypes I, II and III have been reported from livestock, rabbits, guinea pigs and aviary birds (60) and from two of 1370 avian samples from wild birds (59). *Y. intermedia*, *Y. frederiksenii* and *Y. kristensenii* have been reported from mammalian and avian sources (58, 59, 61).

## Conclusion

Based on the epidemiology of these organisms, and the presence in New Zealand of those recognised as infecting birds, it is considered that members of the genera of Enterobacteriaceae discussed above are not potential hazards in commodity.

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## 3.22 Bacteria commonly associated with respiratory disease in birds

### 3.22.1 *Pasteurella multocida* (fowl cholera, avian cholera)

#### 3.22.1.1 Hazard identification

##### Aetiological agent

*Pasteurella multocida* is a gram-negative, non-motile, non-spore forming, rod-shaped bacterium. It grows either aerobically or anaerobically (1, 2). Isolates have been characterized on a number of bases (capsulated / non-capsulated, colour of colonies (related to type of capsule), serological characteristics, biochemical characteristics) but none of these features correlate well with pathogenicity. In their review Glisson *et al* (1) comment on the general principle that genotype and phenotype may not correlate because the latter relies on expression of genes, not simply their presence. Specific examples include observations that virulent strains are capsulated but capsulated isolates may have low virulence and that phenotypic serotype does not consistently correlate with genotype.

##### OIE List

Fowl cholera is included in the OIE list of notifiable diseases.

##### New Zealand Status

“*Pasteurella multocida* (toxigenic strains) – Atrophic rhinitis” (a disease of pigs) and “*Pasteurella multocida* B:2 E:2 – Haemorrhagic septicaemia” (a disease of cattle) are included in the register of unwanted organisms as “notifiable organisms”.

“Acute fowl cholera (*Pasteurella multocida*)” was removed from the list of notifiable organisms in 2000 (3) and no longer appears in the list of unwanted organisms ([www.maf.govt.nz/UO](http://www.maf.govt.nz/UO) ).

*P. multocida* is endemic in the New Zealand avian population with documented diagnoses including chronic disease in laying hens (4, 5), mortalities in turkeys (6), 50% mortality in a flock of 300 turkeys (7) and joint disease in roosters (8). A vaccine (Pabac – ACVM No. A3006) is registered “For the vaccination of healthy chickens and turkeys as an aid in the prevention of fowl cholera, types 1, 3 and 4” ( [www.nzfsa.govt.nz/acvm-register/labels/A003006-label.pdf](http://www.nzfsa.govt.nz/acvm-register/labels/A003006-label.pdf) )

During an investigation into mortalities in rockhopper penguins (*Eudyptes chrysocome*) on Campbell Island, *P. multocida* was identified as a cause of some deaths. The organisms isolated from chicks were classified as Capsule serogroup A, somatic serotype 1 which is the main serotype identified in epizootics of avian cholera in wildlife in North America (9).

## Conclusion

Based on the existence of the disease in New Zealand *P. multocida* (fowl cholera) is not considered to be a potential hazard in the commodity.

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### **3.22.2 Riemerella anatipestifer**

#### **3.22.2.1 Hazard identification**

##### **Aetiological agent**

*Riemerella anatipestifer* was previously called *Pasteurella anatipestifer*. The organism is a non-motile gram-negative rod that grows best in enriched media in an atmosphere with 5 – 10% CO<sub>2</sub> (1).

##### **OIE List**

*Pasteurella anatipestifer* is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

Not listed in the register of unwanted organisms.

*Pasteurella anatipestifer* was diagnosed as the probable cause of an outbreak of disease in ducks in 1974 in which there was 15% mortality (2) and a further incident in which 4 of 16 ducks died was considered typical of *P. anatipestifer* infection (3). Although neither of the isolates for these cases was definitively identified, it appears likely that *R. anatipestifer* is endemic in NZ.

##### **Epidemiology**

*R. anatipestifer* is widely distributed around the world and is recognised, most commonly, when it causes disease in intensively reared ducks. It also causes losses in geese and turkeys. The organism has also been found in pheasants, chickens, guinea fowl, quail, partridges and other waterfowl (4). Searches of electronic databases have not revealed any reports of *R. anatipestifer* in passerine birds. No reports suggesting vertical transmission of *R. anatipestifer* have been located.

Differences in the pathogenicity of strains of *R. anatipestifer* are recognised. The reports of disease in ducks in NZ attributed to this organism suggest that pathogenic strains may exist in this country.

##### **Conclusion**

Based on the evidence that pathogenic *R. anatipestifer* may be present in New Zealand, the lack of evidence that the organism is transmitted through eggs and the lack of evidence that it infects passerine birds, this organisms is not considered to be a potential hazard in the commodity.

## References

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2. Anonymous 1974 Lincoln Animal Health Laboratory. *Surveillance* 1 (4): 20-24.
3. Orr, M.B. 1990 Animal Health Laboratory Network. Review of diagnostic cases – April to June 1990. *Surveillance* 17 (3): 29-31.
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### **3.22.3 Other Pasteurellae**

#### **3.22.3.1 Hazard identification**

##### **Aetiological agent**

*Pasteurella gallinarum* is member of the family Pasteurellaceae.

##### **OIE List**

*P. gallinarum* is not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

Not listed in the register of unwanted organisms.

No record of the diagnosis of *P. gallinarum* in New Zealand has been found.

##### **Epidemiology**

*P. gallinarum* is occasionally reported as a cause of losses in chickens (1, 2). One incident where the organism was associated with disease in Guinea fowl is reported (3). Organisms isolated from rodents and previously reported as *P. gallinarum* have been reclassified away from that terminology (4). Reports of *P. gallinarum* in other species have not been located.

##### **Conclusion**

Based on its limited host range, *P. gallinarum* is not considered to be a potential hazard in the commodity.

##### **References**

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2. Mushin, R.; Bock, R.; Abrams, M. 1977 Studies on *Pasteurella gallinarum*. Avian Pathology. 6 (4): 415-423.
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4. Boot, R.; Bisgaard, M. 1995 Reclassification of Pasteurellaceae strains isolated from rodents. Laboratory Animals 29 (3): 314-319.

### **3.22.4 Ornithobacterium rhinotracheale**

### 3.22.4.1 Hazard identification

#### Aetiological agent

*Ornithobacterium rhinotracheale* is a pleomorphic gram-negative rod which grows slowly on agar with 5% sheep blood in an atmosphere of 5 to 10% CO<sub>2</sub> (1).

*O. rhinotracheale* was first identified in 1993 and named in 1994. Subsequent investigations showed that the bacterium had been present in turkeys since 1981 and rooks since 1983 in Germany and the organisms had also been isolated in Belgium and the United States prior to 1990 (1).

Van Empel (1), quoting others, stated that “It is quite possible that *O. rhinotracheale* infections in poultry prior to 1993 may have been wrongly attributed to viruses or to other bacteria such as *Pasteurella*, *Riemerella* - - - etc - - - .”

#### OIE List

*O. rhinotracheale* is not included in the OIE list of notifiable diseases.

#### New Zealand Status

*Ornithobacterium rhinotracheale* is listed in the register of unwanted organisms

*O. rhinotracheale* has not been identified in New Zealand. Diagnoses of both *Pasteurella* and *Riemerella* infections in poultry in New Zealand (see sections 3.21.1, 3.21.2 and 3.21.3) leave open the possibility that *O. rhinotracheale* may be present (see comments by Van Empel (1) quoted under “aetiological agent” above. Birds from an unknown number of poultry flocks in New Zealand have been tested for *O. rhinotracheale* using imported ELISA kits with negative results (Les With, Poultry Veterinary Services, quoted by N. Christensen, W.H. 2005. Review of Import Risk Analysis: Birds of the Order Passeriformes from the European Union, Draft 14 May 2005).

#### Epidemiology

*O. rhinotracheale* is widespread in poultry flocks in the absence of disease and whether it should be regarded as a primary pathogen is doubtful. It can, however, contribute to acute, highly contagious disease. Contribution to disease is influenced by environmental and management factors together with the presence of other diseases or the involvement of secondary infections. In experimental infections of turkeys, prior infection with turkey rhinotracheitis virus or Newcastle virus aggravated the effects of infection with *O. rhinotracheale*. In broiler chickens Newcastle disease virus had a similar effect, while prior infection with infectious bronchitis virus and bacteria such as *Bordetella avium* and *E. coli* have successively lesser effects. Spread by aerosol has been demonstrated and egg transmission (either transovarial or through cloacal contamination) can occur although contamination rates of egg shells and contents are very low (1, 2).

Disease incidents which have subsequently been recognised as due to *O. rhinotracheale* were observed in ducks in Hungary in 1987, broiler chickens in South Africa in 1991 and turkeys in Germany in 1991 and 1992. Subsequent investigations of culture collections revealed isolates from respiratory tracts of turkeys (1981) and three rooks (*Corvus frugilegus*) (1983) (3). Whether the rooks were diseased is not clear from the information available although most isolates covered in this report had come from birds with respiratory disease. *O. rhinotracheale* is now recognised as present and contributing to disease in South Africa, throughout Europe, in North and South America and in Asia. When the disease was first diagnosed in chickens in Japan in 1999, testing of blood samples previously collected from 1997 to 1999 confirmed that the organism had infected approximately 13% of both meat and laying birds in at least six prefectures during those years (4). In the north central United States serological testing showed infection to be present in 90 to 100% of layer flocks and 43 to 52% of pullet flocks (5).

Reports of *O. rhinotracheale* isolations have been from Galliformes (partridge, pheasant, quail, chicken, turkey, guinea fowl), Struthioniformes (Ostrich), Anseriformes (duck, goose) and Passeriformes (rook) (1). Reports which confirm association between *O. rhinotracheale* and disease, however, are restricted to Galliformes.

Although van Empel (1) proposed that *O. rhinotracheale* had spread rapidly throughout to world, this did not exclude the likelihood that the organism had been present in many parts of the world prior to its recognition and that it might continue to be distributed more widely than yet recognised.

## **Conclusion**

In the absence of confirmation that *O. rhinotracheale* is present in New Zealand, and given the ability of the organism to cause disease, this organism is considered to be a potential hazard in the commodity.

### **3.22.4.2 Risk assessment**

#### **Release assessment**

Only one report of *O. rhinotracheale* infecting passerine species (3) has been located. No diagnoses of disease in passerines or isolations of the organism from birds of that order have been reported since *O. rhinotracheale* was characterised. Although vertical transmission of the organism in gallinaceous species occurs infection rates in eggs are low. The likelihood of infection being transmitted in the commodity is considered to be negligible.

The release assessment for *O. rhinotracheale* in passerine eggs imported to New Zealand is negligible.



## Risk estimation

Based on the negligible release assessment *O. rhinotracheale* is not considered to be a hazard in the commodity.

## References

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### **3.22.5 Bordetella spp.**

#### **3.22.5.1 Hazard identification**

##### **Aetiological agent**

*Bordetella* spp. are small gram-negative rods, strict aerobes and commensals in the upper respiratory tract. They occasionally cause disease. *Bordetella avium* requires differentiation from *Alcaligenes faecalis* which is non-pathogenic. *Bordetella hinzii* (referred to as *B. avium*-like or as *Alcaligenes faecalis* type II prior to 1995) also infects birds but is non-pathogenic (1, 2).

##### **OIE List**

*B. avium* is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

*Bordetella avium* is listed in the unwanted organisms register.

*Bordetella bronchisepticum* and *B. parapertussis* are endemic in New Zealand. *B. avium* has not been identified in NZ.

##### **Epidemiology**

*B. avium* has been identified as a cause of disease in turkeys in North America, Germany and Australia. Its association with disease in Great Britain, France, Israel and South Africa has been in the company of other pathogens (2). *B. avium* causes significant economic losses in the turkey industry with high morbidity and low mortality being the norm. *B. avium* also infects chickens and is an opportunist pathogen in that species. The organism is highly contagious. It is readily transmitted between birds in close contact and will survive for up to six months in litter.(2) No reports suggesting vertical transmission of *Bordetella avium* have been located. Raffel *et al.* (3) reported isolation of *B. avium* from seven of twelve passerine species tested and from mallard ducks, a Canadian goose and a wild turkey in the eastern United States. Ribotyping of isolates from wild birds found most indistinguishable from isolates from turkeys.

##### **Conclusion**

Based on their presence in New Zealand *Bordetella bronchisepticum* and *B. parapertussis* are not considered to be hazards in the commodity.

*B. avium* is considered to be a potential hazard in the commodity.

### 3.22.5.2 Risk assessment

#### Release assessment

Although *B. avium* has been identified in passerine birds (3, 4) and their nests (5) the lack of evidence of vertical transmission in any avian species means that the likelihood of infection in imported passerine eggs is considered to be negligible.

The release assessment for *B. avium* in imported passerine eggs is negligible.

#### Risk estimation

Based on the negative release assessment it is concluded that *B. avium* is not a hazard in the commodity.

#### References

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### **3.22.6 Haemophilus paragallinarum**

#### **3.22.6.1 Hazard identification**

##### **Aetiological agent**

*Haemophilus paragallinarum* is a small gram-negative rod which may take on other shapes such as coccobacilliary or short filaments. They are mobile, facultative anaerobes (1).

##### **OIE List**

*H. paragallinarum* is not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

*Haemophilus paragallinarum* is listed in the unwanted organisms register.

The only report of *Haemophilus* sp. being isolated from respiratory disease in birds, suggestive of infectious coryza, in New Zealand is from turkey poults (2). As searches of electronic databases have failed to reveal reports of *H. paragallinarum* infection in turkeys, and it has been reported that turkeys are refractory to such infection (3), it seems that New Zealand is, most likely, free from *H. paragallinarum*.

##### **Epidemiology**

The natural host of *H. paragallinarum* is chickens and caution has been advised with the interpretation of reports of the organism from other species (1). Searches have failed to reveal reports of the finding of *H. paragallinarum* in passerine birds.

##### **Conclusion**

Based on the organism's host specificity, *H. paragallinarum* is not considered to be a hazard in the commodity.

##### **References**

1. Blackall, P.J.; Matsumoto, M. 2003 Infectious Coryza pp. 691-703. in Diseases of Poultry. Eds Saif, Y.M *et al.* 2003. Iowa State Press.
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3. Tamamoto, R. 1978 Infectious Coryza. Pp 225-232 in Diseases of Poultry. 7<sup>th</sup> Edition Eds Hofstad, M.S. *et al.* Iowa State University Press. Ames, Iowa.

### 3.22.7 Mycoplasma spp.

#### 3.22.7.1 Hazard identification

##### Aetiological agent

*Mycoplasma* spp. are micro-organisms in the class Mollicutes. They are susceptible to desiccation, heat, detergents and disinfectants but are resistant to antibiotics that act by disrupting cell wall synthesis (1).

The *Mycoplasma* spp. and their usual hosts listed by Kleven (2) are

Usual host	<i>Mycoplasma sp.</i>
Chicken	<i>M. gallinarum</i>
Chicken	<i>M. gallinaceum</i>
Chicken	<i>M. glycophilum</i>
Chicken	<i>M. iners</i>
Chicken	<i>M. lipofaciens</i>
Chicken	<i>M. pullorum</i>
Chicken	<i>M. gallorale</i>
Chicken, turkey	<i>M. synoviae</i>
Duck	<i>M. anatis</i>
Duck, goose, partridge	<i>M. imitans</i>
Goose	<i>M. anseris</i>
Pigeon	<i>M. columbinasale</i>
Pigeon	<i>M. columbinum</i>
Turkey	<i>M. gallopavonis</i>
Turkey	<i>M. iowae</i>
Turkey	<i>M. meleagridis</i>
Turkey, goose	<i>M. cloacale</i>
Turkey, chicken, house finch, other	<i>M. gallisepticum</i>
European starling	<i>M. sturni</i>
Various	<i>M. laidlawii</i>
Black vulture	<i>M. corogypsi</i>
Buteo hawk	<i>M. buteonis</i>
Griffon vulture	<i>M. gypis</i>
Saker falcon	<i>M. falconis</i>

##### OIE List

*M. gallisepticum* is included in the OIE list.

## New Zealand Status

*M. iowae* is listed in the register of unwanted organisms and has not been diagnosed in New Zealand.

*M. gallisepticum* is endemic in New Zealand (3, 4, 5).

Positive serology has been reported from routine surveillance for *M. gallisepticum* in chicken and turkeys, *M. synoviae* in chickens and *M. meleagridis* in turkeys. Clinical disease has been associated with all three *Mycoplasma* species (6). Other *Mycoplasma* spp. have not been reported from New Zealand.

The only information on the presence or absence of *Mycoplasma* spp. in native or wild birds in New Zealand is a report of an unidentified *Mycoplasma* sp. isolated from a duck (7) (it was not reported whether the duck was wild or farmed) and negative findings in 10 captive Kiwi from four properties (8). Reports of *Mycoplasma* spp. in caged or aviary birds have not been sighted.

## Epidemiology

Clinical presentation of mycoplasmosis varies with host species and *Mycoplasma* species. However, signs are predominantly the result of infection of the respiratory system. Many infections are sub-clinical. Each of the *Mycoplasma* spp. appears restricted to a limited host range and most have a host preference for a specific species. Spread between birds is by direct or indirect contact and transmission between groups occurs with fomites. Vertical transmission via eggs occurs(2). No reports of human infections with *Mycoplasma* spp. that infect birds have been located.

There is little published information on mycoplasmosis in caged birds. Spira (9) refers to its presence in budgerigars and cockatiels without any identification of *Mycoplasma* species involved, while Gaskin (10) identified *Mycoplasma* spp. in macaws, cockatiels, cockatoos and canaries (the only passerine species).

Evidence of *M. gallisepticum* and *M. synoviae* infections in wild sparrows in the vicinity of poultry farms has been reported from Taiwan (11) and Yugoslavia (12), although the *Mycoplasma* status of these poultry farms is not evident from the information in the abstracts available. Starlings proved refractory to artificial challenge with *M. gallisepticum* (13).

A number of recent reports have indicated the presence of specific species and strains of species in passerines.

*Mycoplasma iowae* – In examinations of indigenous birds in Great Britain (including 32 passeriformes), Amin (14, 15) isolated *M. iowae* from four of eight starlings (*Sturnus vulgaris*), one of three wood pigeons, one eider duck, one cormorant and one heron. The majority of these birds were obtained following road deaths or after being shot with no

history of illness. Twenty one exotic passeriformes from UK zoological gardens were examined with *M. iowae* being isolated from one *Yuhina castaniceps* and a “ruby throated Bulbul” (*Pycnonotus* sp.). Further examinations of wild birds in Great Britain (including corvids, starlings and finches) have not resulted in isolations of *M. iowae* (15).

The primary host of *M. iowae* is thought to be turkeys, and the main routes of transmission in that species are venereal and transovarial. Prevalence in age cohorts of turkeys remains low until after sexual maturity when infection is spread venereally, particularly at the time of artificial insemination (16, 17 ) which is standard practice in most commercial turkey industries. Following administration of infected semen, the organism establishes infection in the oviduct and large numbers of eggs may become infected (16, 18). *M. iowae* causes a range of clinical signs in turkeys, the main one being decreased egg hatchability (2 to 5% reduction) (17). There are differences in opinion as to the significance of *M. iowae* as a pathogen, even to extent that various views are expressed by the same author. For example Jordan in 1985 (18) and Bradbury in 2001 (19) included *M. iowae* as one of four economically important avian mycoplasmas, while in 1996 Al-Ankari and Bradbury (20) concluded that “there is insufficient data to reach any conclusions about the economic significance, if any, of *M. iowae* infections in turkeys, or in chicks or chick embryos” and in 2004 Bradbury (15) commented “- - we have never used PCR to look for this Mycoplasma (*M. iowae*) because it is no longer considered important enough to be of interest”.

The combination of the epidemiology of *M. iowae* and the normally subtle nature of clinical signs presents challenges to biosecurity provisions. It is unlikely that *M. iowae* infection would be detected clinically in turkey flocks of origin, especially if the flock is not yet in lay, and testing of the birds prior to insemination could result in negative results either if *M. iowae* infection is not present or if the prevalence of infection is low. Unless the cocks were tested prior to being used for artificial insemination it would not be possible to ensure that infected cocks were not used by the artificial inseminators, so the prevalence of infection amongst hens could be high at the time that eggs are collected. *M. iowae* infection in eggs may result in reduced hatchability but this would be detected only through testing of dead-in-shell embryos.

Therefore, although *M. iowae* has not been diagnosed in New Zealand, it is considered reasonably likely that it has been introduced through turkey hatching eggs and has remained undetected in the absence of a specific targeted surveillance programme.

*Mycoplasma gallisepticum* (house finch strain) – A specific strain of *M. gallisepticum* has been recognised in wild house finches (*Carpodacus mexicanus*) in the eastern United States and Canada since 1994 (21). RAPD (Random Amplification of Polymorphic DNA) characterisation of isolates found them to be different from any of the poultry strains or vaccine strains used in the study (22). Infections have since been recognised in a number of other species (*Carduelis tristis* (21), *Cyanocitta cristata* (22), *Carpodacus purpureus* (23), *Coccothraustes vespertinus* and *Pinicola enucleator* (24)), all of which are members of the order passeriformes and all except one (*Cyanocitta cristata* – in the Corvidae) are members of the family Fringillidae. Spread amongst wild birds appears to

be horizontal through close contact, including amongst high densities of birds at particular types of feeders (25). Observational studies over the period 1994 to 1998 lead Hartup *et al* (26) to conclude that the pattern of conjunctivitis in wild birds in the eastern United States was consistent with house finches being the primary host and other species being affected by spill-over infections during local epidemics. During the earlier stages of the epidemic of *M. gallisepticum* in house finches in the eastern United States, the prevalence of conjunctivitis varied seasonally from zero to 40% and the prevalence of infection with *M. gallisepticum* varied over a similar range (26). In some areas house finch populations declined to 40% of expected numbers (27, 28). House finches are native to the western United States and were transferred to the east in 1940. Following that transfer the species has spread through large areas of the eastern United States and Canada (29). It has also been transferred to Hawaii (29). The species is not present in Europe.

*Mycoplasma gallisepticum* in Corvidae in Scotland – Using PCR technology, evidence of *M. gallisepticum* has been found in rooks (*Corvus frugilegus*), carrion crows (*Corvus corone*) and jackdaws (*Corvus monedula*) in the vicinity of game bird rearing facilities in Scotland. Attempts to culture *M. gallisepticum* from these birds were unsuccessful. The significance of these PCR findings in the absence of either live organisms or disease is uncertain (30,31). Subsequently, *M. gallisepticum* was isolated from two choughs (*Pyrrhocorax pyrrhocorax*) with conjunctivitis in Scotland (32). The exclusivity of Corvidae species as recognised hosts (or possible hosts in the case of those with only PCR as evidence of infection) is different from the situation in North America where house finches are regarded as the primary hosts and infection of other species as ‘spill over’ infections, not sustainable in the absence of house finches. The associated clinical disease (conjunctivitis), however, is the same although there has been no report of a comparable epidemic. RAPD has been used to compare chough isolates with those from UK pheasants, chickens and turkeys and the chough isolates have appeared to be most similar to those from pheasants (32). DNA comparisons of Scottish and north American isolates have not been carried out.

*Mycoplasma sturni* – *Mycoplasma sturni* was first isolated and characterised from the conjunctiva of a European starling (*Sturnus vulgaris*) with conjunctivitis in the United States (33). Other reports of isolation of this organism in the United States have been from caged and/or aviary housed Northern mocking birds (*Mimus polyglottos*), Blue jays (*Cyanocitta cristata*) (34) and a caged Northern crow (*Corvus brachyrhynchos*) with conjunctivitis in the United States (35). In the latter incident, *M. sturni* was also isolated from in-contact, asymptomatic American robins (*Turdus migratoris*) and a European starling (*Sturnus vulgaris*).

In Scotland *M. sturni* has been isolated from dead, wild *Sturnus vulgaris*, *Corvus corone* (Carrion crow), *Pica pica* (Magpie) and *Turdus merula* (Blackbird) (30). All isolations have been from passerine species in the families Sturnidae, Turnidae and Corvidae. Whereas conjunctivitis was a common finding in the north American reports, there was no clear relationship with disease in the report from Scotland and conjunctivitis was not a



finding. The DNA similarities of *M. sturni* in north America and Scotland have not been assessed (31).

## **Conclusion**

*M. gallisepticum* (house finch strain), *M. gallisepticum* (strain isolated from Corvidae in Scotland), *M. iowae* and *M. sturni* are considered to be potential hazards in the commodity.

On the basis that they are present in New Zealand, *M. gallisepticum* (poultry strains), *M. synoviae* and *M. meleagridis* are not considered to be potential hazards in the commodity.

### **3.22.7.2 Risk assessment**

#### **Release assessment**

Although *M. iowae* is the only potential hazard for which there is specific evidence of transovarial transmission, in the absence of evidence to the contrary, this transmission route is considered possible for all.

On the basis of their presence in passerine birds in Europe and the potential to be transmitted vertically, the release assessments for *M. iowae* and *M. gallisepticum* (strain isolated from Corvidae in Scotland) and *M. sturni* in the commodity are non-negligible.

*M. gallisepticum* (house finch strain) is restricted to the United States and its primary host is, similarly, restricted. The release assessment for this organism in the commodity is negligible.

#### **Exposure assessment**

Based on information from those species in which methods of spread have been studied, *Mycoplasma* spp. are contagious organisms readily spread to other susceptible avian species through close contact or fomites. Vertical transmission may occur. On those bases the exposure assessments for *M. iowae*, *M. gallisepticum* (strain isolated from Corvidae in Scotland) and *M. sturni* are non-negligible.

#### **Consequence assessment**

*Mycoplasma iowae* – Given the conflicting views expressed on the effect of *M. iowae* in turkey flocks, it is considered that the impact of a new introduction of *M. iowae* to New Zealand would be minor. On the bases of this small impact, together with the potential for *M. iowae* to be present in New Zealand already, the consequence assessment for its effects on the poultry industry is considered to be negligible.

No reports of *M. iowae* causing disease in passeriformes or other orders of wild birds have been located.

*Mycoplasma gallisepticum* (strain isolated from Corvidae in Scotland) – The only disease considered associated with this organism has been two cases of conjunctivitis in choughs. There was a high prevalence of PCR evidence of infection in healthy birds (17).

On these bases the consequence of *M. gallisepticum* from Corvidae is considered to be negligible.

*Mycoplasma sturni* – Although Pennycott et al. (31) considered that pathogenicity of *M. sturni* isolated in Scotland could not be excluded, they were unable to identify any relationship with disease in their study. Conjunctivitis, which is the only clinical disease associated with *M. sturni* infection in the United States was not observed in the Scottish birds. Infection in both countries was limited to passerines in three families (Sturnidae, Turdidae and Corvidae). Based on the evidence available it is considered that the consequence of the introduction of the strain of *M. sturni* recognised in Scotland would be negligible.

It is concluded that the consequence assessments for *M. iowae*, *M. gallisepticum* (as recognised in Corvidae in Scotland) and of *M. sturni* (strain present in Scotland) are negligible.

### **Risk estimation**

It is concluded that *Mycoplasma* spp. are not hazards in the commodity.

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## **3.23 Intracellular bacteria**

### **3.23.1 Mycobacterium tuberculosis**

#### **3.23.1.1 Hazard identification**

##### **Aetiological agent**

*Mycobacterium tuberculosis* is the species in the Mycobacterium genus that has humans as its primary natural host. Along with other members of the genus it is an aerobic, non-motile, non-spore forming, acid-fast bacillus (1).

##### **OIE List**

*M. tuberculosis* is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

*M. tuberculosis* is not listed in the Ministry of Agriculture and Forestry unwanted organisms register. It is, endemic in NZ and predominantly a disease of humans. It is a notifiable disease under the provisions of the Tuberculosis Act 1948 administered by the Ministry of Health. Over recent years approximately 400 new cases of human tuberculosis have been diagnosed each year (2).

##### **Epidemiology**

*M. tuberculosis* is a contagious disease that does not spread rapidly but is spread, predominantly by aerosol spread, to close contacts. Tuberculosis in immigrants is sufficiently important as a means of maintaining the disease prevalence for special measures to be in place for managing the risk in immigrants from countries with a high rate of *M. tuberculosis* infection.

There are reports of birds being infected with *M. tuberculosis*.

##### **Conclusion**

*M. tuberculosis* is considered to be a potential hazard in the commodity.

#### **3.23.1.2 Risk assessment**

##### **Release assessment**

The literature contains only one report of a single passerine species (a canary) being infected with *M. tuberculosis* (3). In that article the author reported that this is the only case of *M. tuberculosis* in a non-psittacine bird. Searches of electronic databases have not provided evidence to the contrary.

The release assessment for *M. tuberculosis* in passerine eggs imported from Europe is negligible.

### **Risk estimation**

Based on the negligible release assessment, it is concluded that *M. tuberculosis* is not a hazard in the commodity.

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### 3.23.2 *Mycobacterium avium*

#### 3.23.2.1 Hazard identification

##### Aetiological agent

*Mycobacterium avium* is the species in the *Mycobacterium* genus that has birds as its primary natural host. Along with other members of the genus it is an aerobic, non-motile, non-spore forming, acid-fast bacillus (1). Because of similarities in cultural characteristics, isolates of *M. avium* or *Mycobacterium intracellulare* are commonly reported as “*M. avium* complex”. Within that complex there are over 25 serotypes, three of which are fully pathogenic to birds. The other serotypes are classified within the species *M. intracellulare* and infect pigs and other species with soil and water as the likely source of organisms (2).

##### OIE List

Avian tuberculosis is included in the OIE list of notifiable diseases.

##### New Zealand Status

*M. avium* is not listed in the register of unwanted organisms.

*M. avium* is endemic in NZ birds (2, 3) and causes some of the tuberculosis infections of deer (2). Most cases occur in older free range fowls (4). Diagnoses of avian tuberculosis in other species have been reported from a captive Kiwi (5), a harrier hawk (6), ostriches (7), Fischer lovebirds (8) and peacocks (9). Such diagnoses are treated as routine and culture to confirm the specific organism involved is seldom done.

##### Conclusion

On the basis that *Mycobacterium avium* is endemic in New Zealand it is not considered to be a potential hazard in the commodity.

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### **3.23.3 Other Mycobacteria**

#### **3.23.3.1 Hazard identification**

##### **Aetiological agent**

*Mycobacterium genavense* is the most commonly reported mycobacterial infection of passerine birds. Reports of Mycobacterial species other than *M. genavense*, *M. avium* complex or *M. tuberculosis* are sufficiently uncommon that they will not be considered further in this risk analysis.

*Mycobacterium genavense* is a slow-growing, fastidious mycobacterium that has become of greater interest since its emergence as a pathogen in immuno-compromised people, particularly those with AIDS (1).

##### **OIE List**

*M. genavense* is not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

*M. genavense* is not listed in the register of unwanted organisms.

*M. genavense* has been isolated three times from one human patient in New Zealand. The patient was immuno-compromised, was originally from Africa and had been in New Zealand from 14 years prior to recognition of the infection. The report on this case (2) states that it is thought that the infection was acquired in NZ and that, with the technology now available, further cases of *M. genavense* infection will be recognised in NZ in the future.

##### **Epidemiology**

*M. genavense* was first reported from disseminated infection in human AIDS patients in 1990 (1) and there has been a considerable number of reports since that time (3, 4, 5, 6).

It is also since the early 1990s that diagnoses of *M. genavense* as a pathogen of birds have emerged.

1. Hoop *et al.* (1) achieved 48 mycobacterial isolates suitable for species identification from a total of 5,345 necropsies of pet birds . 34 of those isolates were identified as *M. genavense*.
2. Portaels *et al* (7) diagnosed *M. genavense* as the cause of disease in 27 birds (including 12 passerines) dying at the Antwerp zoo between 1983 and 1994.
3. Of 253 pet birds examined for Mycobacteria in Switzerland 26 were positive and 19 of those were found to be infected with *M. genavense*.(8) and
4. There are several other reports of *M. genavense* infection in individual birds.

The source of infection for birds and humans is not known but is suspected to be either soil or water related.

## Conclusion

*M. genavense* is present in New Zealand, and is not subject to any official control programme, it is not considered to be a potential hazard in the commodity.

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## 3.24 Other bacteria

### 3.24.1 *Francisella tularensis*.

#### 3.24.1.1 Hazard identification

##### Aetiological agent

*Francisella tularensis*, the cause of Tularaemia, is a small gram-negative intracellular bacterium. It is an obligate aerobe and has fastidious growth requirements.(1) *F. tularensis* type A is, effectively, restricted to North America. Type B is found through much of Europe and Asia and is less virulent than type A (2).

##### OIE List

*F. tularensis* is not included in the OIE list.

##### New Zealand Status

*F. tularensis* is listed in the unwanted organisms register and is exotic to New Zealand.

##### Epidemiology

Much of the following information is from a review by Tarnvik *et al* (2). Human tularaemia, attributable to *F. tularensis* type B, occurs throughout most of Europe but with marked regional differences and with major differences at different times in history. A major outbreak occurred in Russia during world war II and lesser epidemics occurred in Kosovo in 2000 and 2003. Repeated outbreaks occur in Finland and central Sweden and occasionally in other locations. There is a low level of tularaemia through most of Europe but with an absence of reports of the disease in Portugal and the British Isles (2).

Past proposals that wild mammals and /or birds form the reservoir(s) for *F. tularensis* are now questioned. Geographic patterns of the occurrence of tularaemia do not coincide with those expected if infection is spread by birds and evidence for persistence of *F. tularensis* in birds has not been found. There is epidemiological evidence supporting the proposal that the reservoir for the organism is in water or water associated (2).

Birds are affected by tularaemia and infection, commonly, results in death. Species most commonly affected are Galliformes species, waterfowl, scavengers and predatory wild birds (3). This distribution is consistent with birds being infected from either water-associated sources or from feeding on other infected animals including small mammals that are also known to become infected.

##### Conclusion

*F. tularensis* is considered to be a potential hazard in the commodity.

### 3.24.1.2 Risk assessment

#### Release assessment

Searches of the literature have revealed only one report of natural infection in a passerine bird. That was in a raven (*Corvus corax*) (4) which included carrion and mammals in its diet. Experimental infection of three hooded crows (*Corvus corone cornix*) was followed by death of one bird during the experimental period and survival of the others until they were killed at 14 to 77 days post-inoculation. *F. tularensis* could not be recovered from any of these birds (5).

Based on the scarcity of reports, the special conditions of the one report and the apparent non-carriage of infection beyond short periods, the release assessment for *F. tularensis* in eggs of the order passeriformes from Europe is negligible.

#### Risk estimation

Based on the negligible release assessment *F. tularensis* is not considered a hazard in the commodity.

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### **3.24.2 Megabacteria**

#### **3.24.2.1 Hazard identification**

##### **Aetiological agent**

“Megabacteria” are large gram-positive rods around 20 times the size of most other bacteria. They have varying susceptibility to antibiotics, are present in small numbers in normal budgerigars and in large numbers in proventriculus of birds supposedly affected by them (1). The organism(s) has not been fully characterised and has no defined taxonomic status. Oliver (2) suggested that the organism may have a nucleus and may be branching. Phalen (3) suggests that the organism is a yeast. This is consistent with the apparent efficacy of Amphotericin B in management of “Megabacteriosis” (4).

##### **OIE List**

Megabacteria are not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

Not listed in the register of unwanted organisms.

“Megabacteria” have been reported from budgerigars and canaries in New Zealand with clinical disease similar to that associated with “megabacteriosis” in other countries (5,6,7).

##### **Epidemiology**

Megabacteriosis is, predominantly, associated with proventriculitis in budgerigars (1) but it has also been associated with similar disease in canaries (8, 6, 9, 10), wild goldfinch and Siskins in the Netherlands (11), wild greenfinch and Siskins in Britain (12) and zebra finches in Italy(13). Reports of clinical disease also come from a number of non-passerine birds. “Megabacteria” have been found in either the faeces or the proventriculus of apparently healthy budgerigars (14, 15) in Australia and Great Britain and in 60 % of wild trapped goldfinches and four of five wild trapped cockatoos in Australia (16). The epidemiology of “Megabacteriosis” is not known. Scanlon and Graham (17) suggested that the organism is part of the normal gut flora of birds. Others consider it to be a pathogen.

Oliver (2) suggested that there might be strain differences in Megabacteria reflected in differences in pathogenicity of different isolates in different bird species. Talltree (18) stated that it is thought that Megabacteria were introduced to Australia with budgerigars imported from England in 1989-1990. The subsequent finding of Megabacteria in a range of species from different orders in Australia might be explained by multiple imports of Megabacteria of different strains or by the ability of the one strain to infect a range of species. The number of reports in which megabacterial infections of both passerine and

psittacine birds are reported is notable. These have come from New Zealand (5), Turkey (9), Brazil (10), Italy (13) and Australia (16). It is likely that, if strain differentiation of Megabacteria exists, strains have the ability to infect multiple avian species. It is also likely that Megabacteria are normal gut inhabitants and that if they do exert pathogenic effects it is as a result of particular environmental conditions or as secondary invaders in association with another organism.

## Conclusion

On the basis that Megabacteria have been reported from both canaries and budgerigars in New Zealand they are not considered to be a potential hazard in the commodity.

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### **3.24.3 Gram positive contaminants (Streptococci/Staphylococci)**

#### **3.24.3.1 Hazard identification**

##### **Aetiological agent**

Organisms discussed in this section are *Streptococcus* spp. and *Staphylococcus* spp.

Staphylococci commonly isolated from birds include *S. aureus*, *S. epidermidis* and *S. hyicus*. *S. aureus* is the main species recognised as a pathogen in poultry although *S. hyicus* has been associated with blepharitis and osteoarthritis in chickens and turkeys (1). Biotyping, phage typing and/or genetic fingerprinting can be used to differentiate avian pathogenic strains and geographic areas of origin. Coagulase positive strains are considered to be pathogenic (1).

Streptococci recognised as causing disease in birds include *S. zooepidemicus* (*S. gallinarum*), *S. bovis* and *S. dysgalactiae* (2). The organism causing septicaemia and death in pigeons and previously identified as *S. bovis* is now designated *S. gallolyticus*. Standard diagnostic laboratory techniques are not likely to differentiate this organism from *S. bovis* (3)

##### **OIE List**

These organisms are not included in the OIE list.

##### **New Zealand Status**

These organisms are not included in the register of unwanted organisms. Numerous species of *Streptococcus* and *Staphylococcus* are present in NZ. Frequently identification of these organisms attracts little attention as they are assigned “secondary”, “opportunistic” or “contaminant” roles. Typing of *Staphylococcus aureus* beyond coagulase positive or negative is seldom (if ever) carried out (Personal observation).

*Staphylococcus* spp. – *S. aureus* has been associated with gangrenous dermatitis in chickens (4) and there is one report of an epidemic in day-old chicks (5). *S. aureus* has been reported from a young fowl with musculoskeletal disease (6) and a secondary role was assigned to *S. aureus* isolated from cases of tenosynovitis in broiler chickens (7).

*S. epidermidis* has been reported from yolk sac infections in imported ostrich eggs (8) and from cases of mastitis in cattle (9)

*S. hyicus* is present in New Zealand and has been, particularly, associated with exudative epidermitis in pigs (10) and it also contributes to bovine mastitis (9).



*Streptococcus* spp. – *S. dysgalactiae* and *S. bovis* are present in New Zealand and recognised as causes of bovine mastitis (11) and *S. zooepidemicus* is also present and particularly associated with reproductive (12) and respiratory (13) disease in horses. It is likely that the organism causing bovine mastitis and other diseases in animals and identified as *S. bovis* will be shown to be *S. gallolyticus*. This organism causes disease in pigeons but has not been reported from other avian species.

## Conclusion

Since the organisms are endemic in New Zealand, neither *Staphylococcus* spp. nor *Streptococcus* spp. are considered to be potential hazards in the commodity.

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## 3.25 Spirochetes

### 3.25.1 *Borrelia anserina* (Avian spirochaetosis)

#### 3.25.1.1 Hazard identification

##### Aetiological agent

*Borrelia anserina* is a large spirochaete, labile in the environment, and sensitive to desiccation. As with other *Borrelia* spp., it is transmitted by arthropod vectors (1).

##### OIE List

*Borrelia anserina* is included in the OIE list.

##### New Zealand Status

*Borrelia anserina* is listed in the register of unwanted organisms.

Neither *B. anserina* nor *Argas* spp. of ticks have been recorded in New Zealand.

##### Epidemiology

*Borrelia anserina* is the cause of avian spirochaetosis, an acute disease of chickens, turkeys, pheasants, geese and ducks. It is reliant on *Argas* spp. of ticks as vectors (1). The major vector for *B. anserina* is *Argas persicus* (the fowl tick) (2) which is widespread in tropical areas of the world, with *Argas africanus* (in Africa) (3) and *A. sanchezi* (in the Americas) (4) also recorded. Baker (2) also includes *Argas reflexus* (the pigeon tick) as a vector of *B. anserina*.

Although reports of *B. anserina* in passerine birds have not been located, *A. persicus* has been found on canaries (2), sparrows (*Passer domesticus*) (5, 6) and barn swallows (*Hirundo rustica*) (6) while *A. africanus* has been reported from several species of swallow (*Hirundo* spp.) and mocking cliffchat (*Myrmecocichla cinerea*) (7).

##### Conclusion

*B. anserina* is considered to be a potential hazard in the commodity.

### 3.25.1.2 Risk assessment

#### Release assessment

Literature searches have not identified reports of *B. anserina* in Great Britain, The Netherlands, France, Italy, Portugal or German. Evans *et al.* (8) did not include *Argas persicus* (the fowl tick) in their record of ticks in the British Isles. Searches of electronic databases have not indicated the presence of *A. persicus* in European countries listed above with exceptions of Italy (9) (where its presence is related to pathways of migratory birds) and Spain (10). This is consistent with Baker's statement (2) that *A. persicus* is largely limited to countries located between latitudes 40 degrees north and 40 degrees south. Neither *A. africanus* nor *A. sanchezi* have been reported in Europe.

No reports suggesting vertical transmission of *B. anserina* in birds have been located.

The likelihood of *B. anserina* infecting passerine birds in Europe is negligible.

#### Risk estimation

It is concluded that *B. anserina* is not a hazard in the commodity.

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### **3.25.2 Borrelia burgdorferi (Lyme disease)**

#### **3.25.2.1 Hazard identification**

##### **Aetiological agent**

*Borrelia burgdorferi* is a large spirochaete, labile in the environment, and sensitive to desiccation. As with other *Borrelia* spp., it is transmitted by arthropod vectors. A number of genotypes (genospecies) have been identified in the USA and Europe (1)

##### **OIE List**

*B. burgdorferi* is not included in the OIE list of notifiable diseases..

##### **New Zealand Status**

*Borrelia burgdorferi* is listed in the register of unwanted organisms.

*Borrelia burgdorferi* has not been identified in New Zealand, nor have the vectors upon which it is reliant.

##### **Epidemiology**

Lyme disease, caused by *Borrelia burgdorferi*, affects dogs, horses, cattle and humans. These species are incidental hosts to an organism that normally cycles between reservoir hosts (small mammals including rodents, birds and lizards) and ticks which act as vectors. The usual hosts for adult ticks are larger mammals which are maintenance hosts for the ticks but are not reservoir hosts for *Borrelia*. The only competent vectors are ticks, generally of the *Ixodes* genus. In Europe, the main vector is *Ixodes ricinus*, in the eastern United States it is *I. scapularis*, in the western US it is *I. pacificus* and in Eurasia it is *I. persulcatus* (1). The distribution of *Ixodes* spp. ticks that are able to transmit the agent of Lyme Disease spreads in a broad band across the entire northern hemisphere (2)

At least 300 species of animals, including birds, are recognised as hosts of *I. ricinus* in Europe (3).

##### **Conclusion**

*B. burgdorferi* is considered to be a potential hazard in the commodity.

#### **3.25.2.2 Risk assessment**

##### **Release assessment**

At least 14 bird species from the order Passeriformes are considered to be competent reservoirs for genospecies of *B. burgdorferi* in Europe (2). Transmission is dependent

upon competent tick vectors. Searches of the literature have not revealed any suggestion that *B. burgdorferi* infects birds eggs or is transmitted vertically in birds.

The release assessment for *B. burgdorferi* in passerine eggs is negligible.

### **Risk estimation**

Based on the negligible release assessment, it is concluded that *B. burgdorferi* is not a hazard in the commodity.

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### **3.25.3 Brachyspira spp.**

#### **3.25.3.1 Hazard identification**

##### **Aetiological agent**

*Brachyspira pilosicoli* is found in the gut of birds and associated with clinical disease. *Brachyspira* (formerly *Serpulina*) species are attracting considerable attention as recently classified inhabitants of the gut of humans, pigs, chickens and other species which are associated with disease commonly termed intestinal (or colonic) spirochaetosis. The genus is differentiated from other spirochaetes on the basis of patterns of haemolysis on blood agar plates and tests for indole production (1).

##### **OIE List**

*Brachyspira* spp. are not included in the OIE list of notifiable diseases.

##### **New Zealand Status**

*Brachyspira* spp. are not listed in the register of unwanted organisms.

*Brachyspira* (*Serpulina*) *hyodysenteriae*, *B. pilosicoli* and *B. innocens* have been isolated from pigs with enteric disease in New Zealand (2, 3).

##### **Epidemiology**

It appears that the first descriptions of avian intestinal spirochaetosis date back to the very early 1900s. More recently, there has been an increased research effort and recognition of the role that *Brachyspira* spp. play in contributing to intestinal pathology in birds and other species. In a dedicated issue of CAB International, Animal Health Research Reviews, Duhamel (4) has reviewed the comparative pathology and pathogenesis of colonic spirochaetosis, Stephen and Hampson (5) have reviewed spirochaete infections in chickens and Jansson *et al.* (6) have provided a review of *Brachyspira* infections in birds together with the results of a study in Swedish game birds.

A number of the *Brachyspira* species have wide host ranges with *B. pilosicoli* being found in natural infections and causing disease in chickens, pigs, dogs, opossums, monkeys and humans (4). A high proportion of feral water birds frequenting a zoological garden in Perth, Australia were infected with *B. pilosicoli* without any association lesions and it appears likely that water birds may be natural reservoirs for this organism (4). Similarly high proportions of birds on game farms in Sweden were found infected. Whether this is a reflection of infection rates in wild birds of the same species is not known. It is suggested that water may act as a source of infection for birds and other species (6).

*B. hyodysenteriae* (or a very similar organism), a well recognised pathogen in pigs, has been recovered from a broiler parent flock in Australia, a rhea in the United States, and from a mallard duck (6).

As increased research attention is applied to *Brachyspira* pathogenesis and the characteristics of the organism, increasing numbers of species are being named (5, 6, 7).

No reports of investigations of *Brachyspira* infections in passerine birds have been located but, given the range of species so far investigated and found to be infected positive findings from passerines seem likely.

No reports suggesting infection of the eggs of birds or of vertical transmission have been located.

With the diagnosis of *B. hyodysenteriae*, *B. pilosicoli* and *B. innocens* in pigs in New Zealand, infection of avian species is likely. It is also very likely that, were investigations into the spirochaetal flora of the intestinal tracts of animals in New Zealand to be pursued, an increased range of *Brachyspira* species and hosts for the genus would be identified.

## Conclusion

Based on *Brachyspira* spp. being endemic in New Zealand they are not considered to be a potential hazard in the commodity.

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## **3.26 Rickettsial agents**

### **3.26.1 *Coxiella burnetii***

#### **3.26.1.1 Hazard identification**

##### **Aetiological agent**

Q fever is a zoonotic disease caused by the rickettsia *Coxiella burnetii*.

##### **OIE List**

Q fever is included in the OIE list.

##### **New Zealand Status**

Q fever (*Coxiella burnetii*) is exotic to New Zealand and is listed in the unwanted organisms register as notifiable organisms.

##### **Epidemiology**

Q Fever is widely distributed throughout the world and found in a wide variety of animals and birds. Q fever has been associated with a large number of species of ticks from several genera (1), however, the exact role that ticks play in transmission is unclear and it has been suggested that the disease is more likely to be spread by inhaling dust contaminated with the agent derived from placentas of animals that have aborted (2). Others have suggested tick faeces in dust as a source of infection. The infection can induce abortion and gynaecological disorders in cows, ewes and goats. It can also, sometimes, be isolated from placentas from normal births. In humans, it causes a febrile influenza-like condition, pneumonia, hepatitis and endocarditis (2). Humans at most risk are those in occupational groups working with animals and in slaughter plants (3).

New Zealand is one of the very few countries from which Q fever has not been reported.

Q fever is known to infect a number of species of both birds and insects (1). Experimental inoculation of six month old hens resulted in the establishment of infection and the development of antibodies. There were no signs of disease (4). There are no reports of disease or mortalities in birds or insects due to infection with *Coxiella burnetii*.

##### **Conclusion**

*Coxiella burnetii* is considered to be a potential hazard in the commodity.

### 3.26.1.2 Risk assessment

#### Release assessment

Serological and/or cultural evidence of infection by *Coxiella burnetii* has been found in a large number of species of birds. Such reports have included several species of the order passeriformes (5, 6, 7, 8). Prevalence rates have varied with geographic location and bird species from 2% to 68%. Sethi et al. (9) attempted to demonstrate egg transmission of *Coxiella burnetii* but were unsuccessful. No reports suggesting that vertical transmission through the eggs of birds plays a role in the epidemiology of *Coxiella burnetii* have been located.

The likelihood of *Coxiella* infection being transmitted through passerine eggs imported to New Zealand from Europe is negligible. Therefore, the release assessment is negligible.

#### Risk estimation

Based on the negligible release assessment it is concluded that *Coxiella burnetii* is not a hazard in the commodity.

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### **3.26.2 Cowdria ruminantium**

#### **3.26.2.1 Hazard identification**

##### **Aetiological agent**

*Cowdria ruminantium* is a Rickettsial organisms targeting vascular endothelium and macrophages.

##### **OIE List**

*C. ruminantium* is not included in the OIE list.

##### **New Zealand Status**

*C. ruminantium* is listed in the register of unwanted organisms.

*C. ruminantium* has not been diagnosed in New Zealand.

##### **Epidemiology**

*C. ruminantium* is the cause of heartwater, a non-contagious disease of ruminants in Africa and parts of the Caribbean. Severity of the disease varies with species with introduced species such as cattle, sheep and goats being most severely affected and some native species being apparently unaffected. The infectious agent is transmitted by ticks of the genus *Amblyomma* (1). While *Amblyomma spp.* are known to infest helmeted guineafowl and ostriches, neither of these species has been shown to be infected by *C. ruminantium* (2,3).

Heartwater does not occur in Europe or Asia or in the Americas with the exception of parts of the Caribbean. Nor does it occur in Australasia. Reports of *Amblyomma spp.* infesting passerine birds have not been located.

##### **Conclusion**

*C. ruminantium* is not considered to be a potential hazard in the commodity.

##### **References**

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452.

### **3.26.3 Aegyptianella pullorum**

#### **3.26.3.1 Hazard identification**

##### **Aetiological agent**

The *Aegyptianella* genus is classified in the *Anaplasmataceae*. *A. pullorum*, *A. botuliformis* and an unidentified *Aegyptianella* sp. have been reported from birds.

##### **OIE List**

*Aegyptianella* are not included in the OIE list.

##### **New Zealand Status**

*Aegyptianella* spp. are listed in the register of unwanted organisms.

##### **Epidemiology**

Aegyptianellosis causes anorexia, diarrhoea and anaemia in affected birds (1).

*A. pullorum* is recognised in Africa, Asia and southern Europe and has been reported from chickens, geese, ducks, quail and ostrich. The organism is spread by ticks with the recognised vectors being *Argas persicus* and *Argas walkerae* (1). *A. pullorum* was reported from blood samples from wild turkey in Southern Texas (2). Corroborative reports of *A. pullorum* in the Americas have not been located. The only other report located of an *Aegyptianella* spp. being found in birds from the Americas is from a single *Amazona aestiva* (*Psittaciformes*) examined at the time of importation into Great Britain (3). Both the CAB abstract of Gothe (4) (full publication not available at this time) and the Merk Veterinary Manual (5) include *Argas radiatus* and *Argas sanchezi* as vectors of *Aegyptianella pullorum*. These ticks are American species and searches of available literature have not enabled confirmation of their roles as vectors.

*Aegyptianella botuliformis* was first reported from helmeted guineafowl (*Numida meleagris*) in the Kruger National Park (6) and subsequently from a range of game species (*Francolinus* spp. and *N. meleagris*) from four locations in southern Africa (7). Detection rates in most species sampled were between 18% and 52%. *Argas* spp. were thought to be the vectors.

The only report located of infection of a passerine species was of a previously undescribed *Aegyptianella* species, from an archived Giemsa stained blood smear from a mountain fulvetta (*Alcippe peracensis*) from Malaysia and held in the collection of the International Reference Centre for Avian Haematozoa in the United Kingdom (8).

## Conclusion

*Aegyptianella pullorum* is considered to be a potential hazard in the commodity.

Neither *A. botuliformis* nor other recognised *Aegyptianella* spp. of birds are considered to be potential hazards in the commodity.

### 3.26.3.2 Risk assessment

#### Release assessment

Literature searches have not identified reports of *Aegyptianella pullorum* in Great Britain, The Netherlands, France or Germany. Tarello (9) reported *Aegyptianella*-like organisms in a sick bittern in Italy and similar findings in two birds of prey lead Tarell and Riccieri (10) to suggest that Aegyptianellosis might enter Italy seasonally with migratory birds. Evans *et al.* (11) did not include *Argas persicus* (the fowl tick) in their record of ticks in the British Isles. Searches of electronic databases have not indicated the presence of *A. persicus* in European countries listed above with exceptions of Italy (12) (where its presence is related to pathways of migratory birds) and Spain (13). This is consistent with Baker's statement (14) that *A. persicus* is largely limited to countries located between latitudes 40 degrees north and 40 degrees south.

The only report located of *Aegyptianella* spp. in a passerine bird was in an avian sample from Malaysia.

No reports suggesting that vertical transmission in birds can take place or might play a part in the epidemiology of *Aegyptianella pullorum* have been located.

The likelihood of passerine eggs imported from Europe carrying *Aegyptianella pullorum* is negligible.

The release assessment is negligible.

#### Risk estimation

Based on the negligible release assessment, it is concluded that *Aegyptianella pullorum* is not a hazard in the commodity.

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### 3.26.4 Other Rickettsia

#### 3.26.4.1 Hazard identification

##### Aetiological agent

Organisms considered in this section are those of the genera *Ehrlichia*, *Neorickettsia*, *Rickettsia*, *Anaplasma*, *Eperythrozoon* and *Haemobartonella*.

##### OIE List

These organisms/diseases are not included in the OIE list.

##### New Zealand Status

*Ehrlichia* spp., *Rickettsia* spp. and *Anaplasma* spp. are listed in the register of unwanted organisms and have not been diagnosed in New Zealand.

*Haemobartonella felis* (1), *H. canis* (2), *Eperythrozoon ovis* (3), *E. wenyoni* (4) and either *E. suis* or *E. parvum* (which may be the same species) (2) have been diagnosed in New Zealand.

##### Epidemiology

The only reports discovered indicating infection of birds with organisms covered in this section were of *Rickettsia tsutsugamushi* (syn. *R. orientalis*) in chickens placed in an area where scrub typhus was endemic in Japan (5), in migratory birds in an area of Russia (6) and an unidentified *Rickettsia* sp. isolated from a parrot (7). *R. tsutsugamushi* is restricted to Asia, parts of Australia and some Pacific Islands (8).

The movement of vector competent ticks on birds migrating between Africa and Eurasia was identified by Kaiser *et al.* (9) as a potential means of spread of vector dependent pathogens, including *Rickettsia* species. Using PCR technology, the DNA of *Anaplasma phagocytophilum* (Syn. *Ehrlichia phagocytophilum*), the cause of human Granulocytic ehrlichiosis, has been identified in *Ixodes ricinus* ticks on migratory passerine birds at a stopover site in southern Sweden (10) suggesting that birds may play a role in the geographic dispersal of the micro-organism. Daniels *et al* (11), also using PCR methodology, found evidence of *A. phagocytophilum* in *Ixodes scapularis* removed from birds, including passerines, in New York State and concluded that it was possible that birds may be acting as reservoirs for *A. phagocytophilum* and that carriage of ticks may result in the seeding of infections into new areas.

No reports suggesting vertical transmission of rickettsia organisms in birds have been located.

## Conclusion

Based on the limited evidence that any *Ehrlichia*, *Neorickettsia*, *Rickettsia*, *Anaplasma*, *Eperythrozoon* or *Haemobartonella* spp. infect birds, the limited geographic distribution of *R. tsutsugamushi* and the lack of evidence that any of the Rickettsial organisms can be transmitted vertically in birds, these organisms are not considered to be potential hazards in the commodity.

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## 3.27 Fungi and yeasts

### 3.27.1 Hazard identification

#### Aetiological agent

Fungi and yeasts considered are:

- Dermatophytes - *Microsporum* spp. and *Trichophyton* spp.,
- *Histoplasma* spp.,
- *Cryptococcus* spp.,
- *Candida* spp.,
- *Aspergillus* spp.,
- Zygomycetes
  - *Absidia* spp,
  - *Mortierella* spp.,
  - *Mucor* spp. and
  - *Rhizopus* spp.
- *Microspora*

#### OIE List

Epizootic lymphangitis (which is caused by *Histoplasma capsulatum* var. *farciminosum*) is included in the OIE list.

#### New Zealand Status

*Histoplasma farciminosum* is listed in the register of unwanted organisms.

#### Epidemiology

Dermatophytes – Kunkle (1) recognised only *Microsporum gallinae* as a primary pathogen causing “Favus” in birds whereas Reavill (2) identified *M. gypseum*, *Trichophyton mentagrophytes*, *T. megninii* and *T. verrucosum* in addition. *T. megninii* is anthropophilic and rarely infects animals (3). Disease attributed to *M. gallinae* is most commonly reported from chickens (2) although neither Kunkle (1) nor Reavill (2) recognise dermatophytosis, of any cause, as a serious disease in birds. Searches of the literature have failed to reveal reports of *M. gallinae* in birds of the order Passeriformes. Keratinophilic fungi of the genera *Chrysosporum*, *Malbranchea* and *Scopulariopsis* have been isolated from the feathers of healthy birds including passerines (4, 5). These organisms are not recognised as pathogens in birds but small numbers of infections in humans have been reported. The organisms are soil saprophytes and birds are not considered to play a significant role in their epidemiology (5).

*Histoplasma capsulatum* – *Histoplasma capsulatum* var. *farciminosum* is closely related to *Histoplasma capsulatum* var. *capsulatum* and is differentiated mainly on the basis of its pathogenicity in horses (6). This organism is a saprophytic mould with cosmopolitan

distribution (7) especially in soil contaminated with bird faeces (8). *H. capsulatum* causes an infectious disease but is not contagious (1). Birds appear to have an innate resistance to infection which may be due to their high body temperature (7) and reports of earlier diagnoses of Histoplasmosis in birds have been questioned by Smith (9). The association of *H. capsulatum* with bird faeces is thought to be due to the effects of the bird faeces on the soil environment allowing the organism to proliferate, not because the organism is excreted by the birds (8). Although *H. capsulatum* is listed in the Landcare Research Institute fungal database as “present in the region” (10), there are no reports of identification of the organism from animals and the only reports of human cases of disease are from people who have travelled internationally (11). Such diagnoses were made in 1998, 2002 and 2003 (12).

*Cryptococcus* spp – *Cryptococcus neoformans* var. *neoformans* has a cosmopolitan distribution being recognised in all continents. *C. neoformans* var. *gattii* has an ecological niche in certain eucalyptus trees. Other varieties and species have restricted distributions and are not associated with disease. While *C. neoformans* causes sporadic disease in many animal species, including humans, disease in birds is uncommon. The organism infects the gut of birds and contaminated soil is the most common source of infection (13). In New Zealand *C. neoformans* has been reported causing disease in humans (14, 15), cats and dogs (16). *C. neoformans* var. *gattii* was diagnosed as the cause of disseminated lesions in a captive North Island kiwi (*Apteryx australis mantelli*) some months after eucalyptus mulch had been placed in its enclosure (17).

*Candida* spp – *Candida albicans* is the most common member of the genus and is distributed world wide. Other species include *C. glabrata*, *C. krusei*, *C. kefyr*, *C. guilliermondii*, *C. parapsilosis*, *C. tropicalis*, *C. lusitanae* and *C. viswanatii*. The latter species is restricted to India whereas all others have wide distributions. *Candida* spp. are common in the intestinal tract, oropharynx, vagina and skin of many animals. They are also widespread in the environment. Most instances of disease associated with *Candida* spp. arise from endogenous infections in animals that become stressed and/or immunocompromised (18). In New Zealand, *C. albicans*, *C. parapsilosis*, *C. tropicalis*, *C. krusei* and others have been isolated from humans (19). *C. guilliermondii*, *C. albicans*, *C. parapsilosis*, *C. famata* and *C. tropicalis* have been reported from bovine sources and *C. albicans*, *C. tropicalis* and *C. krusei* from birds (20).

*Aspergillus* spp – *Aspergillus* spp. are widespread soil inhabitants found in dust, decomposing organic matter and dispersed in the air. *A. fumigatus* is the species most commonly found as an opportunist pathogen but *A. flavus*, *A. nidulans*, *A. niger* and *A. terreus* can also be found associated with disease (21). Aspergillosis has been diagnosed in a wide range of species in New Zealand including native birds such as stitchbirds (22, 23), yellowheads (24) and penguins (25). Bovine abortions and sporadic cases of pneumonia, encephalitis and mastitis (26), along with other disease incidents, have also been attributed to *Aspergillus* species.

*Zygomycetes* – *Absidia* spp., *Mortierella* spp., *Mucor* spp. and *Rhizopus* spp. are fast growing terrestrial fungi largely saprophytic in plant debris and soil.

- ***Absidia corymbifera*** (syn. *A. ramosa* (27)) is the only species in that genus recognised as causing disease in animals (28). This organism is present in New Zealand and is a common contaminant on pathological specimens submitted from animals in the field for diagnostic examination (29).
- ***Mortierella wolfii*** is the only species in this genus recognised as causing disease in animals (28). This organism is present in New Zealand and a cause of bovine abortion and pneumonia (29).
- ***Mucor* spp.** *M. circinelloidea*, *M. indicus* and *M. ramosissinus* are, variously, recognised as pathogens in humans, cattle swine and birds (28). From New Zealand there is a large number of reports of *Mucor* species associated with disease in humans (30, 31) and animals (32, 33, 34 (a selection of references only)) including birds (35). In none of the reports found were the species of *Mucor* determined. From the range of disease entities and the range of species affected, it is assumed that a number of the pathogenic *Mucor* species are present in New Zealand.
- ***Rhizopus* spp.** *Rhizopus* spp. are saprophytic in soil and on plant material and fruit. Some members of the genus are used in fermentation processes, others have pathogenic potential. *R. microsporus* var. *microsporus* and *R. microsporus* var. *rizopodiformis* are considered to be a causes of abortion in pigs and cattle (28). In New Zealand *Rhizopus* spp. (not identified to species level) have been identified as associated with disease in humans (36, 37) and as part of a mixed infection in a case of necrotising pneumonia in an ostrich (35). Fairley (20) commented that *Rhizopus* spp. are part of a complex with *Mucor* and *Absidia* that may be associated with bovine abortions, are seldom cultured, are probably secondary invaders and are generally reported from diagnostic laboratories as “*Mucor/Absidia* species”.

**Microsporidia** – Microsporidia were previously classified as protozoa but are now recognised as fungi. They are obligate intracellular organisms, in vertebrates and invertebrates, that can be transmitted horizontally and vertically (38, 39). Unidentified Microsporidia have been reported from peach-faced lovebirds (*Agapornis roseicollis*) (40, 41), masked lovebirds (*Agapornis personata*) (41), Fischer’s lovebirds (*Agapornis fischeri*) (41), horned puffin chicks (*Fratercula corniculata*) (42) and an amazon parrot (*Amazona ochrocephala oratrix*) (43). *Encephalitozoon* spp. were reported from an *Amazona ochrocephala* (44) *E. hellem* has been reported from budgerigars (*Melopsittacus undulatus*) (45), peach-faced love birds (*Agapornis roseicollis*) (46) and a Gouldian finch (*Erythrura (Chloebeia) gouldiae*) (47). *Enterocytozoon bieneusi* has been reported from chickens (48). Evidence of natural vertical transmission of *Encephalitozoon cuniculi* in chickens has been reported (49).

The reports of Microsporidia from birds have come from healthy birds (41, 46) and birds dying with non-specific disease (42), keratoconjunctivitis (43), diarrhoea and respiratory disease (44), concurrent with Megabacteriosis (45), inadequate growth rates (48), deaths in nestlings (47) and death of embryos in shells (49) suggesting that, as in humans (39, 49), these microsporidia behave as opportunist pathogens.

Disease associated with microsporidial infection in humans occurs mainly, but not exclusively, in immunocompromised (HIV or organ transplant) patients and *Enterocytozoon bieneusi*, *Encephalitozoon* spp., *E. cuniculi* and *E. hellem* have all been associated with such disease (39, 50, 51). The extent of carriage by immunocompetent humans is unclear but studies have shown infection rates of 3.3 to 10% in travellers, 5.9 to 17.4% in children with and without diarrhoea and 17% in elderly persons (39).

*Enterocytozoon bieneusi* and *Encephalitozoon cuniculi* have a wide range of mammalian species amongst their natural hosts.

*Erythrocytozoon bieneusi* has been reported from an HIV positive patient in New Zealand (52).

## Conclusion

Dermatophytes – *M. gypseum*, *T. mentagrophytes*, and *M. verrucosum* have been reported from mammalian species in New Zealand (26, 52). Although scientific references to *T. megninii* being identified in New Zealand have not been located, it is recognised as a cause of tinea capitis in the internet publication by the New Zealand Dermatological Society (53). *T. megninii* is, in any case, anthropophilic (54) and has the potential to enter New Zealand with humans. *M. gallinae* has not been reported from passerine birds.

None of the dermatophytes are considered to be potential hazards in the commodity.

Histoplasma capsulatum – is not a disease of birds and it is doubtful that it can be sustained in their intestinal tract.

*H. capsulatum* is not considered to be a potential hazard in the commodity.

Cryptococcus neoformans – Both *C. neoformans* var *neoformans* and *C. neoformans* var. *gatti* are present in New Zealand. Therefore *C. neoformans* is not considered to be a potential hazard in the commodity.

Candida spp. – Infections of humans and animals in New Zealand by a wide range of *Candida* species have been reported. Therefore *Candida* spp. are not considered to be a potential hazard in the commodity.

Aspergillus spp. are predominantly soil organisms, occasionally filling the role of opportunist pathogens. Aspergillosis is widespread in New Zealand. Therefore *Aspergillus* spp. are not considered to be a potential hazard in the commodity.

The Zygomycetes. Members of the genera *Absidia*, *Mortierella*, *Mucor* and *Rhizopus* are not considered to be potential hazards in the commodity.

Microsporidia. *Erythrocytozoon bieneusi* has been reported as present in New Zealand. While reports of the other microsporidia reported as infecting birds being present in New

Zealand have not been discovered, given the carrier rates reported internationally, the likelihood of these other organisms not being present in New Zealand is negligible. Therefore Microsporidia are not considered to be potential hazards in the commodity.

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## 3.28 Internal parasites

### 3.28.1 Nematodes

#### 3.28.1.1 Hazard identification

##### Aetiological agent

The section covers all nematode parasites relevant to the importation of passerine birds proposed to be imported from Europe to New Zealand.

##### OIE List

There are no nematodes of birds in the OIE list.

##### New Zealand Status

12 nematodes (either species or genera) are listed in the register of unwanted organisms.

61 species of nematodes have been identified from birds in New Zealand. Five of these (*Capillaria* sp., *Capillaria emberizae*, *Porrocecum ensicaudatum* and *Aucuaria skrjabini*) have been identified in passerine birds (1).

##### Epidemiology

A scan of literature databases reveals a large number of nematode parasites of species and genera not recorded in New Zealand but present in passerine birds in Europe. Neither the epidemiology of most nematode parasites of birds, nor their effect on the health of birds, is well described. The lifecycle of most nematodes of vertebrates involves the adult worm living in the gastro-intestinal tract, respiratory tract or some other tissue. Eggs are laid and passed from the host, usually in faeces, to the ground where development of larvae proceeds to a point where the larvae are infective to the host. Most nematode species reinfect the host through the oral route but direct tissue penetration or other means of infection occur with some species. Varying degrees of host specificity are apparent. Under most circumstances, nematodes have relatively little effect on their host, but under conditions of crowding or stress negative effects may occur.

The only report relevant to possible vertical transmission of nematodes in birds was that of Seaton et al. (2). From 36 eggs from turkeys with natural infections of *Ascaridia dissimilis*, they recovered a single ascarid egg. In an experiment, involving the artificial placement of faeces with ascarid eggs on to the shell of eggs, the numbers of ova recovered per egg dropped from 62 at day two of incubation to 3 at day 28.

## **Conclusion**

Exotic nematodes of birds are considered to be potential hazards in the commodity. hazards to susceptible bird in New Zealand.

### **3.28.1.2 Risk assessment**

#### **Release assessment**

Given

1. the lack of reports suggesting vertical transmission of nematode parasites of birds and
2. the very low levels of contamination on eggs from naturally infected turkeys and the rapid die-off of the experimentally introduced ascarid eggs during the incubation period

the likelihood of the introduction of nematodes exotic to New Zealand with passerine eggs imported from Europe is negligible.

The release assessment is negligible.

#### **Risk estimation**

Based on the negligible release assessment it is concluded that nematodes are not a hazard in the commodity.

#### **References**

1. McKenna, P.B. 1998. Check list of helminth and protozoan parasites of birds in New Zealand. *Surveillance* 25 (Special issue): 3-12.
2. Seaton, E.M.; Monahan, C.M.; Morishita, T.Y. 2001 Presence and recovery of *Ascaridia dissimilis* ova on the external shell surface of turkey eggs. *Avian Diseases* 45 (2): 500-503.

### **3.28.2 Trematodes**

#### **3.28.2.1 Hazard identification**

##### **Aetiological agent**

This section covers all trematode parasites relevant to the importation of passerine birds proposed to be imported from Europe to New Zealand.

##### **OIE List**

There are no avian trematodes in the OIE list.

##### **New Zealand Status**

Four trematodes (either species or genera) are listed in the register of unwanted organisms.

31 species of trematodes have been identified from birds in New Zealand. All have come from birds from aquatic environments; none from passerine species.

##### **Epidemiology**

A scan of literature databases reveals a large number of trematode parasites of species and genera not recorded in New Zealand but present in passerine birds in Europe. Neither the epidemiology of most trematode parasites of birds, nor their effect on the health of birds, is well described. Varying degrees of host specificity are apparent. For most, information on lifecycles and intermediate hosts is scant or not reported although there is a general requirement for avian trematodes to require a mollusc as an intermediate host to allow it to complete its lifecycle back the bird as the primary host. Most trematodes have little recognised effect on their host, but occasionally negative effects are attributed in poultry.

Introduction of avian trematodes would require the introduction of either infected molluscs or infected birds which were then able to excrete eggs to ground on which there are vector-competent molluscs.

##### **Conclusion**

Trematodes are not considered to be potential hazards in the commodity.

##### **References**

1. McKenna, P.B. 1998. Check list of helminth and protozoan parasites of birds in New Zealand. *Surveillance* 25 (Special issue): 3-12.

### **3.28.3 Cestodes**

#### **3.28.3.1 Hazard identification**

##### **Aetiological agent**

The section covers all cestode parasites relevant to the importation of passerine birds proposed to be imported from Europe to New Zealand.

##### **OIE List**

There are no cestodes of birds in the OIE list.

##### **New Zealand Status**

5 species or genera of Cestodes are listed in the register of unwanted organisms.

23 species of cestodes have been identified from birds in New Zealand. Five (*Anomotaenia verulainii*, *Aploparaksis*, *Choanotaenia infundibulum*, and *Hymenolepis serpentulus*) have come from passerine species (1).

##### **Epidemiology**

A scan of literature databases reveals a large number of cestode parasites of species and genera not recorded in New Zealand but present in passerine birds in Europe. Neither the epidemiology of most cestode parasites of birds, nor their effect on the health of birds, are well described. Varying degrees of host specificity are apparent. For most, information on lifecycles and intermediate hosts is scant or not reported but the general lifecycle of cestodes requires a primary host in which the parasite develops to sexual maturity, the passage of eggs which are taken up by an intermediate host, the development of an cysticercoïd stage of the parasite in the intermediate host, the eating of the intermediate host by a primary host (bird of suitable species) and the development of the adult cestode in that bird. Most cestode infections are asymptomatic but some do produce disease or pathology, especially when present in large numbers (2).

Introduction of avian cestodes would require the introduction of either infected intermediate hosts or infected birds which were then able to excrete eggs to ground with exposure to competent intermediate hosts.

##### **Conclusion**

Cestodes are not considered to be potential hazards in the commodity.

## References

1. McKenna, P.B. 1998. Check list of helminth and protozoan parasites of birds in New Zealand. *Surveillance* 25 (Special issue): 3-12.
2. McDougald, L.R. 2003 Cestodes and Trematodes pp 961-971 in *Diseases of Poultry*. Eds Saif, Y.M. *et al.* Iowa State Press. Ames, Iowa. 2003.



### **3.28.4 Acanthocephala**

#### **3.28.4.1 Hazard identification**

##### **Aetiological agent**

The section covers all acanthocephalan parasites relevant to the importation of passerine birds proposed to be imported from Europe to New Zealand.

##### **OIE List**

There are no acanthocephalan parasites in the OIE list.

##### **New Zealand Status**

There are no acanthocephalan parasites in the register of unwanted organisms.

Seven acanthocephalan species have been reported from birds in New Zealand, none of them from passerine birds (1, 2, 3).

##### **Epidemiology**

A scan of literature databases reveals a large number of acanthocephalan parasites of species and genera not recorded in New Zealand but present in passerine birds in Europe. Most reports come from eastern Europe but there are also reports of these parasites from countries as far west as Spain. Neither the epidemiology of most acanthocephalan parasites of birds, nor their effect on the health of birds, is well described. Acanthocephalans require development in intermediate hosts in order to become infective to the primary host. Such intermediate hosts for avian acanthocephalans include arthropods, snakes, lizard and amphibians. Varying degrees of host specificity are apparent (4). For most, information on lifecycles and intermediate hosts is scant or not reported.

Introduction of avian acanthocephalans would require the introduction of either infected intermediate hosts or infected birds which were then able to excrete eggs to ground with exposure to competent intermediate hosts.

##### **Conclusion**

Acanthocephalan parasites are not considered to be potential hazards in the commodity.

## References

1. McKenna, P.B. 1998. Check list of helminth and protozoan parasites of birds in New Zealand. *Surveillance* 25 (Special issue): 3-12.
2. McKenna, P.B. 2003 Register of new host-parasite records. *Surveillance* 30 (1): 12-13.
3. McKenna, P.B. 2003. Register of new host-parasite records. *Surveillance* 30 (4): 15-17.
4. McDougald, L.R. 2003 Internal parasites pp 931-961 in *Diseases of Poultry*. Eds Saif, Y.M. *et al.* Iowa State Press. Ames, Iowa. 2003.

### 3.28.5 Protozoa

#### 3.28.5.1 Hazard identification

##### Aetiological agent

The section covers all protozoan parasites relevant to the importation of passerine birds proposed to be imported from Europe to New Zealand.

##### OIE List

There are no protozoal diseases of birds listed in either of OIE lists A or B.

##### New Zealand Status

18 species of protozoa are listed in the register of unwanted organisms. Of these, two (*Babesia* spp. and *Trypanosoma* spp.) have been reported from passerine birds.

30 species of protozoa from birds have been reported in New Zealand. Thirteen of these have been recorded in passerine birds, none from genera included in the list of unwanted organisms (1).

##### Epidemiology

A scan of the literature reveals infection of European passerine birds by a large number of both blood-borne and intestinal protozoal parasites of species and genera not reported in New Zealand birds. Many reports make no mention of pathogenic effects, others suggest minimal effects while a few suggest that the parasites have either negative or positive effects.

On the basis of general epidemiology, there are two main groups of avian protozoa relevant to this risk analysis. A third group (Microspora) previously considered to be protozoa are now classified as fungi.

1. **Intestinal protozoa** -. These include coccidia, cryptosporidia, cochlosoma and other miscellaneous genera. The lifecycle of these parasites is illustrated by that for *Eimeria* (a genus of the coccidian) which involves ingestion of oocysts, release of sporozoites which enter the epithelial cells of the intestinal mucosa, two generations of asexual reproduction, a stage of sexual reproduction producing zygotes which mature to oocysts which are passed in the faeces (2). No reports suggesting that these parasites might be transmitted vertically have been discovered.
2. **Haemoparasites** – These include leucocytozoa, haemoproteus, trypanosoma and sarcocystis. These parasites have two-host lifecycles with those infecting birds having insects or, in the case of sarcocystis, carnivores or scavengers as the

alternative hosts (2). Vertical transmission between avian generations has not been reported.

## **Conclusion**

Protozoal parasites are not considered to be potential hazards in the commodity.

## **References**

1. McKenna, P.B. 1998. Check list of helminth and protozoan parasites of birds in New Zealand. *Surveillance* 25 (Special issue): 3-12.
2. McDougald, L.R. 2003 Protozoal infections pp 973-1023 in *Diseases of Poultry*. Eds Saif, Y.M. *et al.* Iowa State Press. Ames, Iowa. 2003.

## **3.29 External parasites**

### **3.29.1.1 Hazard identification**

#### **Aetiological agent**

The section covers all mites, fleas, ticks and louse flies relevant to the importation of passerine birds proposed to be imported from Europe to New Zealand.

#### **OIE List**

No ectoparasites of birds are included in the OIE list.

#### **New Zealand Status**

None of the ectoparasites listed in the register of unwanted organisms are parasites of birds.

Large numbers of ectoparasites of birds are present in New Zealand and a substantial number of them have been identified from passerine birds (1).

#### **Epidemiology**

There is a large number of ectoparasites of birds in Europe that have not been reported from New Zealand. Many are important as vectors of diseases. Others may cause negative effects by virtue of their own parasite status.

Reference to vertical transmission of ectoparasites of birds relate to either transfer of the ectoparasites from adult to hatched chicks (2, 3). Ectoparasites of birds are not transmitted transovarially. Kells and Surgeon (4) investigated the potential for northern fowl mites (*Ornithonyssus sylviarum*) to be dispersed between properties on hatching eggs. Although initial cross contamination from egg carts artificially contaminated with mites to other egg carts took place, under simulated hatching conditions all mites were dead within 96 hours.

#### **Conclusion**

Ectoparasites are not considered to be potential hazards in the commodity.

#### **References**

1. Bishop, D.M.; Heath, A.C.G. 1998. Parasites of birds in New Zealand – Check List of ectoparasites. Surveillance 25 (5) Special edition: 11-31.
2. Tompkins, D.M.; Jones, T.; Clayton, D.H. 1996 Effect of vertically transmitted ectoparasites on the reproductive success of swifts (*Apus apus*) Functional ecology 10 (6): 733-740.

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