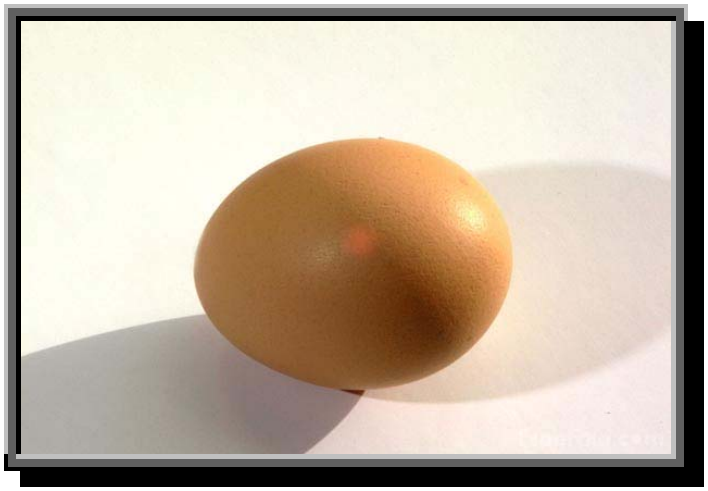
Import risk analysis: Hatching eggs from chickens *(Gallus gallus)* from the European Union, Canada, the United States of America, and Australia

FINAL



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Policy and Risk
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Approved for general release

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Executive Summary

This risk analysis considers the biosecurity risks associated with the importation of hatching eggs of chickens (*Gallus gallus*) from the European Union, Canada, the United States of America, and Australia.

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
- type 1 avian paramyxoviruses
- *Salmonella* Gallinarum-Pullorum
- *Salmonella* Typhimurium DT104
- *Salmonella* Enteritidis
- *Ornithobacterium rhinotracheale*

These organisms were classified as hazards in the commodity and options for the effective management of these risks have been presented. Risk management options discussed in this document include:

- The breeding establishments could be either free from avian influenza for at least 21 days prior to the collection of the eggs, or the parent flock could be tested for group A influenza viruses with negative results.
- The breeding establishments could have routine surveillance for avian paramyxovirus-1, and be free from avian paramyxovirus-1 for at least 21 days prior to the collection of the eggs.
- The breeding establishments could be free from fowl typhoid, pullorum disease, *Salmonella* Enteritidis and have no evidence of *Salmonella* Typhimurium DT104.
- The flock could be free from *Ornithobacterium rhinotracheale* on the basis of testing a sample of laying birds.
- Eggs could be hatched and chickens be held in quarantine in a MAF-approved avian transitional facility until required tests for avian influenza viruses and avian paramyxovirus-1 are completed with negative results and a biosecurity clearance given.

1. Introduction

This risk analysis examines the biosecurity risks posed by the importation of hatching eggs of chickens (*Gallus gallus*) from specified countries.

2. Commodity Definition

The commodity is hatching eggs of chickens (*Gallus gallus*) from the European Union, Canada, the United States of America, and Australia. The eggs will be sourced from poultry breeding flocks compliant with the standards described in Chapter 6.3 of the 2008 *OIE Terrestrial Animal Health Code* (1) (or equivalent) and be clean (free of faeces) when collected, unwashed and have intact shells (uncracked). Following collection, the eggs will be disinfected in accordance with Chapter 6.3 of the OIE *Code* (or equivalent).

3. Background

Import Health Standards (IHSs) are available for chicken hatching eggs from Australia, Great Britain, the United States of America, and Canada. These were developed prior to the implementation of the current policy of requiring scientifically based risk analyses as the basis for development of all IHSs. A company within the New Zealand poultry industry wishes to import chicken hatching eggs from Europe. It is considered appropriate that the scope of the risk analysis required to support the development of the IHS for that importation be broadened to include the range of countries specified in the commodity definition. This will ensure that all IHSs for chicken hatching eggs have the same technical base.

4. Risk Analysis Methodology

The methodology used in this risk analysis follows the guidelines as described in MAF Biosecurity New Zealand's *Import Risk Analysis Animals and Animal Products*(2)[1] and in Section 2 of the *OIE Terrestrial Animal Health Code* (1).

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

4.1. PRELIMINARY HAZARD LIST

The hazard identification process begins with the collation of a list of organisms possibly associated with the commodity. Table 1 shows these organisms, together with some of the key information considered in determining whether or not each organism should be considered further. This list was compiled from the text *Diseases of Poultry* 11th Edition, Editor Saif, Y.M., and from searches of the scientific literature.

[1] Risk analysis projects started after 12 April 2006 will follow procedures as outlined in Biosecurity New Zealand's *Risk Analysis Procedures Version 1* which can be seen at http://www.biosecurity.govt.nz/files/ pests-diseases/surveillance-review/risk-analysis-procedures.pdf

Figure 1. The risk analysis process.

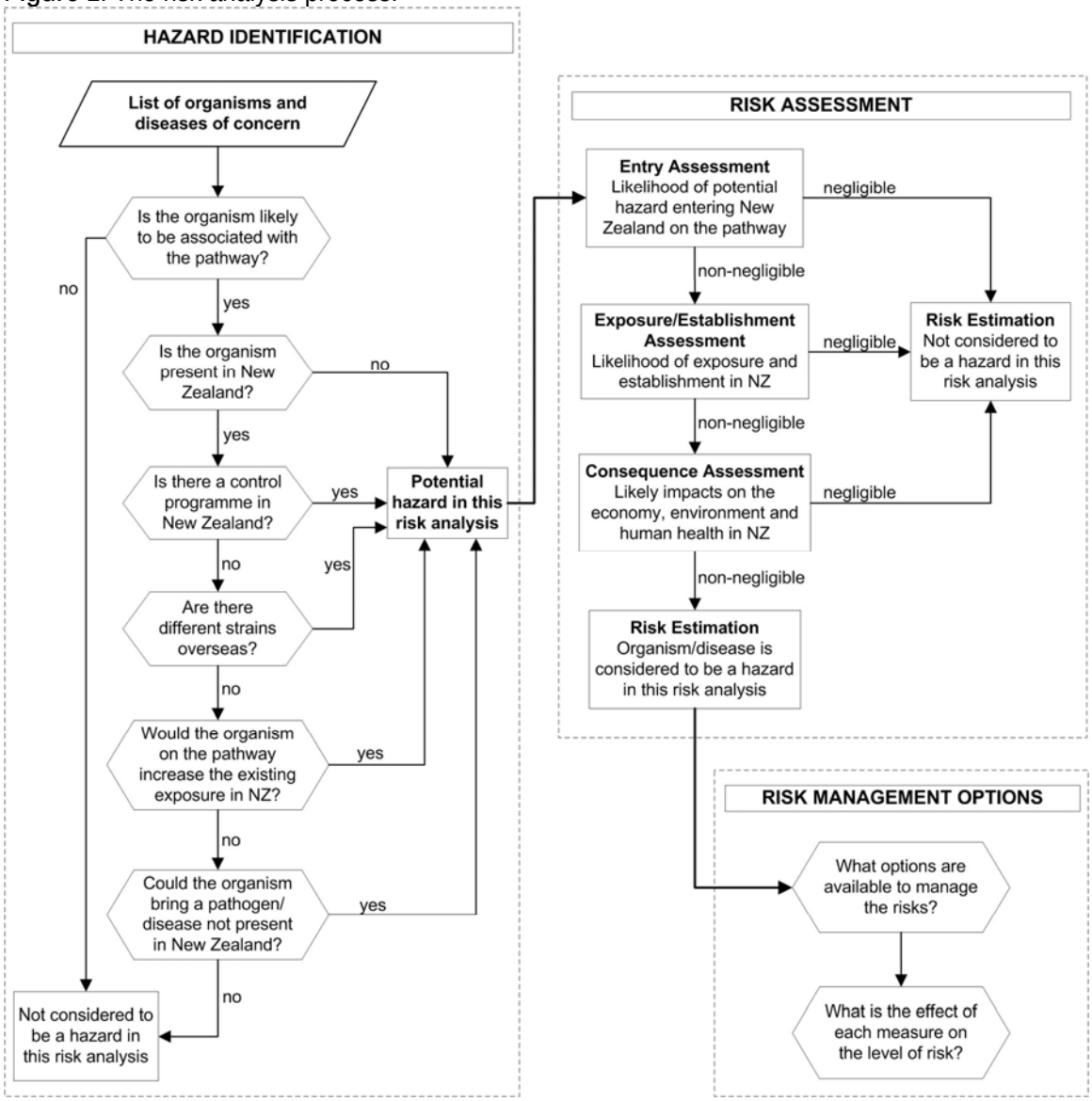

Table 1. Organisms potentially associated with the commodity

Organism/Disease	Present in New Zealand?	Evidence of more virulent strains overseas?[1]	Under official control or unwanted?[2]	Infection in eggs?[3]	Needs further consideration?
Orthomyxoviridae					
Avian influenza	Yes	Yes	Exotic strains notifiable	Yes	Yes
Paramyxoviridae					
Avian paramyxovirus 1 (APMV-1)	Yes	Yes	Notifiable (Exotic strains)	Yes	Yes
APMV-2	No?	Yes?	Other exotic organism	Yes?	Yes
Pneumoviruses	No?	N.A.	None	Yes?	Yes
Herpesviridae					
Marek's disease	Yes	Yes	Other exotic organism (Exotic strains)	No	No
Laryngotracheitis	Yes	Yes	None	No	No
Coronaviridae					
Infectious bronchitis	Yes	Yes	Unwanted (exotic strains)	Yes	Yes
Adenoviridae					
Group I avian adenoviruses (Inclusion body hepatitis)	Yes	No	None	Yes	No
Group II avian adenoviruses (Avian adenovirus splenomegaly)	No	N.A.	None	No	No
Group III avian adenoviruses (Egg drop syndrome)	Yes	No	None	Yes	No
Poxviridae					
Avipoxvirus	Yes	No	None	No	No
Circoviridae					
Gyrovirus (Chicken infectious anaemia)	Yes	No	None	Yes	No
Birnaviridae					
Infectious bursal disease	No	N.A.	Notifiable	No	No
Alphaviruses					
Equine encephalitides	No	N.A.	Notifiable	No	No
Flaviviridae					
West Nile virus	No	N.A.	None	No	No
Reoviridae					
Rotavirus	Yes	No	None	No	No
Viral arthritis, tenosynovitis	Yes	Yes	None	Yes	Yes
Picornaviridae					
Avian encephalomyelitis	Yes	No	None	Yes	No
Astroviridae					
Astrovirus (Avian nephritis virus)	Uncertain	N.A.	None	Yes	Yes

Table 1. (continued)

Organism/Disease	Present in New Zealand?	Evidence of more virulent strains overseas?[1]	Under official control or unwanted?[2]	Infection in eggs?[3]	Needs further consideration?
Retroviridae					
Leucosis/sarcoma complex viruses	Yes	No	None	Yes	No
Reticuloendotheliosis	Yes	No	None	Yes	No
Hepesviruses					
Big liver and spleen disease virus	No	Yes	Other exotic organism	No	No
Bacteria associated with enteric and generalised infections in birds					
Salmonellae	Yes/No	Yes	*S.* Pullorum and *S.* Gallinarum notifiable. Other exotic serovars and phage types unwanted	Yes	Yes
Campylobacter spp.	Yes	No	None	Yes	No
Escherichia coli	Yes	No	None	Yes	No
Bacteria commonly associated with respiratory disease in birds					
Pasteurella multocida	Yes	Yes	None	No	No
Pasteurella gallinarum	No	N.A.	None	No	No
Riemerella anatipestifer	Yes	No	None	No	No
Ornithobacterium rhinotracheale	No	N.A.	Other exotic organism	Yes	Yes
Haemophilus paragallinarum	No	N.A.	Other exotic organism	No	No
Bordetella avium	No	N.A.	Other exotic organism	No	No
Mycoplasma gallisepticum	Yes	No	None	Yes	No
Mycoplasma synoviae	Yes	No	None	Yes	No
Mycoplasma iowae	No	N.A.	Other exotic organism	Yes	Yes
Pseudomonas spp.	Yes	No	None	Yes	No
Intracellular bacteria					
Mycobacterium tuberculosis	Yes	No	None	No	No
Mycobacterium avium	Yes	No	None	No	No
Other mycobacteria	Yes (Some)	Yes	Other exotic organism (exotic strains)	No	No
Other bacteria					
Francisella tularensis	No	N.A.	Other exotic organism	No	No
Megabacteria	Yes	No	None	No	No
Gram positive contaminants (e.g. staphylococci/streptococci/ enterococci)	Yes	No	None	No	No
Proteus/*Providencia* group	Yes	No	None	Yes	No
Klebsiella spp.	Yes	No	None	Yes	No
Acinetobacter spp.	Yes	No	None	Yes	No

Table 1. (continued)

Organism/Disease	Present in New Zealand?	Evidence of more virulent strains overseas?[1]	Under official control or unwanted?[2]	Infection in eggs?[3]	Needs further consideration?
Other bacteria (cont)					
Citrobacter spp.	Yes	No	None	Yes	No
Flavobacterium spp.	Yes	No	None	Yes	No
Alcaligenes spp.	Yes	No	None	Yes	No
Serratia spp.	Yes	No	None	Yes	No
Hafnia alvei	Yes	No	None	Yes	No
Bacillus spp. (Not *Bacillus anthracis*)	Yes	No	None	Yes	No
Clostridium perfringens	Yes	No	None	Yes	No
Chlamydophila psittaci	Yes	No	None	Yes	No
Spirochetes					
Borrelia anserina (Avian spirochaetosis)	No	N.A.	Other exotic organism	No	No
Borrelia burgdorferi (Lyme disease)	No	N.A.	Other exotic organism	No	No
Brachyspira spp.	Yes	No	None	No	No
Rickettsial agents					
Coxiella burnetii	No	N.A.	Notifiable organism	No	No
Aegyptianella pullorum	No	N.A.	None	No	No
Other Rickettsia	Yes/No	Yes	Some are in the in register	No	No
Fungi and yeasts					
Enterocytozoon bieneusi	Yes	No	None	No	No
Encephalitozoon cuniculi	Yes	N.A.	None	Yes	No
Other fungi and yeasts	Yes/No	Yes	None	No	No
Internal parasites					
Nematodes, cestodes, protozoa	Yes/No	Yes	None	No	No
External parasites					
Ticks, mites, lice	Yes/No	Yes	Unwanted (Some genera)	No	No

[1] 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 New Zealand, 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.

[2] Based on the information from the register of unwanted organisms at http://www.biosecurity.govt.nz/pests-diseases/registers-lists/unwanted organisms/

[3] For the purposes of this analysis, infection of eggs is considered to take place if the literature contains references to vertical transmission of the organism or to infection in eggs.

4.2. RISK ASSESSMENT

In the following sections of this analysis, for each organism identified as requiring further consideration in Table 1, the epidemiology is discussed, including a consideration of the following questions:

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

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

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

In the case of this risk analysis, an exception to this framework is made for the treatment of salmonellae considered of particular concern to human health. *Salmonella* Typhimurium DT 104 and *S*. Enteritidis (various phage types) have been isolated in New Zealand but are not established in poultry. Codes of practice in the poultry industry (3) are such that importation of poultry stock infected with these organisms would require imposition of control and eradication procedures that would result in very high costs to the importer and, potentially, the broader industry.

If importation of the commodity is considered likely to result in an increased exposure of people to other potentially zoonotic organisms already present in New Zealand, then these organisms are also considered to be potential hazards.

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

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

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

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

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

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

4.3. RISK MANAGEMENT

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

4.4. RISK COMMUNICATION

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

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

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

5. Avian Influenza Virus

5.1. HAZARD IDENTIFICATION

5.1.1. 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 – 16) and neuraminidase (N1 – 9) (4). Strains of AI are commonly separated into highly pathogenic strains (HPAI) and low pathogenic strains (LPAI) on the basis of their pathogenicity in chickens. All HPAI virus isolates have been subtypes H5 or H7 but not all H5 or H7 isolates have been highly pathogenic (4, 5).

5.1.2. 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 Chapter 10.4 of the *OIE Terrestrial Animal Health Code* (1).

5.1.3. New Zealand status

Avian influenza H5 and H7 are listed in New Zealand's unwanted organisms register.

AI viruses have been isolated from healthy wild mallard ducks in New Zealand (6-9). Subtypes identified have included H4N6, H1N3, and H5N2 (6, 10). The H5N2 isolates were shown to be non-pathogenic (6, 10). In 2008 a H5N1 virus was isolated from mallards. However, this isolate is a low pathogenicity strain, unlike the high pathogenic strain responsible for the world-wide pandemic of avian influenza (183).

A survey in the 1990s found no evidence of AI virus infection in 54 pigeons trapped at three locations in New Zealand or in samples from 55 native birds (11). These negative findings received support from a 2003 serological survey of 560 pigeons (domestic and wild) sampled from Auckland, Wellington, Christchurch, and Dunedin (12). AI viruses have never been diagnosed in New Zealand domestic poultry.

5.1.4. Epidemiology

Three reviews of the epidemiology of AI (5, 13, 14) and Chapter 10.4 of the *OIE Terrestrial Animal Health Code* (1) form the main bases for this section.

The main means of spread is through virus passed in faeces or respiratory secretions. Spread may be directly into areas occupied by poultry, through contamination of water, or through carriage on fomites. Outdoor poultry flocks are more vulnerable to exposure to wild birds, and are therefore affected with AI more frequently than flocks maintained indoors. AI virus may be maintained in poultry flocks as LPAI or LPAI may be introduced into an area by infected wild birds, mainly waterfowl, with gulls and sea birds playing a lesser role. Because of the relatively high prevalence of AI viruses in migratory waterfowl, commercial flocks located outdoors and on migratory pathways appear to be at highest risk. Secondary spread is through transfer of infection from faeces, most commonly by people moving between flocks or properties. Spread of AI within live-bird markets has been found to be an important means of

dissemination between poultry farms in both Asia (15-17) and the north eastern United States (18, 19). Market hygiene (including control of interspecies contact) has been found to be a critical factor in controlling spread within these environments.

Virulent H5 and H7 strains apparently arise by mutation from low pathogenic strains some time after the transfer of infection from the wild host to poultry. The ability of these mutated viruses to infect multiple tissues results in their high pathogenicity. 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 (20) and differences in clinical and pathological presentation of natural AI infections in different species (21).

In the reviews by Alexander (22) and Senne (23) of reports of avian influenza over the period from 1997 to 2002, ten subtypes of AI are listed as having been identified in chickens. Of these, four are NAIs, being either H5 or H7. The remaining six non-NAIs are discussed briefly below:

- H1N1 subtype was identified in chickens in Canada in 1998 (23). It was a virus of this subtype that caused the pandemic in humans in 1918.
- H2N2 has been reported from the eggs of healthy chickens in the United States (24). An H2N2 virus was responsible for a human influenza pandemic in 1957.
- H3N6 subtype has been identified in chickens, quail, ducks and caged birds in Asia (22, 25), and in chickens in the United States (23) but no reports attributing pathogenicity to this subtype have been located.
- An incident of H6N2 infection in flocks of laying chickens in California was associated with respiratory disease and decreased egg production (23).
- H9N2 subtype was recorded from chickens in Europe, Iran, Saudi Arabia (22), and the United States (23). Guo *et al*. (26) cite several reports of H9N2 infecting humans in China and Hong Kong. Viruses of this subtype have been reported as causing epidemics of avian influenza in turkeys in the United States (27) and chickens in Iran (28).
- Birds infected with H10N7 virus in Ontario, Canada showed respiratory disease and kidney necrosis (23).

Horimoto and Kawaoka (29) 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.

5.1.5. Hazard identification conclusion

NAI viruses must be considered to have the potential to lead to the development of disease and are classified as potential hazards in the commodity.

There are also a number of non-NAI subtypes with the capacity to cause disease in poultry. The full potential for such disease relationships is not understood and genetic changes in non-NAI strains, or encounters with new potential hosts, may result in disease.

Therefore all avian influenza viruses are classified as potential hazards in the commodity.

5.2. RISK ASSESSMENT

5.2.1. Entry assessment

AI virus has been isolated from the internal contents of eggs from naturally-infected layer and breeder flocks with clinical disease and from an infected layer flock with no clinical signs (30). Unpublished work by Brugh cited by Swayne and Beck (31) identified HPAI virus in 85 to 100 percent of eggs laid on days 3 and 4 following experimental inoculation. Although no reports of transmission of infection to chicks via infected eggs have been located, 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 (32).

The entry assessment for AI virus in chicken eggs is considered to be non-negligible.

5.2.2. Exposure assessment

AI virus is spread via respiratory aerosols, faeces, fomites, and people, with the faecal-oral route being the most important. Any infection in hatchlings derived from imported eggs is likely to spread to other birds through either direct or indirect contact.

The exposure assessment for AI viruses is considered to be non-negligible.

5.2.3. Consequence assessment

There is a high likelihood that the importation of HPAI viruses could result in epidemic disease in New Zealand poultry with high mortalities and disruption of the poultry industries and export trade in poultry products.

The low susceptibility of birds in the wild and the imposition of control measures in the event of an incident of HPAI infection are likely to limit consequences on wild birds.

The potential for LPAI strains to cause disease in birds in New Zealand cannot be excluded; nor can the possibility of LPAI strains becoming pathogenic following mutation or genetic recombination.

The intensive mixing of humans, pigs, and ducks seen in Asia which is considered to contribute to the development of new strains of influenza virus pathogenic to humans, does not occur 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 considered to be very low. The epidemiology of the development of strains of AI virus pathogenic to humans is such that AI viruses in this commodity are not considered to be a potential hazard to human health.

AI viruses in this commodity are considered to be a hazard to the New Zealand poultry industry and to the economy. There may be effects on other birds, including native species, particularly water fowl, but such effects are likely to be relatively small.

The consequence assessment for AI is considered to be non-negligible.

5.2.4. Risk estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and avian influenza viruses are classified as a hazard in the commodity. Therefore, risk management measures can be justified.

5.3. RISK MANAGEMENT

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

i. Eggs could come from an AI-free country, zone, or compartment and be derived from parent flocks which had been kept in an establishment free of all AI viruses for at least 21 days prior to and at the time of the collection of the eggs.

ii. As indicated in Chapter 10.4 of the *OIE Terrestrial Animal Health Code* (1), birds could be randomly tested using virus detection or isolation tests, and serological methods, and the frequency of testing should be based on the risk of infection and at a maximum interval of 21 days. Tests methods used could detect all group A influenza viruses. The number of birds sampled would depend on the expected minimum prevalence of infection and the level of confidence required for its detection. The only report found that documented within-flock seroprevalence rates is that of Al-Natour and Abo-Shehada (33) in Jordan. In flocks from which 30 sera were tested using an ELISA targeting subtype H9N2, between 1 (3 percent) and 30 (100 percent) sera were positive from individual flocks. 95 percent confidence that the seroprevalence in the flock is below 5 percent is a reasonable basis for determining sample numbers.

iii. Eggs could be hatched under secure quarantine conditions in New Zealand and a sample of hatchlings tested prior to clearance.

iv. Cloacal or choanal swabs from live birds or hatchlings could be cultured for AI virus using methods described in the *OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* (34) or tested by PCR methods that detect group A influenza viruses (35).

v. Birds and/or hatchlings could be kept in contact with specific pathogen free chickens during the quarantine period and then these chickens tested for infection with AI using methods identified above.

6. Avian Paramyxoviruses (APMV-1 TO 9)

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Nine "prototype" virus strains of paramyxovirus are recognised in birds. They are in the genus Avulavirus (35), are differentiated on serological grounds, and identified as avian paramyxoviruses 1 to 9 (APMV-1 to 9) (36).

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 (37) 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 in the virus (34).

6.1.2. OIE list

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

6.1.3. 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 (38). Pharo *et al*. (39) 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 of low virulence.

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 (36), only APMV-1, 4 and 6 could be concluded to be present. Testing did not include APMV-5 (40).

Stanislawek *et al*. (41) 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. Because of cross reactivity between APMVs, the presence of other APMVs in caged or wild birds could not be excluded.

6.1.4. Epidemiology

Newcastle disease virus (NDV) is widely distributed and there is almost universal use of ND vaccines in commercial poultry throughout the world (36). Transmission between birds may be through either inhalation or ingestion. Geographic spread may be aided by movement of

live birds, contact between animal groups, and movement of people and/or fomites. Contamination of waterways, ponds, and surface water has also been proposed as means of spread of NDV (42, 43). Infection in groups of birds may present with signs varying from high morbidity and high mortality to inapparent infections depending upon viral strain and host species. There are reports of ND causing mild transient conjunctivitis and, occasionally, fever in humans. Reports of human to human transmission have not been located (36).

Mutation of an APMV-1 virus of low virulence was proposed as the most likely source of high virulence virus that caused a Newcastle disease outbreak in Australia (44, 45).

Live vaccines, using lentogenic virus, are frequently used for induction of a relatively short-lived initial immune response in young chickens. Immunity may be reinforced by the later administration of live vaccine of greater potency and/or by use of an inactivated product. Vaccine viruses have been recovered from young chickens up to 19 days after vaccination (48-48). While vaccination provides protection against serious disease, it does not always prevent infection or excretion of the virus (36).

Up to 1988, APMV-1 infection had been reported from 241 species of birds from 27 orders with differences in clinical presentation even between species within the same genus (49). Further identifications have taken place since that time and Alexander (36) proposed that the majority of, if not all, birds are susceptible to infection.

APMV-2 (also called Yucaipa virus) is widespread in poultry, particularly chickens and turkeys (36). In these species it commonly causes mild respiratory disease, although more severe disease has been reported in turkeys (50). In wild and caged birds APMV-2 has been recorded from Europe, Asia, Africa, and American countries with most isolations being from passerine birds (36). Disease associated with APMV-2 has not been reported from non-poultry species.

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. APMV-3 strains infecting caged birds differ from those infecting turkeys (51).

APMV-4 viruses have been isolated only from ducks and geese and have not been associated with disease (36, 40).

APMV-5 has been reported only from pet budgerigars in a unique epizootic in Japan between 1974 and 1976 (52).

APMV-6 has been isolated from turkeys, in which it may cause mild disease, and from ducks and geese in which disease association has not been reported (36).

APMV-7 has been reported from pigeons, doves, turkeys, and ostriches. It has been associated with mild respiratory disease in turkeys (36) but searches for reports of pathogenicity in other species have not been successful.

APMV-8 and 9 have been reported from ducks and geese (36) but reports suggesting pathogenicity have not been located.

6.1.5. Hazard identification conclusion

Based on host specificity and their ability to cause disease, both APMV-1 and APMV-2 are classified as potential hazards in the commodity.

6.2. RISK ASSESSMENT

6.2.1. Entry assessment

Pospisil *et al*. (53) and Capua *et al*. (54) findings of lentogenic and virulent Newcastle disease virus respectively, in eggs and chickens from infected hens support contentions that APMV-1 may be transmitted transovarially. Chen and Wang (55), 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*. (56) found that Yucaipa virus and Bangor virus strains (both members of APMV-2) isolated from finches grew in eggs and that some embryos survived.

Vertical transmission of APMVs is regarded as controversial (36) and an OIE recognized expert in Newcastle Disease (Dr.Paul Selleck, CSIRO, Australian Animal Health Laboratory) was approached for an expert opinion. Dr Selleck (pers. comm.[2]) commented that he had been able to isolate virulent NDV from the albumen of eggs laid by vaccinated and then experimentally challenged chickens, indicating that, under certain circumstances, NDV can be transmitted vertically. In unvaccinated chickens, infection with virulent NDV causes almost immediate cessation of laying due to infection of the oviduct. Eggs laid by unvaccinated chickens that are infected with virulent NDV are unlikely to hatch as the virus will kill them. Therefore the opportunity to spread in eggs is reduced or eliminated. With non-virulent NDV, virus replication is confined to the gut and/or respiratory tract. It is possible that non-virulent NDV may contaminate an egg surface but if the egg is well washed or the surface disinfected the chances are greatly reduced. APMV-2 should fall in the same category as non-virulent NDV but there is no direct experimental evidence one way or the other.

The entry assessment for APMV-1 in the commodity is therefore considered to be non-negligible. As there is no evidence for vertical transmission of APMV-2, the entry assessment for APMV-2 is considered to be negligible.

6.2.2. Exposure assessment

If hatching eggs carrying exotic strains of APMV-1 were imported the most likely outcome would be the introduction of the virus into the hatchery. The potential routes of spread from an infected hatchery could include mechanical spread (primarily by the movement of people and equipment), movement of infected birds from the hatchery (live or dead), and airborne spread.

MAF's 2001 import risk analysis on APMV-1 in hatching eggs[3] concluded that the exposure pathways for APMV-1 are primarily those that rely on direct or indirect contact with faecal material. Spread by humans or human activities associated with poultry flocks is the most

[2] Selleck P. Virologist, CSIRO Australian Animal Health Laboratory, Victoria, Australia. E-mail to Stephen Cobb, September 2006

[3] Pharo H (2001) *Import risk analysis:* avian paramyxovirus type 1 in hens' hatching eggs, MAF, Wellington. See:www.biosecurity.govt.nz/pests-diseases/animals/risk/avian-paramyxovirus-ra.pdf

likely way that APMV-1 virus imported in hatching eggs would escape from the hatchery environment and result in exposure of avian species in New Zealand.

The exposure assessment for APMV-1 is therefore considered to be non-negligible.

6.2.3. 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 spreads rapidly could disrupt current sero-surveillance (57). The establishment of additional strains of APMV-1 might also increase the likelihood of the mutation of a virus to velogenic form. The introduction of a velogenic strain would have serious consequences for the poultry industry and could result in substantial mortalities in wild and/or caged birds.

There are reports indicating that both velogenic and vaccine strains of APMV-1 (36, 58, 59) from poultry can cause disease in humans. APMV-1 infections in humans have most commonly been reported in association with conjunctivitis, but some reports have referred to chills, headaches and fever. Given the presence of a lentogenic strain of APMV-1 in New Zealand, the mild and transient nature of the disease and the infrequency of such reports, any consequences to human health are likely to be minor.

The introduction of APMV-1 in the commodity would be associated with non-negligible consequences to the New Zealand poultry industries and to human health. The consequence assessment for APMV-1 is therefore considered to be non-negligible.

6.2.4. Risk estimation

Because the entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and APMV-1 is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

6.3. RISK MANAGEMENT

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

i. Eggs could originate from flocks in a country, zone or compartment free of APMV-1.

ii. Eggs could be derived from flocks in which vaccination for Newcastle disease using live vaccines has not taken place within the four weeks prior to collection of eggs, and from flocks that have been certified as being free from APMV-1.

iii. The flock from which eggs are to be collected could be tested for evidence of APMV-1. Such a sample could be sufficiently large to provide confidence that APMV-1 is not present.

iv. Eggs could be hatched under secure quarantine conditions in New Zealand and material from embryos, dead-in-shell chicks, or hatchlings could be tested for APMV-1 before release, with the sample size being chosen according to the required confidence in detecting the expected minimum prevalence. Hatched chickens could be held in contact

with chickens of New Zealand origin from flocks shown to be free of APMV-1, which are then tested for APMV-1 after the period of exposure.

Test procedures available include culture for APMV using methods described in Chapter 2.1.15 of the *OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* (34). Any APMV isolate could be serotyped, particularly for APMV-1, paying particular attention to the antigens and antisera used to avoid erroneous identification (34). Alternatively, on the basis of the epidemiology and release assessment presented above, it could be assumed that any APMV isolated is APMV-1.

Haemagglutination tests, haemagglutination inhibition tests, and ELISAs can be used in the serological diagnosis of Newcastle disease. The OIE *Manual* does not prescribe a test for international movement of animals or animal products although the haemagglutination inhibition test is listed as an "alternative test" suitable for the diagnosis of disease within a local setting, and for use in the import/export of animals after bilateral agreement (34). Validation of tests has mainly focussed on APMV-1 in poultry and Alexander (36) comments on the need for care in reagent selection. Vaccination may cause positive serological test results.

7. Pneumovirus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Family: Paramyxoviridae; Subfamily: Pneumovirinae; Genus: Metapneumovirus, avian pneumovirus (APV) has proved difficult to culture and, for that reason, much of the research involving identification of infected birds has used PCR technology.

7.1.2. OIE list

Not listed.

7.1.3. New Zealand status

Turkey rhinotracheitis virus is listed in the unwanted organisms register.

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

7.1.4. Epidemiology

Avian pneumovirus subgroups

The terms avian pneumovirus and turkey rhinotracheitis virus are interchangeable. Phylogenetic analyses indicate that APV subgroup C (APVc) is distinct from APV subgroups A and B (APVa and APVb) (60-63). Two isolates originating from turkeys in France in 1985 had distinct nucleotide sequences more closely resembling those of APVa and APVb and were proposed to be classified as subgroup D (64). As these two turkey isolates are the only ones classified in subgroup D, this subgroup is not considered further in this risk analysis.

APVa and APVb have a wide geographic distribution through Africa, Asia, Europe, and South America (62, 65) where they cause swollen head disease of chickens and rhinotracheitis of turkeys. APVc is recognised in the United States where it was first described in Colorado, from where it has been eradicated and is now confined to Minnesota, North Dakota, South Dakota, Iowa, and Wisconsin (65, 66). APVc was also reported in association with coughing and a drop in egg production in a flock of Muscovy ducks in France (67).

Serological testing of 10,000 chicken and turkey sera in Canada during 1990-92 found no evidence of avian pneumovirus (69). Since that time there have been no reports of serological, clinical, or pathological evidence of APV in Canada. The only report located suggesting possible evidence of APV in Canada identified APV RNA in a single pooled sample from 16 snow geese (*Chen caerulescens*) shot in Saskatchewan (66).

Chickens are not refractory to experimental infection with APVc (70) but natural infections of chickens with APV have not been reported from North America and Halvorson (pers.

comm.[4]) stated that "we" (the College of Veterinary Medicine at the University of Minnesota) have never detected APVc (or any other pneumovirus) in chickens in Minnesota even though approximately 50 percent of the turkey flocks are seropositive.

Conclusion 1

Based on the evidence that APVc is genetically distinct from APVa and APVb, and the absence of evidence that it causes natural infections in chickens, APVc is not classified as a potential hazard in the commodity.

Conclusion 2

There is no evidence that either APVa or APVb are present in the United States or Canada.

APVa and APVb

APV causes turkey rhinotracheitis, a serious disease which may have both high morbidity and mortality, and it is a major contributory factor in SHS of chickens (65). In turkeys, morbidity and mortality can be high (up to 100 percent and 50 percent respectively). In chickens, SHS is less dramatic with morbidity and mortality rates of 4 percent and 2 percent respectively being more common and then usually in association with other pathogens. Direct contact is thought to be the main means of transmission between birds (65). Population seroprevalence data presented by a number of authors (66, 71) indicate seroconversion of virtually all birds in individual infected flocks. This was the case for six turkey flocks tested using ELISA after the development of clinical disease, three broiler flocks prior to the development of disease in Italy (72) and farmed ostriches in Zimbabwe (68). Introduction of a high-passage strain of APV to 0.2 percent of birds in each of ten flocks of 2 – 4 week old turkeys, each with 20,000 to 50,000 birds, was considered to have resulted in transfer of infection to all birds within 10 days (73).

The description of SHS provided by Pattison *et al*. (74) in the United Kingdom was one commencing with coughing then developing to opisthotonus, incoordination, swelling around the eyes and over the head, discharges from eyes and/or ears, and a green diarrhoea. Although the morbidity rate was low (around 1 percent), the case mortality rate approached 100 percent. Of 14 broiler breeder farms observed, 10 seroconverted to APV, SHS developed on six, and a decrease in egg production was noticed on a further three.

Horizontal transmission

Spread of APV is thought to be through direct contact. Two experiments testing the ability of APV to spread between groups of turkeys found that the virus did not bridge gaps of two feet (75) or one metre (76) between cages of groups of birds even though virus was being transmitted between challenged and unchallenged birds within the infected cages and the flow of air in the rooms was from infected cages toward those uninfected. Suspicions that fomites and movement of people may contribute to local spread (65) are supported by evidence that APVc can survive for several weeks at 4°C, for at least seven days while drying at room temperature (77) and in poultry litter for at least three days at room temperature (20-25°C) and 14 days at 8°C (78). The role of wild birds in the dissemination of infection is speculative.

[4] Halvorson, D., College of Veterinary Medicine, University of Minnesota, USA. E-mail to Bruce Simpson, 9 May 2006.

Vertical transmission

The proposition that APVs may be transmitted vertically is supported by reports of pathology and viral RNA in the uterus and oviduct following experimental infections of turkeys (79) and chickens (80) with isolates of APVa and APVb respectively. In both reports, the viral presence was associated with gross pathology of the reproductive tract including egg peritonitis and in the second report (80) intravenous administration of virus had lead to 70 percent of birds in a group of 24 becoming ill and over 50 percent developing diarrhoea. One bird died, two were killed while moribund and another six birds were killed between seven and 11 days post-infection while ill. APV antigen was detected in the oviduct epithelium of six of the birds that died or were killed. In the report by Jones *et al*. (79) APV was detected in the epithelium of the reproductive tract on days seven and nine post-infection but virus was not identified in the ovary. They concluded that there was no evidence to-date (1988) that the virus is transmitted through eggs. While Cook *et al*. (80) suggested that infection of the reproductive tract might contribute to the decrease in egg production commonly seen in APV infection, and the faults in egg quality observed less frequently, they made no comment on its significance with respect to egg borne transmission.

Three other reports of experimental infection of chickens with European isolates of APV and investigations of pathology of, and possible APV presence in, tissues of the reproductive tract have been located (81, 82). No evidence of APV infection (viral recovery, positive immunofluorescent staining, or pathology) was found in tissues of the reproductive tract.

Shin *et al*. (83) obtained APVc PCR positive results from pooled egg contents from turkey breeder flocks and from choanal swabs from day-old poults at a hatchery. However, attempts to culture APV from the choanal swabs and from eggs were unsuccessful. Clinical disease consistent with APV infection had been observed in breeder flocks five weeks prior to hatching of the affected poults. Comparable reports from regions where APVa and/or APVb are present have not been located.

No reports of avian pneumovirus in Australia have been found and it is generally accepted that the virus is not present in that country (65).

7.1.5. Hazard identification conclusion

APVs subgroup A and subgroup B are classified as potential hazards in the commodity.

On the basis of phylogenetic differences and the lack of evidence that APV subgroup C infects chickens under field conditions, APV subgroup C is not classified as a potential hazard in the commodity.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

There is no firm evidence that APV can be transmitted through eggs, however, the observations by Shin *et al*. (83) of APV RNA in turkey eggs and young turkeys, and their epidemiological observations, support the hypothesis that this may be possible for APVc. The findings of APV antigen in the reproductive tracts of chickens and turkeys (79, 80) suggest

that such a transmission route may also be available for APV in Europe. However, pathology of the reproductive tract of chickens has been observed only in birds with severe disease.

Given the uncertainty in published literature surrounding vertical transmission of APVs, Dr. Richard E. Gough (Department of Avian Virology, VLA Weybridge, United Kingdom) was approached for an expert opinion. Dr. Gough commented (pers. comm.[5]) that virtually all the reports describe laboratory studies in which birds were infected with large doses of virus either intravenously or intraocularly rather than natural infection. His view was that, whilst replication of the virus can take place in the reproductive tract of chickens, there is no evidence that the virus can be transmitted via the egg contents. The most compelling evidence for the vertical transmission of APV is the study by Shin *et al*. (2002) (83) in neonatal turkeys, originating from breeders infected with APVc virus, in which PCR positive products were detected in egg contents and newly hatched chicks. These results had been discussed at several meetings and the fact that live virus was not isolated from the samples has caused some people to question the validity of the results. As far as Dr. Gough was aware, these results had not been confirmed elsewhere.

Reflecting these comments from Dr. Gough and the lack of evidence supporting vertical transmission of APVa or APVb, the entry assessment for APVa and APVb in the commodity is therefore considered to be negligible.

7.2.2. Risk estimation

Because the entry assessment is negligible, the risk estimate is negligible and APVa and APVb are not classified as hazards in the commodity. Therefore, risk management measures are not justified.

[5] Gough RE. Dept. of Avian Virology, VLA Weybridge, United Kingdom. E-mail to Stephen Cobb, October 2006.

8. Infectious Bronchitis Virus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Infectious bronchitis virus is a Coronavirus within the Coronaviridae.

8.1.2. OIE list

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

8.1.3. New Zealand status

Infectious bronchitis virus (IBV) is endemic in New Zealand (84, 85) with viruses appearing to fall into four serotypes distinct from those viruses tested from other countries (86, 87). Vaccination is now widespread and a high proportion of birds are serologically positive.

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

8.1.4. 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 (88). Although IBV causes pathology of the oviduct (89-91) and embryonated eggs are routinely used for culture of IBV (88), literature searches have found only one suggestion of virus infection of eggs from infected hens (an unreferenced comment by Cavanagh and Naqi (88)). Literature searches have failed to find other reports of the isolation of IBV from eggs or reports suggesting vertical transmission.

8.1.5. Hazard identification conclusion

With the only traceable reference to IBV infecting eggs being an unreferenced comment by Cavanagh and Naqi (88), and with an inability to locate any reports suggesting that vertical transmission of IBV takes place, exotic strains of IBV are not classified as a potential hazard in the commodity.

9. Avian Infectious Arthritis

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Avian infectious arthritis is caused by reoviruses.

9.1.2. OIE list

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

9.1.3. New Zealand status

Avian infectious arthritis is not listed in the unwanted organisms register.

The first report of reoviruses from New Zealand poultry (92) was from fertile eggs with no association with disease. Subsequently, reovirus was isolated from broiler chickens in a flock suffering a mortality rate in excess of 6 percent associated with respiratory disease and atrophy of both the thymus and bursa (93). Howell (94) reported diagnoses of reovirus infections in birds with tenosynovitis and diarrhoea. Earlier reports from routine surveillance for evidence of reovirus infection in poultry recorded positive serology and the presence of disease (95). Later reports did not include reference to disease but did record vaccination and a high prevalence of positive titres (96).

Although Green *et al*. (92) concluded that their five isolates were serologically indistinguishable they also proposed that it was likely that other serotypes would be present in New Zealand. No reports of further serotyping of reoviruses from New Zealand, or of comparing New Zealand isolates with those from overseas, have been located.

9.1.4. Epidemiology

In his review, Rosenberger (97) states that reovirus infections of birds are common around the world. Most infections are subclinical, however, reovirus has been associated with arthritis, tenosynovitis, respiratory, hepatic, enteric, and other diseases. The viruses may spread readily both horizontally and vertically and there are strain variations based on serotypes and pathogenicity.

Both live-attenuated and inactivated vaccines are used for the control of disease associated with reoviruses. Vaccines derived for a single strain have been used with success in many parts of the world (97), suggesting that the protective immune response may not be closely related to the antigen(s) responsible for differentiation of serotypes.

9.1.5. Hazard identification conclusion

Given the range of pathology that has been associated with reoviruses in New Zealand, the worldwide prevalence of subclinical infections of poultry and other birds, and the large number of birds that have been imported to New Zealand without biosecurity measures that

would prevent the entry of virus, the proposal by Green *et al*. (92) that other serotypes will be present remains likely.

On the above basis it is concluded that reoviruses are not a potential hazard in the commodity.

10. Avian Nephritis Virus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Avian nephritis virus is in the Avastrovirus genus of the Astroviridae. Culture of avian nephritis virus (ANV) is difficult, with chicken kidney cells being the most commonly used medium, and even then growth and induction of cytopathic changes appear to be influenced by conditions in cell cultures and the strain of ANV (98).

10.1.2. OIE list

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

10.1.3. New Zealand status

ANV is not listed in the unwanted organisms register.

ANV has not been isolated in New Zealand but antibodies to the virus have been identified in pooled sera tested overseas and renal pathology consistent with the disease has been observed (94).

10.1.4. Epidemiology

ANV infections are commonly subclinical or associated with only mild clinical signs including transient diarrhoea in one-day-old chickens, reduced weight gain from one to two weeks of age, and runting and/or nephropathy in older birds (98). There are few reports of disease in the literature. The virus is widely distributed in many countries with evidence coming from serological surveys such as those in England (99), Ireland (100), and Hungary (101). Surveys have also identified the presence of the virus in SPF flocks (102). ANV is transmitted through direct or indirect contact and there is only circumstantial evidence suggesting vertical transmission (98).

Although the published evidence for ANV in New Zealand is scant, it is consistent with the situation in other countries prior to active surveillance for the organism. On the basis of Howell's report (94) of positive serology and pathology consistent with ANV infection, it is concluded that ANV is present in this country.

10.1.5. Hazard identification conclusion

On the basis that ANV is present in New Zealand and that the strain(s) present are able to cause disease, it is concluded that ANV is not a potential hazard in the commodity.

11. Salmonellae

11.1. HAZARD IDENTIFICATION

11.1.1. 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. 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 (103).

11.1.2. OIE list

The only *Salmonella* serotype affecting poultry and included in the OIE list of notifiable diseases is *S.* Gallinarum-Pullorum.

11.1.3. New Zealand status

The salmonellae designated as unwanted organisms are *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).

11.1.4. Epidemiology

Salmonella Abortusovis

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

Salmonella Dublin

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. For example, during 2002 over 80 percent of *Salmonella* cases in cattle were attributed to *S.* Dublin, whilst less than 1 percent of porcine salmonellosis cases were associated with *S*. Dublin and a smaller proportion (not reported) in chickens (104).

Salmonella Typhimurium PT 44

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. In the reports discovered all isolations have been from humans or cattle. 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.

Salmonella 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*) (105). 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:$Z_{4,}Z_{32}$ and 18:Z_4,Z_{23} cause serious disease in turkeys. Chickens are affected infrequently and infection of humans, sheep, and dogs, have also been reported (106-108).
2. *S. diarizonae* serotypes 61:k:1,5 and 61:1,v:5 are common is sheep and there have been small numbers of isolations of each from humans, snakes, and other reptiles (106, 107).
3. Snakes, turtles, other reptiles, and amphibians are infected by a wide range of serotypes of *S. arizonae* (106, 107). Weiss (107) reported the identification of 51 serotypes from snakes with 17 of those 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.

11.1.5. Hazard identification conclusion

On the basis of host specificity and/or host preference, *S.* Abortusovis, *S.* Dublin, *S.* typhimurium PT 44, and *S. arizonae* and *diarizonae* (other than *S. arizonae* serotypes 18:$Z_{4,}Z_{32}$ and 18:Z_4,Z_{23}) are not classified as potential hazards in the commodity and are excluded from further consideration in this risk analysis.

S. Gallinarum-Pullorum, *S. arizonae* serotypes 18:$Z_{4,}Z_{32}$ and 18:Z_4,Z_{23}, *S.* Typhimurium DT104, *S.* Enteritidis PT4, and *Salmonella* spp. (exotic, affecting animals) are classified as potential hazards in the commodity. In addition, all salmonellae reported to be egg borne will be considered.

12. *Salmonella enterica* subsp. *enterica* serovar Gallinarum-Pullorum

12.1. HAZARD IDENTIFICATION

12.1.1. 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 (109). Because of changes in nomenclature and because of the existence of chicken and turkey host-adapted strains, the literature is dominated by references to *S.* Gallinarum and *S.* Pullorum.

12.1.2. OIE list

S. Gallinarum-Pullorum is included in the OIE list of notifiable diseases.

12.1.3. New Zealand status

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

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

12.1.4. Epidemiology

The natural hosts for *Salmonella enterica* subsp. *enterica* serovar Gallinarum-Pullorum (*S.* Gallinarum-Pullorum) are chickens. The organism occurs in most countries. Following infection, flock morbidity and mortality can be highly variable. Transovarial 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 (109).

12.1.5. Hazard identification conclusion

S. Gallinarum-Pullorum is classified as a potential hazard in the commodity.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

S. Gallinarum-Pullorum has been eradicated from commercial flocks in the USA, Canada, Australia, and most of the countries in Western Europe (110). Outbreaks do still occur in some countries with incidents reported to the OIE from France, Italy, and Denmark during 2004 (111). Control or eradication programmes generally focus on commercial flocks and it is possible that flock infections arise from non-commercial birds.

The entry assessment is considered to be non-negligible.

12.2.2. Exposure assessment

Vertical transmission is a recognised component of the epidemiology of *S.* Gallinarum-Pullorum and horizontal transmission within a hatched group is highly likely. Although most commercial hatcheries maintain high levels of biosecurity protection, this cannot be relied upon to prevent early spread from an infected flock.

The exposure assessment is considered to be non-negligible.

12.2.3. Consequence assessment

S. Gallinarum-Pullorum infection within a flock could result in direct losses through illness and mortality. There may also be consequential losses due to control measures and disruption of international trade.

Infection may affect birds other than *Gallus gallus* but losses are likely to be restricted to the poultry industry and, possibly, non-commercial poultry. *S.* Gallinarum-Pullorum is highly host adapted to chickens and, to a lesser extent, turkeys. Clinical signs are seldom seen in other species (109). *S.* Gallinarum-Pullorum does not cause disease in humans or other non-avian species.

The consequence assessment is considered to be non-negligible.

12.2.4. Risk estimation

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

12.3. RISK MANAGEMENT

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

i. The likelihood of eggs carrying *S.* Gallinarum-Pullorum could be greatly reduced by testing birds in the laying flock to ensure that they do not carry infection and importing eggs only from flocks recognised by the appropriate veterinary administration as being free of infection.

ii. Chapter 6.3 of the OIE *Code* (1) describes requirements for monitoring poultry breeding flocks and hatcheries for Salmonella (article 6.3.9). Assurance could be required that eggs come from breeding establishments where this testing has not identified *S.* Gallinarum-Pullorum.

13. *S. arizonae* serotypes 18:Z_4,Z_{32} and 18:Z_4,Z_{23}.

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

S. arizonae serotypes 18:Z_4,Z_{32} and 18:Z_4,Z_{23} are covered in this section.

13.1.2. OIE list

Salmonella arizonae is not included in the OIE list of notifiable diseases.

13.1.3. New Zealand status

S. arizonae is listed in the register of unwanted organisms.

13.1.4. Epidemiology

S. arizonae is a recognised pathogen of turkeys (112) and Crespo *et al*. (108) reported epidemiological evidence that *S. arizonae* is transmitted vertically in turkeys.

Silva *et al*. (113) reported naturally occurring disease in chickens attributable to *S. arizonae* (18:Z_4,Z_{32}). Birds showed signs of blindness, central nervous system disease, and death with 5 percent of the birds affected. The ability of *S. arizonae* to penetrate the shells of chicken eggs and infect embryos after dipping of eggs into broth cultures (114) and to infect chickens following either subcutaneous or oral inoculation (115) has been demonstrated.

13.1.5. Hazard identification conclusion

It is concluded that *S. arizonae* is classified as a potential hazard in the commodity.

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

In their report of arizonosis in chickens, Silva *et al*. (113) noted that they had been unable to find earlier reports of *S. arizonae* infection in that species. Searches by this author have failed to reveal any other reports. Literature searches have not identified reports of *S. arizonae* being found in microbiological examinations of chicken eggs or derived products. On the basis that only one report of *S. arizonae* infection in chicken has been located and that being from Sao Paulo in 1980, it is concluded that the likelihood of *S. arizonae* infection in the commodity is very low.

The entry assessment is considered to be negligible.

13.2.2. Risk estimation

Because the entry assessment is negligible, the risk estimate is negligible and *S. arizonae* is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

14. *Salmonella* Typhimurium DT 104 carrying resistance to several antibiotics.

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

This section considers *S.* Typhimurium DT104 carrying resistance to several antibiotics. Non-multi-resistant strains of *S.* Typhimurium DT104 fall into the same category as "other salmonellae" considered in section 16. Multi-resistant strains have been of particular concern because of a global epidemic of such strains and the difficulties in treating infected humans.

14.1.2. New Zealand status

S. Typhimurium DT104 is listed in the register of unwanted organisms.

S. Typhimurium DT104 is isolated from humans and non-human sources in New Zealand relatively infrequently. In his review, MacDiarmid (116) reported that *S.* Typhimurium DT104 had been isolated from seven non-human samples between 1997 and 2001 and that three of the isolates were multi-drug resistant. From 1992 to 2001, 37 of 39 isolates from human sources were multi-drug resistant. None of these isolates came from poultry or poultry environments. Strains carrying resistance to several antibiotics are of particular concern to public health authorities.

14.1.3. Epidemiology

Although multi-resistant *S.* Typhimurium DT104 (DT104) is of prime concern in this risk analysis the epidemiology is similar to that of non-resistant strains. DT104 has a broad host range including cattle, sheep, goats, pigs, poultry, humans, dogs, cats, horses, and a number of other species (117-120).

In Britain, the earliest reports of DT104 were from humans in the early 1960s (118-120). The first isolations of a multi-resistant strain were from a migratory gull and an imported parrot in 1984, with further isolations from imported exotic birds during 1985 and 1986 (121). These isolations were followed by an epidemic of multi-resistant DT104 strains involving cattle, sheep, pigs, poultry, and other species that peaked in cattle in 1995 (122) and has since declined (123). Cattle are considered to be the principle reservoir host. 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 2004 (124).

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 (125), in parallel with that in Great Britain. Threlfall (119) identified reports of human infections with multi-resistant *S.* Typhimurium DT104 through much of Europe, the Middle East, South Africa, Trinidad, and the Philippines. Data illustrating recent trends outside Great Britain has not been located so the extent to which the epidemic is following the downward trend evident in Great Britain is unknown.

DT104 has not been reported from poultry sources in either Australia or New Zealand.

Although infection of poultry (particularly turkeys but also chickens) with DT104 is widely recognised, its epidemiology in these species has not been well documented. It has been thought that some *Salmonella* 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 (117, 126, 127). Even during the period following the recognition of the epidemic of DT104 this organism ranked lowly amongst *Salmonella* isolates from poultry and poultry-related environments in the USA (128, 129) and the Netherlands (130). In the United Kingdom during 2004, routine monitoring of the *Salmonella* status of commercial poultry flocks revealed no DT104 infections in breeding flocks and only 3 percent of all *Salmonella* isolates from broiler and layer flocks were identified as *S*. Typhimurium.

Establishment of infection in the intestinal tract and faecal excretion of DT104 by chickens (131, 132), and contamination of poultry shed environments (133) and feedstuffs (128) have been demonstrated.

Infection of eggs following experimental infection of chickens with DT104 has been demonstrated (131). Leach *et al*. (134) found that contamination of eggs was more frequent when hens had been infected intranasally than when infected orally. The significance of this finding is uncertain as it is generally considered that the faecal-oral route of infection is the most common means of transmission of salmonellae. Surveys, using samples of six boxed eggs, found prevalences of 0.01 percent in England and Wales in 1991, 0.04 percent in England in 1995/6 and nil from 4,753 samples from throughout the UK in 2003 (135). Benson *et al*. (129) reported that 250,000 eggs, from the north-eastern United States, had been examined for salmonellae with very few *S*. Typhimurium isolates and with none of them being DT104.

14.1.4. Hazard identification conclusion

On the basis that infection of eggs with DT104 is known to occur, and the ability of DT104 to cause disease in humans, it is concluded that *S*. typhimurium DT104 is classified as a potential hazard in the commodity.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

Although available evidence indicates that DT104 infection of eggs is rare, for reasons described above the entry assessment is considered to be non-negligible.

14.2.2. Exposure assessment

Although confirmatory evidence has not been sighted, the likelihood of infection of hatchlings from infected eggs is considered non-negligible. The exposure assessment is therefore considered to be non-negligible.

14.2.3. Consequence assessment

The consequences of the introduction of DT104 with chickens hatched from imported eggs is likely to be limited to the commercial operations of importing companies so long as

importations are into breeding establishments with hatchery systems meeting the standard required by the Poultry Industry Association of New Zealand (Inc.) (3) and the Egg Producers Federation of New Zealand (Inc). These codes of practice impose requirements for *Salmonella* monitoring and control throughout the hierarchy of breeding establishments. Costs arising from detection of DT104 in a grandparent or parent breeding hatchery would be considerable but limited to the company.

Importation of the commodity through channels other than commercial operations and without the support of subsequent screening programmes under quarantine-like conditions carries a likelihood that DT104 could establish in non-commercial poultry with the potential to lead to infection in livestock and humans.

Although some of the earliest reports of multi-resistant strains of DT104 came from wild birds and a parrot, the reservoir host is considered to be cattle. As a pathogen in wild birds, multi-resistant DT104 presents no greater hazard than the wide range of salmonellae already present in New Zealand. DT104 is not considered to be a hazard to the environment.

The consequence assessment is considered to be non-negligible.

14.2.4. Risk estimation

Because the entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and DT104 is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

14.3. RISK MANAGEMENT

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

i. Chapter 6.3 of the OIE *Code* (1) describes requirements for monitoring poultry breeding flocks and hatcheries for Salmonella (article 6.3.9). Assurance could be required that eggs come from breeding establishments where this testing has not identified *S.* Typhimurium DT104.

ii. Birds in the laying flock could be tested to ensure that they do not carry infection and eggs could be imported only from flocks recognised by the appropriate veterinary administration as being free of infection. The OIE *Manual* does not prescribe a test for international movement of animals or animal products although agent identification is listed as an “alternative test” suitable for the diagnosis of disease within a local setting, and for use in the import/export of animals after bilateral agreement (34). There are numerous methods for isolation of *Salmonella* in use world-wide and culture techniques using pre-enrichment, enrichment, or selective plating media are described by the OIE (34).

15. *Salmonella* Enteritidis

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Although only phage type 4, from amongst the range of *S.* Enteritidis phage types, is listed in the New Zealand register of unwanted organisms, this section covers all phage types.

15.1.2. New Zealand status

S. Enteritidis phage type 4 (PT4) is the second most common *S.* Enteritidis phage type isolated from humans in New Zealand with 128 isolates being recorded between 2000 and 2004 (116). A high proportion of human isolates come from people with histories of recent international travel (136). Seven isolates have been recorded from non-human sources between 1990 and 2003 with none of those being recorded from poultry or poultry related sources (116, 136).

15.1.3. Epidemiology

The earlier stages (1979 to 1987) of an international pandemic of human disease attributed to *Salmonella* Enteritidis affecting North and South America, Europe, and possibly southern Africa were described by Rodrigue *et al*. (137). Human cases were attributed to consumption of eggs or poultry from infected chickens. The decline in *S.* Enteritidis incidents in poultry followed introduction of control measures and codes of practice in the industry. This pandemic was associated with *S.* Enteritidis PT4 in Great Britain and continental Europe (138) or with PT13, PT8, and PT4 (in declining order) in North America (139).

The on-farm epidemiology of *S.* Enteritidis and the basis for the development of the pandemic have been reviewed by Guard-Petter (141). In a retrospective view of the epidemic of S. Enteritidis, Rabsch *et al*. (141) 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 (142-144) and the decline in flock immunity following control of *S.* Gallinarum-Pullorum allowed widespread infection of poultry.

In her review, Guard-Petter (140) generalises in saying that *S.* Enteritidis causes no clinical disease in chickens but has a unique characteristic of infecting the eggs of infected hens. Although these comments might not apply to all phage types of *S.* Enteritidis, they appear to be relevant to those that have contributed to the pandemic of human disease originating from poultry. Infection is located both in the egg (145) and on the surface of shells (145-147). Eggs are considered to be the major source for human *S.* Enteritidis infections. Control of *S.* Enteritidis in hen houses is difficult because of the ability of the organism to survive in the environment and because of its ability to infect other species, including mice, which can reintroduce the organism even after otherwise adequate cleaning. *S.* Enteritidis may infect eggs prior to shell formation and vertical transmission to hatchlings may occur (145). In a small proportion of birds, *S.* Enteritidis PT4 infection may be maintained until birds enter lay (148).

The European Union Council Directive 92/117/EEC requires that all countries have mandatory procedures in place to control *Salmonella* infections in poultry breeding and production systems. In the UK, implementation of these procedures has been followed by marked reductions in *Salmonella* infections in poultry, especially in breeding hatcheries where there has been only one episode during the years 2002 to 2004 (104, 124, 149), which was in an establishment producing eggs for pharmaceutical use. Comparable reports of the status in other EU countries or in the United States have not been located. *S.* Enteritidis is reported in Australia but is not established in the poultry industry.

15.1.4. Hazard identification conclusion

It is concluded that *S.* Enteritidis is classified as a potential hazard in the commodity.

15.2. RISK ASSESSMENT

15.2.1. Entry assessment

On the basis that *S.* Enteritidis may infect eggs, the entry assessment is considered to be non-negligible.

15.2.2. Exposure assessment

Vertical transmission of *S.* Enteritidis has been demonstrated. There is a non-negligible likelihood that hatchlings from infected eggs will be infected and that some hatchlings will excrete organisms contaminating the environment, thus leading to infection of others.

The exposure assessment is considered to be non-negligible.

15.2.3. Consequence assessment

The consequences of the introduction of *S.* Enteritidis with chickens hatched from imported eggs is likely to be limited to the commercial operations of importing companies so long as importations are into commercial breeding establishments with hatchery systems meeting the standard required by the Poultry Industry Association of New Zealand (Inc.) (3) and the Egg Producers Federation of New Zealand (Inc). These codes of practice impose requirements for *Salmonella* monitoring and control throughout the hierarchy of breeding establishments. The difficulty of eradicating *S.* Enteritidis from poultry sheds is such that costs of establishment of such infection in a grandparent or parent breeding hatchery would be high.

Importation of the commodity through channels other than commercial operations and without the support of subsequent screening programmes under quarantine-like conditions carries a likelihood that *S.* Enteritidis could establish in non-commercial poultry, with the potential to lead to infection in livestock and humans. *S.* Enteritidis is not a hazard to the environment.

The consequence assessment is considered to be non-negligible.

15.2.4. Risk estimation

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

15.3. RISK MANAGEMENT

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

i. Chapter 6.3 of the OIE *Code* (1) describes requirements for monitoring poultry breeding flocks and hatcheries for Salmonella (article 6.3.9). Assurance could be required that eggs come from breeding establishments where this testing has not identified *S.* Enteritidis.

ii. Birds in the laying flock could be tested to ensure that they do not carry infection and eggs could be imported only from flocks recognised by the appropriate veterinary administration as being free of infection. The OIE *Manual* does not prescribe a test for international movement of animals or animal products although agent identification is listed as an "alternative test" suitable for the diagnosis of disease within a local setting, and for use in the import/export of animals after bilateral agreement (34). There are numerous methods for isolation of *Salmonella* in use world-wide and culture techniques using pre-enrichment, enrichment, or selective plating media are described by the OIE (34). In addition, indirect and competitive ELISAs are described for serological diagnosis of *S.* Enteritidis infection, and the whole blood test for immunological diagnosis of *S.* Gallinarum-Pullorum may be used to detect *S.* Enteritidis although the sensitivity of this test is low (34).

16. Other Salmonellae

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

The salmonellae addressed in this section are those serotypes and phage types not covered in Sections 12 to 15.

16.1.2. New Zealand Status

In New Zealand, over the period 1999 to mid 2005, typing of *Salmonella* isolates from humans yielded over 140 serotypes/phage types (150-154). During the same period typing of isolates from animals, animal feeds and their environment yielded over 80 serotypes/phage types (150-153, 155). The frequency with which specific types were isolated during each year varied greatly with many of the serotypes/phage types being 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 and most were from travellers or immigrants. With many *Salmonella* infections being subclinical, the full range of serovars and phage types present in New Zealand and the extent of introductions to the country are unknown.

16.1.3. 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 organisms surviving for varying periods of time in different environmental niches. Host specificity or host preference varies between *Salmonella* serotypes and phage types.

Transovarial transmission requires that the organism infects the ovary and/or oviduct (156). 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 (157). The *Salmonella* serovars/DTs infecting the reproductive tracts of chickens and turkeys are highly host adapted and De Buck *et al* (156) observed that tropism for the reproductive tract is shown by *S.* Abortusequi and *S.* Abortusovis, both of which are highly host adapted for horse and sheep respectively.

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 contamination following the hatching of infected eggs (158).

16.1.4. Hazard identification conclusion

As the commodity definition requires that eggs be sanitised to OIE standards and that infection of the contents of clean dry eggs with intact shells will arise only from host-adapted strains of salmonellae, the likelihood of infection by miscellaneous salmonellae is very low.

Given the requirement of other biosecurity considerations that breeding establishments and hatcheries must comply with OIE standards, it is concluded that “other salmonellae” are not classified as a potential hazard in the commodity.

17. *Ornithobacterium rhinotracheale*

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

Ornithobacterium rhinotracheale is a pleomorphic gram-negative rod which grows slowly on agar with 5 percent sheep blood in an atmosphere of 5 to 10 percent CO_2 (159).

O. rhinotracheale was first identified in 1993. Subsequent investigations showed that the bacterium had been present in Germany in turkeys since 1981 and rooks since 1983, and the organism had also been isolated in Belgium and the United States prior to 1990 (159).

Van Empel (159), 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."

17.1.2. OIE list

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

17.1.3. New Zealand status

O. 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 leave open the possibility that *O. rhinotracheale* may be present (see comments by Van Empel quoted above). Birds from an unknown number of flocks in New Zealand have been tested for *O. rhinotracheale* using imported ELISA kits with negative results (Les With, Poultry Veterinary Services, quoted in reference (57)).

17.1.4. Epidemiology

O. rhinotracheale is widespread in poultry flocks in the absence of disease and it is doubtful if it should be regarded as a primary pathogen. Contribution to clinical 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 disease virus aggravated the effects of *O. rhinotracheale*. In broilers, 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.

Surveys of poultry flocks in the north-central region of the United States (160), Turkey (161), and Brazil (162) have all shown high proportions of flocks to be infected and prevalence within layer flocks to be in excess of 50 percent. ELISAs have proven to be effective tools for survey purposes.

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) (168). *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 collected from 1997 to 1999 confirmed that the organism had infected approximately 13 percent of both meat and laying birds in at least six prefectures during those years (169). In the north central United States serological testing showed infection to be present in 90 to 100 percent of layer flocks and 43 to 52 percent of younger birds (160). Seroprevalence in infected breeding flocks is high with between 65 percent and 95 percent of birds in infected flocks returning positive serology results (162, 170, 171).

Reports of *O. rhinotracheale* isolations have been from Galliformes (partridge, pheasant, quail, chicken, turkey, guinea fowl), Struthioniformes (Ostrich), Anseriformes (duck, goose) and Passeriformes (rook) (159). Reports which confirm association between *O. rhinotracheale* and disease, however, are restricted to Galliformes.

No reports of *O. rhinotracheale* in Australia have been located and Australia treats this organism as one of concern in the importation of birds. On that basis, it is considered that *O. rhinotracheale* is not present, or recognised, in Australia.

Van Empel and others have reported that vertical transmission (either through transovarial or through cloacal contamination of eggs) can occur although contamination rates of egg shells and contents are very low (159, 163). These reports are supported by that of van Veen *et al*. (164) which provided experimental epidemiological evidence of infection being transmitted from infected turkey hatchlings to SPF chickens in incubators. Further evidence of vertical transmission is provided by El-Gohary (165), who reported that *O. rhinotracheale* was recovered from chick embryos and one-day-old chicks sampled from hatcheries, where low hatchability and high mortality in newly hatched chicks were being observed. Back *et al.* (166) were able to isolate *O. rhinotracheale* from the ovaries and oviduct of turkey breeder hens when these birds were artificially infected by either the intranasal, intravenous, or intramuscular route and concluded that the possibility of egg transmission of this agent could not be ruled out.

Varga *et al*. (167), on the other hand, using 12 strains of *O. rhinotracheale*, were unable to isolate organisms from inoculated egg shells after 24 hours at 37°C or from inoculated eggs beyond 14 day post inoculation. These authors concluded that *O. rhinotracheale* is not transmitted via eggs.

17.1.5. Hazard identification 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 classified as a potential hazard in the commodity.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

Vertical transmission through transovarial or cloacal contamination of eggs has been described and *O. rhinotracheale* has been recovered from chick embryos and one-day-old chicks. Therefore, although there is some conflict in reports as to whether eggs can carry infection and whether vertical transmission can take place, on balance it is concluded that there is sufficient evidence to suggest a non-negligible likelihood that chicken eggs imported from countries where *O. rhinotracheale* is present could be infected.

The entry assessment for *O. rhinotracheale* in the commodity from Europe and North America is considered to be non-negligible.

The entry assessment for *O. rhinotracheale* in the commodity from Australia is considered to be negligible.

17.2.2. Exposure assessment

Reports indicate that infected eggs can result in infected hatchlings and that the organism can be transmitted to other birds in contact.

The exposure assessment is considered to be non-negligible.

17.2.3. Consequence assessment

Many infections with *O. rhinotracheale* are subclinical but disease does occur in poultry, especially in chickens and turkeys. The severity of disease is affected by environmental conditions and by the presence of other diseases. In some incidents disease can be severe with clinical signs of respiratory disease, production losses and mortalities.

Infection has not been reported from humans, so *O. rhinotracheale* does not pose a risk to human health.

O. rhinotracheale has not been reported to cause disease in non-poultry species or in non-avian hosts. Therefore, the organism does not pose a risk to New Zealand's native fauna.

As a result of the potential impact of *O. rhinotracheale* on the poultry industry in New Zealand, the consequence assessment is considered to be non-negligible.

17.2.4. Risk estimation

Because the entry assessment for eggs imported from Australia is negligible, the risk estimate is negligible and *O. rhinotracheale* is not classified as a hazard in the commodity imported from Australia.

For the commodity imported from countries other than Australia, since the entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and *O. rhinotracheale* is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

17.3. RISK MANAGEMENT

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

i. Tools available for detection of infected flocks and/or infected birds include the culture of swabs from the infraorbital sinus or nasal cavity although these can provide false negative results, especially when plates are overgrown with fast growing contaminants.

ii. ELISAs could be used which cross-react with different serotypes to test parent flocks. Commercial ELISA kits are available in Europe. Serum plate agglutinations tests (SPAT) are also available which may be serotype-specific, although these may have low (65 percent) sensitivity (163).

    Eggs to be imported from Europe, Canada or the United States could come only from flocks shown to be free of *O. rhinotracheale* on the basis of serological testing of a sample of laying birds. Testing 60 samples would detect a seroprevalence of at least 5% in a flock with 95% confidence. A sample size of 90 would be required to detect a seroprevalence of at least 5% in a flock with 99% confidence.

iii. Given that infected flocks could be expected to have a seroprevalence of 50-95% (see 3.8.1.4), a smaller sample size could be considered. For example, 11 samples would enable detection of a seroprevalence of at least 25% in a flock with 95% confidence (16 samples for 99% confidence), whereas 5 samples would detect a seroprevalence of at least 50% with 95% confidence (7 samples for 99% confidence).

iv. Eggs could be imported from Australia without sanitary measures for *O. rhinotrcheale*.

18. *Mycoplasma iowae*

18.1. HAZARD IDENTIFICATION

18.1.1. 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.

18.1.2. OIE list

M. iowae is not included in the OIE list.

18.1.3. New Zealand status

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

18.1.4. Epidemiology

The primary host of *M. iowae* is thought to be turkeys and, although lateral transmission occurs, the main means of spread 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 (173, 173). Following administration of infected semen, the organism establishes infection in the oviduct and large numbers of eggs may become infected (173, 174). *M. iowae* causes a range of clinical signs in turkeys, especially decreased egg hatchability (2 to 5 percent reduction) (172). There are differences in opinion as to the significance of *M. iowae* as a pathogen, even to extent that differing views may be expressed by the same author. For example Jordan in 1985 (174) and Bradbury in 2001 (175) included *M. iowae* as one of four economically important avian mycoplasmas, while in 1996 Al-Ankari and Bradbury (176) 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 (pers. comm.[6]) 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".

Bradbury and Klevin (172) stated that "isolation of *M. iowae* from chickens is not uncommon" and experimental infection of chick embryos and of recently hatched chickens has been demonstrated (177-179). The only reports of naturally occurring infection of chickens located by this author are those of Yoder and Hofstad (180), Bencina *et al*. (181), and Aly *et al*. (182). The report from Bencina *et al*. is clear that the chickens infected with *M. iowae* were from a mixed age flock in which birds were reared in close contact and several species of *Mycoplasma* were present. Comparable information is less clear from the other reports but that of Yoder and Hofstad states that isolation of up to three strains of *Mycoplasma* from individual chickens was not uncommon, suggesting that flock hygiene was

[6] Bradbury, J.M. Personal communication to Bruce Simpson 23 July 2004.

poor. No reports of *M. iowae* being isolated from natural infections in housed chickens managed under good hygienic conditions have been found.

No reports of *M. iowae* in Australia have been located and Australia treats this organism as one of concern in the importation of turkeys. On that basis, it is considered that *M. iowae* is not present in Australia.

18.1.5. Hazard identification conclusion

It is concluded that *M. iowae* is classified as a potential a hazard in the commodity imported from Europe or North America.

It is concluded that *M. iowae* is not classified as a potential hazard in the commodity imported from Australia.

18.2. RISK ASSESSMENT

18.2.1. Entry assessment

M. iowae is only likely to be present in eggs from unhoused, poorly managed chicken flocks exposed to other bird species and with multiple age groups in contact with one another. However, it is unlikely that *M. iowae* would be present in eggs from well managed breeding flocks. On that basis, the entry assessment for *M. iowae* in the commodity is considered to be negligible.

18.2.2. Risk estimation

Because the entry assessment is negligible, the risk estimate is negligible and *M. iowae* is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

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