Import Risk Analysis:

Egg powders from all countries

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May 2008

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

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Policy and Risk
MAF Biosecurity New Zealand

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1.   Executive Summary

This document is a qualitative analysis of the biosecurity risks associated with the import of shelf-stable spray-dried powders, prepared from clean eggs, containing less than 100mg/kg of eggshell remains, egg membrane, and other particles, into New Zealand from all countries.

For imported whole egg, egg yolk, and egg albumen powders, heat-resistant variants of exotic group 1 avian adenoviruses associated with hydropericardium syndrome (Angara disease) (FAdV-4) are considered to be a hazard. Options presented for FAdV-4 risk management include:

   i.   Assurance that eggs used have been derived from flocks in countries or geographic regions where Angara disease has not been recognised.

   ii. Testing to ensure source flock freedom from FAdV-4.

   iii. Further heat treatment of manufactured powders to destroy any FAdV-4 present.

Exotic avian influenza (AI) viruses are considered to be a hazard in imported egg albumen powders. AI viruses are not considered to be a hazard in whole egg and egg yolk powders. Options presented for AI virus risk management include:

   i.   Further heat treatment of manufactured egg albumen powders that have not already been subject to heat treatment proven to destroy AI viruses.

   ii. Testing source flocks to ensure freedom from AI viruses.

   iii. Certification that eggs used to manufacture egg albumen powders have originated from flocks in countries, zones, or compartments which are free of notifiable AI as described in Article 2.7.12.3 of the OIE *Code*.

2. Introduction

This risk analysis has been developed in response to a request from the Animals Import Section of MAF Biosecurity New Zealand. The risk analysis will be used as basis for an import health standard (IHS) for the importation of egg powders from all countries into New Zealand.

3. Scope

This qualitative risk analysis is limited to the description of the biosecurity risks involved in the importation of shelf-stable dried powders derived from hens' eggs (*Gallus gallus*).

4. Commodity Definition

4.1 COMMODITY DESCRIPTION

The commodities considered in this risk analysis are shelf-stable dried powders derived from hens' eggs (*Gallus gallus*). Three product types are under consideration, namely whole egg powder, egg albumen powder, and egg yolk powder. Products imported into New Zealand from all countries are under consideration.

4.2 PROCESSING

A number of international standards exist regarding the cleanliness of eggs used in the manufacture of egg products such as egg powders. For example:

> EEC directive 89/437/EEC Chapter V (special hygiene requirements for the manufacture of egg products); para 4 specifies that "dirty eggs must be cleaned before being broken; this must be carried out in a room which is separate from the breaking room or from any room where exposed egg contents are handled. Cleaning procedures must be such as to prevent contamination or adulteration of the egg contents. Shells must be sufficiently dry at the time of breaking to prevent adulteration of the egg contents by the remains of the cleaning water."

> Canadian Processed Egg regulations require that 4. (1) (c) processed eggs shall be prepared from eggs that are free from dirt and other foreign matter.

Standards also exist to limit the quantity of eggshell which is permitted in the final product:

> EEC directive 89/437/EEC Chapter VI (analytical specifications); para 2(c) specifies that "the quantity of eggshell remains, egg membrane, and other particles in the egg product must not exceed 100 mg/kg of egg product."

Several stages are involved in the manufacture of these egg products. Eggs are washed, sanitised, scanned, broken, and filtered. Egg yolk and whole egg products then undergo high temperature short time (HTST) pasteurisation. Products are then spray dried at an inlet temperature of 155°C to 175°C and outlet temperature of 70°C to 80°C. Following the spray drying process, egg powders may also undergo hot room treatment before storage and shipping. Following drying, albumen powders undergo a prolonged final hot room treatment.

Further discussions with importers of these products have indicated the following heat treatment conditions may be used during manufacture:

Table 1. Heat treatments used in the manufacture of egg powders

Product	Min Temperature	Holding Time
Brand X whole egg	65.5 °C	3 minutes
Brand X egg albumen	64 °C	14 days
Brand X egg yolk	65.5 °C	4 to 6 minutes
Brand Y whole egg	60 °C	3.5 minutes
Brand Y egg yolk	60 °C	3.5 minutes
Brand Y egg albumen	60 °C	8 days
Brand Y enzyme modified egg yolk	65 °C	3.5 minutes
Brand Z whole egg	62 °C	3.5 minutes
Brand Z egg albumen	54.4 °C	7 days

Other final treatments that have been documented for egg albumen powders (Baron et al 2003) include storage at 67°C for about 15 days and storage at 75°C to 80°C for 15 days.

Article 3.6.5.1 of the current *Terrestrial Animal Health Code* of the World Organisation for Animal Health (OIE 2007) describes the following times for industry standard temperatures as being suitable for the inactivation of highly pathogenic notifiable avian influenza (HPNAI) virus present in egg products:

Table 2. OIE conditions for the inactivation of HPNAI virus present in eggs and egg products

Product	Temperature	Time
Whole egg	60 °C	188 seconds
Whole egg blends	60 °C	188 seconds
Whole egg blends	61.1 °C	94 seconds
Liquid egg white	55.6 °C	256 seconds
Liquid egg white	56.7 °C	228 seconds
10% salted yolk	62.2 °C	138 seconds
Dried egg white	67 °C	0.83 days
Dried egg white	54.4 °C	21.38 days

Based on manufacturer's data, this document will examine the risks associated with whole egg powders and egg yolk powders pasteurised at greater than 60°C for a period of no less than 3.5 minutes and egg albumen powders which undergo a final heat treatment of greater than 54.4°C for a period of no less than 7 days.

4.3   USE OF IMPORTED COMMODITIES

Most of these commodities are used in the food industry, predominantly involving further cooking. Some egg powders will also be marketed direct to consumers, specifically targeting the camping/sailing market.

4.4   COMMODITY DEFINITION CONCLUSION

The commodity for this risk analysis is defined as imported spray-dried egg powders prepared from clean[1] eggs, containing less than 100mg/kg of eggshell remains, egg membrane and

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[1] For the purposes of this risk analysis, 'clean' is defined as visibly free from dirt or other foreign matter. Any eggs which are not clean should be washed and dried to a standard equivalent to that described above for EEC directive 89/437/EEC Chapter V before processing – see section 4.2 (Processing).

other particles, that have been subject to pasteurisation at greater than 60°C for a period of no less than 3.5 minutes (egg powders and egg yolk powders), or a final heat treatment of greater than 54.4°C for a period of no less than 7 days (egg albumen powders).

5 Methodology of Risk Analysis

The methodology used in this risk analysis follows the guidelines as described in MAF Biosecurity New Zealand's *Risk Analysis Procedures – Version 1*[2] and in Section 1.3 of the *Terrestrial Animal Health Code* (OIE 2007).

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

5.1 HAZARD IDENTIFICATION

The first step in the risk analysis is hazard identification. The process begins with the collation of a list of organisms that might be associated with eggs used in the manufacture of this commodity (the preliminary hazard list). The diseases/agents of interest are those that could be transmitted in or on these eggs and could infect domestic, feral, or wild animals that occur in New Zealand, and man. In this case the preliminary hazard list was complied from *Diseases of Poultry*, 11th Edition, 2003, Ed Y.M. Saif, Iowa State Press. The diseases/agents identified in the preliminary hazard list are shown below in Table 3.

Organisms in the preliminary hazard list are subjected to further analysis to determine whether they should be considered potential hazards (see Section 6) and all organisms considered to be potential hazards are subjected to a full risk analysis.

5.2 RISK ASSESSMENT

Under the MAF Biosecurity New Zealand and OIE methodologies, risk assessment consists of:

a) Entry assessment -   the likelihood of the organism being imported in 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, exposure, 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 understand that not all of the above steps may be necessary in all risk assessments. The MAF Biosecurity New Zealand and OIE methodologies make it clear that if the likelihood of entry is negligible for a potential hazard, then the risk estimate is

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[2] See: www.biosecurity.govt.nz/files/pests/surv-mgmt/surv/review/risk-analysis-procedures.pdf

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.

Figure 1. The risk analysis process.
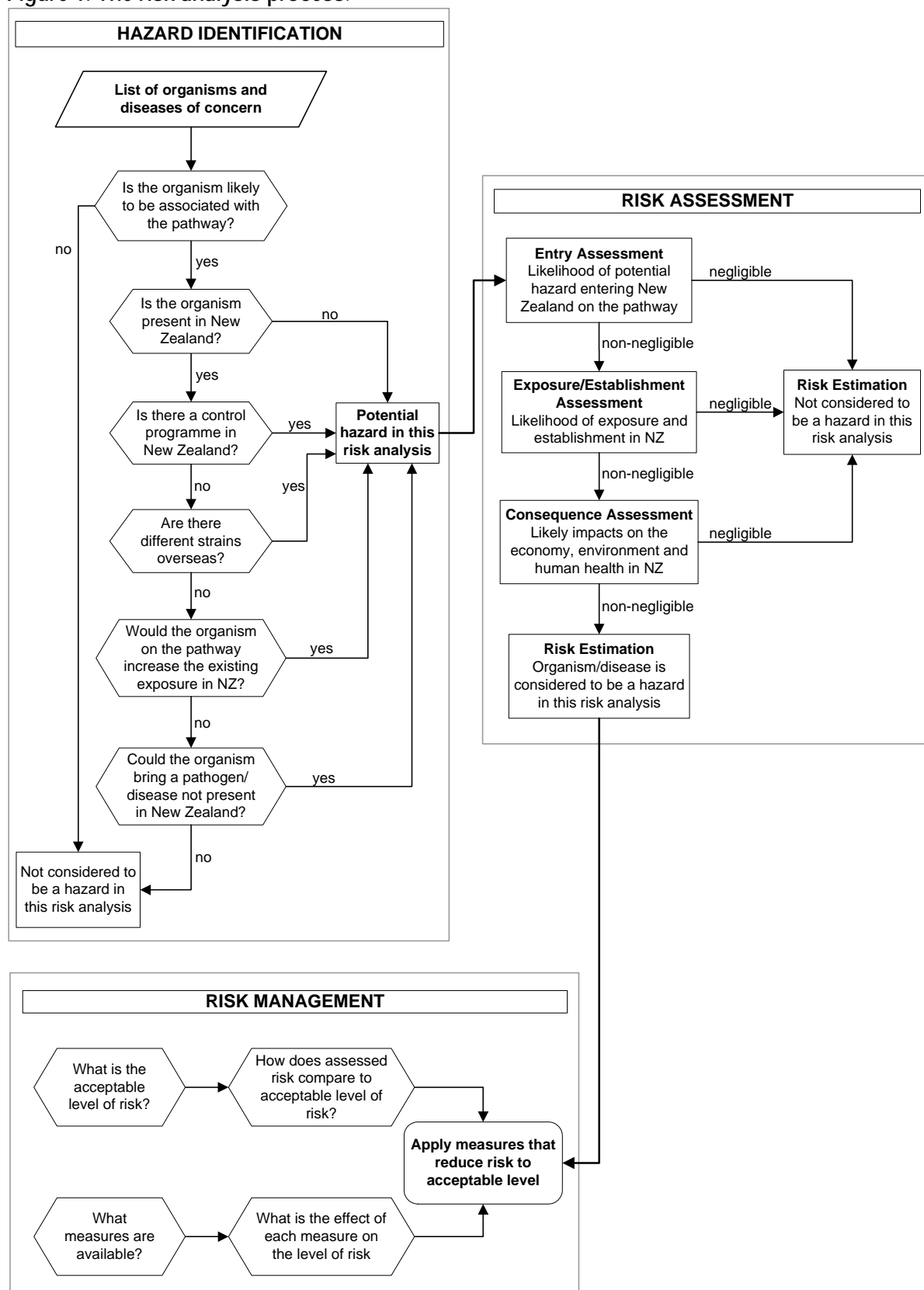
5.3   RISK MANAGEMENT

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).

5.4   RISK COMMUNICATION

This draft import risk analysis is issued 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 they consider necessary or preferable.

Following this period of public consultation on this draft document, a review of submissions will be produced and a decision-making committee will determine whether any changes need to be made to this draft risk analysis.

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

6. Preliminary Hazard List

Under MAF Biosecurity New Zealand's risk analysis framework, an assessment is made as to whether or not the risk good (as described in the commodity definition) could act as a potential vehicle for the introduction of organisms or diseases into New Zealand.

The criteria for classification as diseases/organisms not being potential hazards in this risk analysis are:
- Disease agents that are known to be present in New Zealand are not potential hazards.
- Disease agents that have not been demonstrated as being transmitted in eggs of *Gallus gallus* are not potential hazards.
- Disease agents that will be inactivated by the processing conditions defined for the commodity are not potential hazards.

If importation of a risk good is considered likely to result in an increased exposure of people to a potentially zoonotic organism, even if it is already present in New Zealand, then that organisms should also be considered a potential hazard.

Vertical transmission (i.e. transmission in or on eggs) is a feature of a number of poultry pathogens. These are listed in Table 3 in alphabetical order. This list was compiled from *Diseases of Poultry*, 11th Edition, 2003, Ed Y.M. Saif, Iowa State Press.

Table 3: Preliminary hazard list

Disease	Agent	OIE Notifiable	NZ Status	In egg	On or in shell
Angara disease	Aviadenovirus (Adenoviridae)	No	Exotic	Yes	Yes
Arizonosis	*Salmonella* Arizonae	No	Exotic	Yes	Yes
Avian encephalomyelitis	unassigned (Picornaviridae)	No	Present	Yes	Yes
Avian influenza	Influenzavirus A (Orthomyxoviridae)	Yes	Notifiable	Yes?	Yes
Avian leukosis/sarcoma	Alpharetrovirus (Retroviridae)	No	Exotic strains	Yes	Yes
Avian nephritis types 1-3	unassigned (Picornaviridae)	No	Present	Yes?	Yes?
Avian paramyxovirus types 2 & 3	Rubulavirus (Paramyxoviridae)	No	Exotic	Yes?	Yes?
Avian pox virus	Poxvirus (Poxviridae)	No	Present	No	Yes?
Avian spirochaetosis	*Borrelia anserine*	No	Exotic	No	Yes
Avian tuberculosis	*Mycobacterium avium intracellulare*	No	Exotic strains	Yes	Yes
Campylobacteriosis	*Campylobacter jejuni* and others	No	Exotic strains[3]	No	Yes
Chicken infectious anaemia	Gyrovirus (Circoviridae)	No	Present	Yes	yes
Colibacillosis	*Escherichia coli* 0111, 0157:H7 and others	No	Exotic strains[4]	Yes	Yes
Derzsy's disease (goose parvovirus infection)	Parvovirus (Parvoviridae)	No	Exotic	Yes?	Yes?

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[3] Campylobacteriosis does occur in New Zealand although the quinolone-resistant forms that have been documented overseas have not been seen in this country.

[4] Most *E.coli* serotypes recovered from birds are pathogenic only to birds although there is the potential for these organisms to act as a source of genetic material encoding for antimicrobial resistance and virulence factors (Chulasiri and Suthienkul 1989).

Table 3 (continued)

Disease	Agent	OIE Notifiable	NZ Status	In egg	On or in shell
Duck hepatitis types 1 & 3	unassigned (Picornaviridae)	Yes	Notifiable	No	Yes?
Duck hepatitis type 2	Astrovirus (Astroviridae)	Yes	Notifiable	No?	Yes?
Duck septicaemia	*Riemerella anatipestifer*	No	Exotic[5]	No	Yes?
Duck viral enteritis (duck plague)	unassigned (Herpesviridae)	No	Notifiable	Yes?	Yes?
Egg drop syndrome 76	Aviadenovirus (Adenoviridae)	No	Present	Yes	Yes
Fowl cholera	*Pasteurella multocida*	Yes	Notifiable	No	Yes?
Fowl typhoid	*Salmonella* Gallinarum	Yes	Notifiable	Yes	Yes
Group 1 adenovirus infections	Aviadenovirus (Adenoviridae)	No	Exotic strains	Yes	Yes
Haemorrhagic enteritis, Group 2 adenovirus splenomegaly	Aviadenovirus (Adenoviridae)	No	Exotic	No	Yes?
Infectious bronchitis	Coronavirus (Coronaviridae)	Yes	Exotic strains	Yes	Yes
Infectious bursal disease	Avibirnavirus (Birnaviridae)	Yes	Notifiable	No	Yes?
Infectious coryza	*Haemophilus paragallinarum*	No	Exotic	No	Yes?
Infectious laryngotracheitis	Herpesvirus (Herpesviridae)	Yes	Present	No	Yes?
Marek's disease	Herpesvirus (Herpesviridae)	Yes	Exotic strains	No	Yes?
Muscovy duck virus	Parvovirus (Parvoviridae)	No	Exotic	Yes?	Yes?
Mycoplasmosis	*M. gallisepticum*	Yes	Present	Yes	Yes?
	M. meleagridis	No	Exotic	Yes	No
	M. synoviae	Yes	Present	Yes	Yes?
	M. iowae	No	Exotic	Yes	Yes
Newcastle disease, APMV-1	Rubulavirus (Paramyxoviridae)	Yes	Notifiable	Yes?	Yes
Ornithobacteriosis	*Ornithobacterium rhinotracheale*	No	Exotic	Yes?	No
Paratyphoid salmonellae	*Salmonella* Enteritidis (vertical), and many other serotypes (by shell contamination)	No	Exotic	Yes	Yes
Psittacosis	*Chlamydophila psittaci*	Yes	Exotic strains[6]	Yes	Yes
Pullorum disease	*Salmonella* Pullorum	Yes	Notifiable	Yes	Yes
Quail bronchitis	Avian adenovirus type 1 (Adenoviridae)	No	Exotic	No?	Yes?
Reticuloendotheliosis	Gammaretrovirus (Retroviridae)	No	Present	Yes	Yes
Rotavirus infections	Rotavirus (Reoviridae)	No	Present	No	Yes?
Turkey rhinotracheitis	Avian pneumovirus (Paramyxoviridae)	Yes	Exotic	No	Yes?
Turkey viral hepatitis	unidentified (Picornaviridae)	No	Exotic	Yes?	Yes?
Viral arthritis	unassigned (Reoviridae)	No	Present	Yes	Yes?

Key: "Yes?" and "No?" indicate that there is some uncertainty surrounding whether the agent is associated with a particular transmission route. "Yes?" indicates that the balance of probabilities suggests that transmission may occur by that route, and "No?" indicates that, although it is unlikely, transmission cannot be discounted.

As described above, any diseases/organisms from the preliminary hazard list which are thought to be exotic to New Zealand, and are known to be transmitted in the eggs of *Gallus gallus,* and are unlikely to be inactivated by the processing conditions defined for this commodity are considered to be potential hazards requiring further risk assessment.

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[5] Although *Riemerella anatipestifer* was recorded in New Zealand in 1974, there have been no reports of this disease since then. Correspondence with the Investigation and Diagnostic Centre (Wallaceville), and with specialist poultry veterinarians in New Zealand suggests that this disease is now not present in commercial poultry.

[6] Psittacosis occurs in birds in New Zealand although some strains and serotypes are considered exotic.

6.1 DISEASE AGENTS PRESENT IN NEW ZEALAND

The following agents are known to be present in New Zealand. It is also considered that importation of egg powders would not provide an exposure to people in New Zealand that is significantly greater than currently occurs. These are therefore not considered to be potential hazards in this risk analysis:

- Avian encephalomyelitis virus, avian nephritis viruses types 1-3, avian pox virus, egg drop syndrome 76 virus, infectious laryngotracheitis virus, and reovirus (viral arthritis) are recognised as being present in New Zealand (Howell 1992). Chicken infectious anaemia virus, *Mycoplasma gallisepticum,* and *Mycoplasma synoviae* are also present in New Zealand poultry (Anon 2005a).

- Serological investigations indicate that, although clinical disease is unusual in New Zealand poultry, infection with reticuloendotheliosis virus is widespread (Howell et al 1982).

- Rotaviruses have been recovered from poultry in New Zealand (Saifuddin et al 1989).

- Avian tuberculosis (*Mycobacterium avium-intracellulare*) is known to be present in New Zealand (Anon 2005b). Exotic serovars of *Mycobacterium avium-intracellulare* may exist although no difference in pathogenicity between these and endemic strains has been reported.

From the preliminary hazard list, strains of the following disease/agents are considered to be exotic to New Zealand and require further consideration:

Angara disease (Adenoviridae)
Arizonosis (*Salmonella* Arizonae)
Avian influenza (Orthomyxoviridae)
Avian leukosis/sarcoma (Retroviridae)
Avian paramyxovirus types 2 & 3 (Paramyxoviridae)
Avian spirochaetosis (*Borrelia anserine*)
Campylobacteriosis (*Campylobacter jejuni* and others)
Colibacillosis (*Escherichia coli* 0111, 0157:H7 and others)
Derzsy's disease (goose parvovirus infection) (Parvoviridae)
Duck hepatitis types 1 & 3 (Picornaviridae)
Duck hepatitis type 2 (Astroviridae)
Duck septicaemia (*Riemerella anatipestifer*)
Duck viral enteritis (duck plague) (Herpesviridae)
Fowl cholera (*Pasteurella multocida*)
Fowl typhoid (*Salmonella* Gallinarum)
Group 1 adenovirus infections (Adenoviridae)
Haemorrhagic enteritis and group 2 adenovirus splenomegaly (Adenoviridae)
Infectious bronchitis (Coronaviridae)
Infectious bursal disease (Birnaviridae)
Infectious coryza (*Haemophilus paragallinarum*)
Marek's disease (Herpesviridae)
Muscovy duck virus (Parvoviridae)
Mycoplasmosis (*Mycoplasma meleagridis* and *M. iowae*)
Newcastle disease (Paramyxoviridae)

Ornithobacteriosis (*Ornithobacterium rhinotracheale*)
Paratyphoid salmonellae (*Salmonella* Enteritidis etc.)
Psittacosis (*Chlamydophila psittaci*)
Pullorum disease (*Salmonella* Pullorum)
Quail bronchitis (Adenoviridae)
Turkey rhinotracheitis (Paramyxoviridae)
Turkey viral hepatitis (Picornaviridae)

6.2   DISEASE AGENTS NOT TRANSMITTED IN EGGS

The commodity definition requires that eggs used for the manufacture of egg powders to be imported into New Zealand must be free of dirt or other foreign matter, or cleaned to a standard equivalent to that described in EEC directive 89/437/EEC Chapter V, and that contamination of egg pulp by eggshell remains, egg membranes and other particles is less than 100mg/kg. Therefore, organisms which have been shown to be transmitted through surface contamination of eggs but for which there is no evidence of transmission in the egg contents, are not considered to be potential hazards in this risk analysis. Organisms for which there is no evidence of transmission in the contents of hens' eggs are not considered to be potential hazards in the commodities.

- There has been no demonstrated transmission of *Borrelia anserine* in eggs so avian spirochaetosis requires no further consideration (Barnes 2003).

- Derszy's disease has never been identified in chickens. Muscovy duck disease is associated with a parvovirus that is similar to, but antigenically distinct from, Derszy's disease virus. Muscovy duck disease has never been identified in chickens (Gough 2003a). Derszy's disease and Muscovy duck disease are therefore not considered to be potential hazards.

- Duck virus hepatitis types 1 and 3 are associated with picornaviruses. Limited information is available regarding DHV3. Under natural circumstances, DHV1 appears to only infect young ducklings whilst field observations have suggested that chickens and turkeys are resistant. However, there are a number of reports indicating successful experimental infection of poultry with DHV1. Transmission of DHV1 via faecal contamination of eggs is likely, whereas there is no evidence to suggest transmission of DHV1 in eggs (Woolcock 2003). As indicated above, organisms which have been shown to be transmitted through surface contamination of eggs, but for which there is no evidence of transmission in the egg contents, are not considered to be potential hazards in this risk analysis.

- Duck hepatitis type 2, causal agent DHV2 (an astrovirus), occurs only in ducks (Woolcock 2003). Although limited information is available relating to this pathogen, it does not appear to infect chickens and is, therefore, not considered to be a potential hazard.

- Under natural circumstances, duck septicaemia (*Riemerella anatipestifer*) does not occur in chickens although, under experimental conditions, it was observed that the injection of $8x10^6$ organisms into the foot pad could result in the death of week-old chicks. Transmission of this agent occurs through bodily secretions with infection occurring through inhalation or via skin wounds. As there are no reports of transmission in faeces or in eggs (Sandhu 2003), *R. anatipestifer* is not considered to be a potential hazard in this risk analysis.

- Under experimental conditions, infection of domestic chickens with duck viral enteritis (DVE) has been demonstrated using virus adapted by serial passage in embryonated eggs (Jansen 1968). Naturally-occurring infection has never been recorded. DVE is shed in the oral-respiratory secretions and faeces with transmission occurring through aerosols and ingestion. Conflicting reports exist regarding the transmission of DVE in eggs. As natural infection of chickens with DVE is not recognised, no further consideration is required.

- Transmission of *Pasteurella multocida* (fowl cholera) usually occurs through secretions from the mouth, nose, and conjunctiva of diseased birds. Faeces very seldom contain viable *P. multocida*. There is no evidence of transmission within eggs (Glisson et al 2003) so *P. multocida* is not considered to be a potential hazard.

- Unlike group 1 adenoviruses, there is no evidence that group 2 adenoviruses can be transmitted in eggs (Pierson and Fitzgerald 2003) so these need not be considered further.

- There is no evidence that infectious bursal disease virus (IBDV) is carried within the egg although it is likely that virus may be present on the surface of the shell as transmission of the disease is via the faecal-oral route (Lukert and Saif 2003). IBDV is unlikely to be present in egg pulp and, as indicated above, is therefore not considered to be a potential hazard. In the review of submissions for the Belovo egg powder risk analysis (MAF 2003), MAF further explored the likelihood of IBD contamination of egg pulp and, for reference, the relevant section of that review is included here in Appendix 1.

- Transmission of *Haemophilus paragallinarum* does not occur in eggs and is most likely to occur through respiratory secretions (Blackall and Matsumoto 2003). The agent of infectious coryza is not, therefore, a potential hazard.

- Marek's disease occurs in New Zealand although the highly pathogenic strains of the virus found overseas are thought not to be present. However, transmission of Marek's disease virus within the egg does not occur (Witter and Schat 2003).

- *Mycoplasma meleagridis* infection is restricted to turkeys and has not been recorded in chickens (Chin et al 2003) so will not be considered as a potential hazard here.

- Quail bronchitis is associated with a serotype I avian adenovirus (QBV). Clinical disease is restricted to quail although chickens and turkeys can be infected experimentally resulting in few or mild clinical signs (Yates and Fry 1957). Naturally-occurring infection of chickens and transmission in eggs has not been documented (Jack et al 1987) so this organism is not considered to be a potential hazard.

- As well as turkeys, infection of chickens, pheasants, and guinea fowl with turkey rhinotracheitis virus has been demonstrated (Gough et al 1988). There is no evidence that this virus can be transmitted in eggs (Gough 2003b) so it is not considered to be a potential hazard in this risk analysis.

- Turkey viral hepatitis (TVH) is most likely associated with a picornavirus infection. Disease has only been recorded in turkeys, and studies have shown that chickens are refractory to infection (Tzianabos and Snoeyenbos 1965). TVH is not, therefore, considered to be a potential hazard here.

From the preliminary hazard list, strains of the following disease/agents are considered to be both exotic to New Zealand and possibly transmitted through the contents of hen's eggs and require further consideration:

>Angara disease (Adenoviridae)
>Arizonosis (*Salmonella* Arizonae)
>Avian influenza (Orthomyxoviridae)
>Avian leukosis/sarcoma (Retroviridae)
>Avian paramyxovirus types 2 & 3 (Paramyxoviridae)
>Campylobacteriosis (*Campylobacter jejuni* and others)
>Colibacillosis (*Escherichia coli* 0111, 0157:H7 and others)
>Fowl typhoid (*Salmonella* Gallinarum)
>Group 1 adenovirus infections (Adenoviridae)
>Infectious bronchitis (Coronaviridae)
>Mycoplasmosis (*Mycoplasma iowae*)
>Newcastle disease (Paramyxoviridae)
>Ornithobacteriosis (*Ornithobacterium rhinotracheale*)
>Paratyphoid salmonellae (*Salmonella* Enteritidis etc.)
>Psittacosis (*Chlamydophila psittaci*)
>Pullorum disease (*Salmonella* Pullorum)

6.3 DISEASE AGENTS INACTIVATED BY PROCESSING CONDITIONS

Egg powders considered in this risk analysis are subject to heat treatment during manufacture at a temperature greater than 60°C for a period of no less than 3.5 minutes (egg powders and egg yolk powders), or subject to a final heat treatment at greater than 54.4°C for a period of no less than 7 days (egg albumen powders). Powders are also subject to additional heating through the spray-drying process used during manufacture which typically uses an inlet temperature of 155°C to 175°C and outlet temperature of 70°C to 80°C.

The following diseases/agents are likely to be inactivated by the processing conditions defined for this commodity and are therefore not considered to be potential hazards in this risk analysis:

6.3.1 *Salmonella* spp.

Studies on liquid whole egg artificially inoculated with sixteen different Salmonella serotypes and subjected to a range of pasteurisation temperatures demonstrated that none of these organisms were able to survive exposure to 60°C beyond a period of 2.9 minutes (Mitscherlich and Marth 1984a). Gast has commented that, with the exception of a few distinctly thermoresistant strains, salmonellae are generally quite susceptible to destruction by heat (Gast 2003). A review of studies on the thermal resistance of Salmonellae demonstrated D-values[7] for Salmonella isolates in liquid whole egg of 0.2 to 1.0 minutes at 60°C and D-values of 0.28 to 1.1 minutes at 60°C in liquid egg yolks (Doyle and Mazzotta 2000). *Salmonella* Senftenberg isolates were also described with D-values of 5.6 minutes at 60°C in liquid whole egg, and 11.8 minutes at 60°C in liquid egg yolks although this serotype has been identified in cattle, sheep, pigs, and poultry in New Zealand (Clark et al 2002). Given the pasteurisation temperatures defined here, and the further heating associated with the spray-drying process, exotic *Salmonella* spp. are not considered to be a potential hazard in this commodity.

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[7] D-values (decimal reduction times) refer to the amount of time taken at a certain temperature to kill 90 percent of the organisms being studied.

6.3.2   Paramyxoviruses

Newcastle disease virus (NDV) and other avian paramyxoviruses are sensitive to heat and the rate of destruction depends on a number of factors. Studies into the thermal stability of avian paramyxoviruses have shown that all virus activity is destroyed within 1 minute at 100°C. Within 5 minutes at 56°C, infectivity, haemagglutinative activity, and immunogenicity are destroyed (Foster and Thompson 1957). More recent studies have shown that heat application to egg products artificially infected with NDV can result in virus inactivation when using temperatures and times of heat application similar to those used commercially, with D-values of <18 to <20 seconds in homogenized whole egg at 61°C and D-values of <0.25 to 0.3 days in dried egg whites at 55°C reported (Swayne and Beck 2004). NDV and other avian paramyxoviruses are, therefore, not considered to be potential hazards in this risk analysis.

6.3.3   Retroviruses

Retroviruses (such as those associated with avian leucosis/sarcoma) are relatively sensitive to inactivation by heat with the half life of these viruses at 37°C varying from 100 minutes to 540 minutes (average around 260 minutes) (Vogt 1965). Avian retroviruses have been shown to be rapidly inactivated at high temperatures, and the Rous sarcoma virus has a half-life of 8.5 minutes at 50°C and 0.7 minutes at 60°C (Dougherty 1961). Exotic strains (subgroup J) of avian leucosis/sarcoma virus may be present in egg pulp although these will be inactivated by processing conditions and are, therefore, not considered to be potential hazards here.

6.3.4   *Escherichia coli*

E.coli possesses no unique resistance capabilities and has a susceptibility pattern to chemical and physical agents typical of vegetative, gram-negative bacteria (Barnes et al 2003). *E.coli* has been shown to be inactivated by pasteurisation, and proper cooking readily destroys *E. coli*. At 64.5°C no viable bacteria were isolated from milk (D'Aoust et al 1988). The processing conditions defined for these commodities will therefore be sufficient to inactivate any exotic strains of *E.coli* present in egg pulp.

6.3.5   *Campylobacter* spp.

Campylobacters are extremely sensitive to desiccation and studies have shown that a suspension of *C. jejuni* impregnated onto filter paper will not survive beyond 2 hours at 20°C (Luechtefeld et al 1981). There is no evidence to suggest that significant transmission of Campylobacters occurs by either transovarian infection or by penetration of the egg shell after oviposition. Artificial contamination of eggs with a faecal suspension of *C.jejuni* showed that the organism was no longer viable after 16 hours and that 50 percent of infected eggs were free of viable campylobacters within 10 hours (Shane et al 1986). *Campylobacter* spp. are not considered to be potential hazards in this risk analysis.

6.3.6   Infectious bronchitis

Serological studies show that infectious bronchitis is widespread in New Zealand although isolates from this country appear to be unrelated to field isolates or vaccine strains from Europe or North America (Lohr and Locher 1983). Studies have shown that most strains of infectious bronchitis virus are inactivated at temperatures of 56°C after 15 minutes and at 45°C after 90 minutes (Cavanagh and Naqi 2003). Exotic isolates of infectious bronchitis may therefore be present in egg pulp although these will be destroyed by the processing conditions described and are, therefore, not considered to be potential hazards here.

6.3.7   *Mycoplasma* spp.

Although the natural host of *Mycoplasma iowae* is the turkey, it is not uncommon for *M. iowae* to be isolated from chickens (Bencina et al 1987). Transmission of *M. iowae* in turkey eggs has been demonstrated (McClenaghan et al 1981) and faecal transmission of this agent is also likely (Mirsalimi et al 1989). However, as this agent is predominantly a pathogen of

turkeys, little information regarding its transmission in chicken eggs is available. Mycoplasmas are fragile organisms (Quinn et al 1994). Sealed cultures of *Mycoplasma agalacticae* subsp. *agalacticae* have been shown to survive less than 8 minutes at 53°C (Mitscherlich & Marth 1984b) and cultures of *Mycoplasma gallisepticum* innoculated into eggs were found to be inactivated after 10 minutes at 54°C (egg white) or after 10 minutes at 56°C (egg yolk) (Mitscherlich & Marth 1984c). Given the processing temperatures defined here, and the further heating associated with the spray-drying process, *Mycoplasma iowae* is not considered to be a potential hazard in this commodity.

6.3.8   *Ornithobacterium rhinotracheale*

Ornithobacterium rhinotracheale will grow at temperatures up to 42°C, and it is likely that, as with most other vegetative forms of bacteria, it is quickly inactivated at temperatures much higher than that. A closely related bacterium, *Haemophilus paragallinarum,* is destroyed at 50°C in 2 minutes (Mitscherlich and Marth 1984d). Therefore, *Ornithobacterium rhinotracheale* is not considered to be a potential hazard in this commodity.

6.3.9   *Chlamydophila psittaci*

Exposure of infectious tissue homogenates of *Chlamydophila psittaci* to 56°C for 5 minutes is sufficient for inactivation of the organism (Page 1959). Any exotic strains present in egg pulp will be inactivated by the processing conditions and are not considered to be potential hazards in this risk analysis.

6.4   PRELIMINARY HAZARD LIST CONCLUSION

As described above, any diseases/organisms from the preliminary hazard list which are thought to be exotic to New Zealand, and are known to be transmitted in the eggs of *Gallus gallus,* and are unlikely to be inactivated by the processing conditions defined for this commodity are considered to be potential hazards requiring further risk assessment.

Although adenoviruses are generally regarded as being inactivated by exposure to temperatures of 56°C for 30 minutes, avian adenoviruses show more variability and appear to be more heat resistant with some strains surviving exposure to 60°C or even 70°C for 30 minutes (McFerran and Adair 2003). The processing conditions defined for this commodity cannot, therefore, be relied upon to inactivate heat resistant avian adenoviruses.

Swayne and Beck (2004) demonstrated that a low pathogenicity avian influenza virus strain (LPAI/NY/H7N2) had a D-value of <20 seconds in homogenized whole egg at 61°C, and a D-value of 0.5 days in dried egg white at 55°C. However, this study also demonstrated that inactivation of a high pathogenicity AI strain (HPAI/PA/83) in dried egg white would require exposure to 54.4°C for 21.38 days. Final heat treatment of egg albumen powders at 54.4°C for a period of 7 days cannot, therefore, be relied upon for inactivation of avian influenza viruses.

From the preliminary hazard list (Table 3), exotic group 1 avian adenoviruses and avian influenza viruses are considered to be potential hazards requiring further risk assessment. No other diseases/organisms have been identified from the preliminary hazard list which are considered to be exotic to New Zealand, and are known to be transmitted in the eggs of *Gallus gallus,* and will not be inactivated by the processing conditions defined for this commodity.

7 Group 1 Avian Adenoviruses

7.1 HAZARD IDENTIFICATION

7.1.1 Aetiological agent

The role of most group 1 avian adenoviruses as pathogens is not well defined with the exception of those associated with quail bronchitis and Angara disease (McFerran and Adair 2003).

As described in 6.2 above, for quail bronchitis, naturally-occurring infection of chickens has not been documented (Jack et al 1987) so the aetiological agent of this disease requires no further consideration here.

Angara disease (also known as hydropericardium syndrome) is associated with FAdV-4 viruses. Full classification of this agent is:

Family Adenoviridae; Genus Aviadenovirus (Group 1 avian adenoviruses); Species Fowl adenovirus C; Serotype Fowl adenovirus 4 (FAdV-4)

7.1.2 OIE list

Not listed

7.1.3 New Zealand status

Group 1 avian adenoviruses are considered commonplace in New Zealand, and the majority of them have a limited role, if any, as primary pathogens (Howell 1992).

Angara disease was first described in Pakistan in 1987 and was subsequently recognised in India, Kuwait, Iraq, Japan, and the former U.S.S.R. What is thought to be a less severe form of this disease has also been described in South and Central America and Mexico (McFerran and Adair 2003). Angara disease has not been recognised in New Zealand.

7.1.4 Epidemiology

Angara disease was first recognised at Angara Goth, an extensive broiler producing area near Karachi, Pakistan (Jaffery 1988). Further characterisation of this disease has described the accumulation of straw-coloured jelly-like fluid in the pericardial sac, discolouration and inflammation of the liver with basophilic intranuclear inclusion bodies, renal congestion, and up to 70 percent mortality (Khan et al 2005). The appearance of the heart floating in pericardial fluid has been described as similar to a deshelled leechy (lichee) fruit, hence the use of alternative names "leechy disease" or "litchi disease" in India (Balamurugan and Kataria 2004). Disease usually occurs in 3-5 week old broiler birds and, other than mortality, clinical signs of disease are rare.

Initial studies into the aetiology of this syndrome hypothesized that the disease was associated with a nutritional disorder. However, transmission of disease using subcutaneous inoculation of an infected liver homogenate indicated an infectious aetiology (Khan et al 2005). Further studies have demonstrated a role of FAdV-4 in this disease (McFerran and Adair 2003). Whilst some FAdV-4 viruses are able to cause Angara disease on their own, it appears that other strains require the assistance of immunosuppressive agents such as the chicken infectious anaemia virus (Toro et al 2000).

Vertical transmission is important in the spread of group 1 avian adenoviruses. Virus can also be found in faeces, the tracheal and nasal mucosa, and kidneys so transmission via all excretion routes is considered possible.

Although adenoviruses are generally regarded as being inactivated by exposure to temperatures of 56°C for 30 minutes, avian adenoviruses show more variability and appear to be more heat resistant with some strains surviving exposure to 60°C or even 70°C for 30 minutes (McFerran and Adair 2003). Studies on the agent of Angara disease have indicated that it may persist following processing at 60°C for 30 minutes or 50°C for 1 hour, although it is likely to be inactivated following processing at 60°C for 1 hour, 80°C for 10 minutes, or 100°C for 5 minutes (Afzal et al 1991).

7.1.5 Hazard identification conclusion

Angara disease has not been described in New Zealand, and it is assumed that FAdV-4 is exotic. This agent may be present in egg pulp and, based on the available data, heat resistant strains should be considered a hazard in whole egg or egg yolk powders which have been pasteurised at 60°C for a period of 3.5 minutes or in egg albumen powders which have been subject to a final heat treatment of 54.4°C for a period of 7 days.

7.2 RISK ASSESSMENT

7.2.1 Entry assessment

Although Angara disease was first described in broiler birds, disease has also been described in both layer and breeder flocks (McFerran and Adair 2003). Mortality in infected flocks begins at around three weeks of age, peaks at 4-5 weeks then declines (Khan et al 2005). Adenovirus excretion within a broiler flock will peak between 4 and 6 weeks of age, whilst in layer replacements peak excretion of adenoviruses between 5 and 9 weeks is described. This peak excretion in layer flocks would occur before point of lay (typically 15-17 weeks).

However, a second peak of adenovirus excretion has been recognised around the period of peak egg production in layer flocks and it has been suggested that this second peak is associated with the stress of egg production or increased levels of sex hormones. Adenovirus production at this point ensures maximum viral transmission to the next generation (McFerran and Adair 2003).

FAdV-4, the likely aetiological agent of Angara disease, may therefore be found in egg pulp derived from eggs taken from infected flocks. Heat resistant strains of avian adenoviruses have been described that are likely to survive processing conditions associated with egg powder manufacture.

The likelihood of entry is therefore considered to be non-negligible.

7.2.2 Exposure assessment

For Angara disease to infect birds in New Zealand, susceptible birds would have to be exposed to infected egg powders.

Most imported egg powders are destined for further processing within the catering industry (which will usually involve further cooking) and it is unlikely that waste from these practices would be disposed of in sites where access by susceptible wild avian species may occur. Some egg powders will also be marketed directly to the camping/sailing market. It is possible that wild birds would have access to uneaten product prepared in these environments.

Disease has been described in pigeons (Naeem and Akram 1995) and it has been possible to reproduce disease in broilers using liver suspensions from infected pigeons. Transmission of FAdV-4 to wild bird species therefore appears possible and disease could pass from these to domestic poultry.

Given the use of the commodity, the likelihood of direct exposure to domestic poultry is considered to be negligible. However, the likelihood of exposure to wild avian species is considered to be low but non-negligible.

7.2.3 Consequence assessment

There is a low but non-negligible likelihood that wild birds could be exposed to FAdV-4 and, on farms with poor biosecurity, infection could pass from wild birds to domestic poultry. Angara disease has been associated with huge economic losses to the poultry industry in Pakistan where infection has resulted in mortality rates of up to 70 percent in broiler flocks (Khan et al 2005).

The pathogenicity of FAdV-4 is likely to be potentiated by immunosuppressive infections such as chicken infectious anaemia, which is known to be present in New Zealand.

Eradication of FAdV-4 infection, once established in a flock, is likely to be difficult due to the highly effective transmission of avian adenoviruses through both horizontal and vertical routes.

Group 1 avian adenoviruses have no known public health significance (McFerran and Adair 2003). The consequences to human health are therefore considered to be negligible.

In addition to chickens, avian adenovirus serotypes have been recovered from turkeys, pigeons, budgerigars, and a mallard duck, and probable fowl isolates have come from guinea fowl and pheasants. In addition, suspect adenovirus particles have been observed by electron microscopy using tissues from kestrels, herring gulls, peach-faced lovebirds, a rose-ringed parakeet, budgerigars, rosella, red-rumped parakeets, eclectus parrots, common murre, a cockatiel, and a tawny frog-mouth (McFerran and Adair 2003). Given these findings and the evidence that FAdV-4 is likely to have caused disease in pigeons (Naeem and Akram 1995), the consequences of introduction on native bird species are considered to be non-negligible.

In conclusion, the introduction of FAdV-4 could impact upon native bird species. Where there is poor biosecurity, FAdV-4 may spread from wild species into domestic poultry causing losses to this industry. The consequences of introduction of FAdV-4 into New Zealand are therefore considered to be non-negligible.

7.2.4 Risk estimation

Because the entry, exposure, and consequence assessments are non-negligible, the risk estimate is considered to be non-negligible and FAdV-4 is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

7.3 RISK MANAGEMENT

7.3.1 Options available

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

- Assurance could be required that eggs used have been derived from flocks in countries or geographic regions where Angara disease has not been recognised.

- The source flock could be tested to ensure freedom from FAdV-4. Diagnostic tests for avian adenovirus using PCR are available. A PCR capable of amplifying an avian adenovirus 421-bp DNA product from the 12 serotypes of Group 1 and serotypes from Group 2 and 3 has been developed (Xie et al 1999). A PCR has also been developed specifically for the detection of FAdV-4 (Ganesh, Suryanarayana, and Raghavan 2002). It could therefore be possible to test the source flock to ensure freedom from FAdV-4.

- Studies of liver homogenate extracts have shown that heat treatment of 60°C for greater than one hour destroys FAdV-4 infectivity (Afzal et al 1990 cited by Cowen 1992, Afzal et al 1991). Further heat treatment of manufactured powders could therefore be used to destroy any FAdV-4 present.

8. Avian Influenza Viruses

8.1 HAZARD IDENTIFICATION

8.1.1 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) (Alexander et al 2000). Strains of AI are commonly separated into highly pathogenic strains (HPAI) and low pathogenic strains (LPAI) on the basis of their pathogenicity in chickens.

8.1.2 OIE list

Notifiable avian influenza (NAI) viruses are listed by the OIE. NAI refers to any avian influenza virus of H5 or H7 subtypes or any AI virus with pathogenicity above limits set in Chapter 2.7.12 of the *Terrestrial Animal Health Code* (OIE 2007).

8.1.3 New Zealand status

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

AI viruses have been isolated from healthy wild mallard ducks in New Zealand (Austin and Hinshaw 1984, Stanislawek 1990, Stanislawek 1992). Subtypes identified have included H4N6, H1N3, and H5N2 (Austin and Hinshaw 1984, Stanislawek et al 2002). The H5N2 isolates were shown to be non-pathogenic (Austin and Hinshaw 1984, Stanislawek et al 2002). More recent studies of wild waterfowl have recovered H1, H2, H4, H7, H10 and H11 subtypes of AI in New Zealand (Tana et al 2007).

8.1.4 Epidemiology

AI infections of domestic poultry can produce a range of syndromes, from subclinical disease to severe, systemic disease with near 100 percent mortality (Swayne and Halvorson 2003). A wide range of wild and domestic birds can be infected, especially free-living aquatic birds. AI viruses have been identified from more than 90 species of free-living birds although most infections in free-living birds have not been associated with clinical disease (Swayne and Halvorson 2003).

Horizontal transmission of infection commonly occurs as AI viruses are excreted from the nares, mouth, conjunctiva, and cloaca of infected birds. High levels of virus are also found in the tissues of infected birds and virus may also be spread between birds by direct contact, through aerosol droplets, or via contaminated fomites (Swayne and Halvorson 2003).

There is limited evidence for vertical transmission although 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 (Cappucci et al 1985). Unpublished work by M. Brugh cited by Swayne and Beck (2004) identified AI virus in 85 to 100 percent of eggs laid on days 3 and 4 following experimental inoculation.

As described above, Swayne and Beck (2004) demonstrated that a low pathogenicity AI virus strain (LPAI/NY/H7N2) had a D-value of <20 seconds in homogenized whole egg at 61°C and a D-value of 0.5 days in dried egg white at 55°C. This study also demonstrated that a high pathogenicity strain (HPAI/PA/83) had a D-value of <19 seconds in homogenized whole

egg at 61°C but inactivation of this strain in dried egg white would require exposure to 54.4°C for 21.38 days.

8.1.5   Hazard identification conclusion

Given the processing conditions defined for this commodity, avian influenza viruses are not considered to be a potential hazard in egg powders which have been pasteurised at 60°C for a period of 3.5 minutes.

The study by Swayne and Beck (2004) demonstrates that avian influenza viruses in albumen powders may not be inactivated following treatment at 54.4°C for 7 days. Exotic strains of avian influenza should therefore be regarded as potential hazards in egg powders which have been subject to a final heat treatment of 54.4°C for a period of 7 days (egg albumen powders).

8.2   RISK ASSESSMENT

8.2.1   Entry assessment

The incubation period in naturally infected birds is quoted as being from 3 to 14 days (Swayne and Halvorson 2003). AI virus has been detected in 85 to 100 percent of eggs laid on days 3 and 4 post experimental inoculation although no virus was detected on days 1 and 2 post inoculation (unpublished work by M. Brugh cited by Swayne and Beck 2004). Therefore, virus is likely to be present in the eggs taken from an infected poultry flock before clinical signs of infection are seen.

As discussed above, studies have shown that strains of AI may persist in the face of a final heat treatment of 54.4°C for a period of 7 days.

The likelihood of entry is therefore considered to be non-negligible.

8.2.2   Exposure assessment

For AI to infect birds in New Zealand, susceptible birds would have to be exposed to infected egg powders.

Most imported egg powders are destined for further processing within the catering industry (which will usually involve further cooking) and it is unlikely that uncooked waste from these practices may be disposed of in sites where access by susceptible wild avian species may occur. Some egg powders will also be marketed directly to the camping/sailing market. It is possible, but highly unlikely, that wild birds will have access to uncooked product prepared in these environments.

Given the use of the commodity, the likelihood of exposure to domestic poultry is considered to be negligible. However, the likelihood of exposure to wild avian species is considered to be low but non-negligible.

8.2.3   Consequence assessment

AI viruses usually produce no mortality or morbidity in wild birds although, occasionally, dead wild birds have been identified on farms with HPAI outbreaks (Swayne and Halvorson 2003). Alexander et al (2000) commented that, in the vast majority of influenza outbreaks occurring in domestic poultry, spread from free-living birds appears to be the most likely mechanism of primary infection, the evidence being weighty though mainly circumstantial. Therefore, domestic poultry farms in New Zealand with poor biosecurity could become infected from exposed wild birds. Infection of domestic poultry could result in severe disease with high mortality.

The intensive mixing of humans, pigs, and ducks, seen in Asia and considered to contribute to the development of new human 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 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.

In view of the above, the consequence assessment is considered to be non-negligible.

8.2.4 Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate is considered to be non-negligible and exotic strains of avian influenza are classified as a hazard in egg albumen powders. Therefore, risk management measures can be justified.

8.3 RISK MANAGEMENT

8.3.1 Options available

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

- Swayne and Beck (2004) demonstrated that exposure of dried egg white to 54.4°C for 21.38 days or to 67°C for 0.83 days, would be sufficient to inactivate any AI present. Therefore, egg albumen powders that have been subject to a final heat treatment of no less than 54.4^{o}C for a period of at least 21.38 days or to no less than 67°C for a period of at least 0.83 days (consistent with Article 3.6.5.1 of the *Code*) could be imported without sanitary measures.

- Source flocks could be tested to ensure freedom from AI viruses. All influenza A viruses have antigenically similar nucleocapsid and matrix antigens. AGID tests could be used to test the flock of origin for serological evidence of avian influenza viruses (OIE 2004).

- Alexander et al (2000) stated that the accumulating evidence that HPAI viruses arise from LPAI H5 or H7 viruses infecting chickens or turkeys suggests that when viruses of these subtypes spread from free-living birds there is a potential that they may become virulent. Given the AI subtypes that have been identified in New Zealand (Section 8.1.3) it might be reasonable to restrict sanitary measures to the control of notifiable AI (NAI) as defined by the OIE (OIE 2007). In which case, eggs used in imported egg powders could be certified to have originated from flocks in countries, zones, or compartments which are free of NAI as described in article 2.7.12.3 of the *Code* (OIE 2007).

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APPENDIX 1

Extract from the 2003 review of submissions on the Belovo Egg Powders Risk Analysis, which further explores the issue of the likelihood of IBD contamination of egg pulp:

As pointed out in MAF's 1999 chicken meat risk analysis, the starting titre of virus in the particular product is of vital importance when considering the effect of temperature on virus inactivation in chicken products, as virus infectivity declines at a predictable rate at any given temperature. Therefore, when considering the likelihood of viable IBD virus being present in egg powder, it is first necessary to consider how the virus might get into the egg pulp prior to it being processed, and at what level it might be present in egg pulp.

As pointed out in the risk analysis, since IBD virus is transmitted by the faecal-oral route and is not transmitted *in* eggs, the only way that the virus could find its way into egg pulp would be by faecal contamination of eggs and by such contamination being transferred to the egg pulp during the process of egg breaking.

In order to assess the likelihood and degree of this happening, it is first necessary to be quite clear about the pathogenesis of IBD infection. When IBD virus is introduced into the gastrointestinal tract of chickens, it is picked up by gut macrophages and lymphoid cells as soon as 4 hours post infection, and these cells carry the virus to the bursa of Fabricius, via the portal blood. Within a few days of infection, high titres of IBD are found in various organs. Titres as high as 10^6 to 10^8 EID_{50} per g of tissue are found in bursa, spleen, thymus, liver of chickens that have died of IBD, and this is the result of the pronounced secondary viraemia that follows the massive replication of the virus in specific bursal lymphoid cells. However, since there is little replication in peripheral lymphocytes (mature cells), without that massive replication in the bursa, infection remains confined to small numbers of gut-associated cells, in which case there is negligible viraemia. In birds where there is less bursal involvement, the titre of virus in tissues is correspondingly less, and where there is no bursal involvement (e.g. in birds that do not have any residual bursal tissue), the titre of virus in tissues is considerably lower than the levels mentioned above. Thus, the level of virus in faeces depends on the degree of bursal involvement.

Thus, as pointed out in response to the DOC submission, MAF considers that the key epidemiological points relevant to the likelihood of IBD virus being present on eggs are as follows:
- Incubation period is 2-3 days.
- For infection to take place, birds must have a functional bursa of Fabricius – this means that infection is not commonly seen in birds under 3 weeks of age.
- Maximum susceptibility is between 3 and 6 weeks of age, corresponding to the period of maximum bursal development, and in endemic situations, infection outside this age range would be unlikely;
- Involution of the bursa begins at about 12 weeks and is usually complete by 20 weeks.
- In unvaccinated flocks (which would be rare in endemic areas) it would be normal for all susceptible birds to very quickly become infected after introduction, as the virus is extremely contagious.
- In non-endemic situations, infection is possible if the virus were introduced into a flock up to about 16 weeks of age.

- Laying normally begins at around 20-24 weeks of age for meat breeders, and from 15-19 weeks for commercial layers.
- The virus is present in the faeces of infected birds for up to 2 weeks post-infection.

In light of the above facts, MAF considers that the likelihood of IBD virus being present in the faeces of layers in an endemic IBD situation is low. Even if the virus were introduced into an unvaccinated flock shortly before point of lay, the likelihood of infection occurring in any birds is low, as most birds would have been exposed in early life. For the same reasons, the likelihood of spread would also be low.

Nevertheless, although it might be somewhat unlikely, it is possible to imagine a scenario whereby IBD virus could be present in the faeces of laying hens. If the virus were introduced into a non-vaccinated flock, shortly before point of lay (or if a sufficiently different strain was introduced into a vaccinated flock such that it was not covered by the existing vaccination schedule), infection could occur in the few birds that had sufficient bursal tissue remaining. Following infection in these birds, the virus could be excreted in faeces for up to two weeks, so that in such flocks some birds could be laying eggs contaminated with faeces for perhaps the first 2 weeks of the laying period . This appears to have been the situation alluded to in the paper cited in the AFFA TIP, which was cited in the PIANZ submission as evidence that infection of layers is possible.[8]

However, there several reasons to suspect that the above scenario is of minor significance as far as egg pulp is concerned. Even if some layers in a flock were infected with IBD virus, considering the amount of virus likely to be in faeces of chickens that are laying eggs (considerably less than in tissues of acutely infected birds) and the amount of faeces likely to be present on eggs (even dirty eggs carry only milligrams of faeces), the amount of virus on the surface of any single egg laid by infected birds is likely to be low.

Further, since the EU directive requires dirty eggs to be washed (and disinfected, although this is targeted at bacteria likely to cause food poisoning, not IBD), the amount of virus present on the shells of eggs at the time of breaking is likely to be very low. And notwithstanding that some egg shell inevitably finds its was into the egg pulp during egg breaking, the provisions of Council Directive 89/437/EEC [Chapter V of the Annex] aim to minimise such contamination, and sampling has to be carried out to show that egg shell and membrane contamination does not exceed 100 mg/kg of the egg pulp [point 2(c) of Chapter VI of the Annex]. Therefore, only a small proportion of any virus on the shell would find its way into egg pulp.

Also, it must be remembered that since IBD is a virus, it means that, unlike the situation with bacterial contamination, it would not multiply in the egg pulp prior to further processing.

Finally, it is worth noting that there are important differences between the production processes for egg powders and those for chicken meat. While in egg powder production there is uniform mixing of the product, the focus for chicken meat remains on an individual carcass and the likelihood of it having been viraemic at slaughter. Thus, there are substantial dilution factors present in this situation (many thousands of eggs make up a single batch of egg pulp for processing into egg powder) that do not occur with chicken meat.

In view of the above factors, it is considered that the amount of IBD virus (titre) in egg pulp would be negligible.

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[8] van den Berg TP, Gonze M, Meulemans G (1991). Acute infectious bursal disease in poultry: isolation and characterisation of a highly virulent strain. Avian Pathology 20: 133-43