Import Risk Analysis: Pigeons (Columba livia) from Australia

FINAL

ISBN 978-0-478-35156-9 (Print)
ISBN 978-0-478-35157-6 (Online)

13 August 2009

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Policy and Risk
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Approved for general release

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Executive Summary

This analysis examines the risks posed by infectious or parasitic agents when importing pigeons from Australia.

Eighty three organisms/diseases of concern of pigeons are considered (Table 1). Of these 17 are classed as preliminary hazards and are subject to a risk assessment. As a result of this, a non-negligible risk is identified with the following hazards:

- Avian paramyxovirus 1 (low pathogenicity)
- Avian influenza virus (low pathogenicity)
- Birnavirus
- Exotic *Salmonella* spp.
- *Coxiella burnetti* (Q fever)
- Protozoal blood parasites (haematozoa)
- Internal parasites
- External parasites

Options are presented for the effective management of risk, including isolation in quarantine for suitable periods, testing for disease agent or antibodies to the agents, and treatment for internal and external parasites.

1. Introduction

Domestic pigeons (*Columba livia)* are abundant and widespread in New Zealand and Australia. The first imports into New Zealand are thought to have been in the 1850s, and imports of small numbers of homing and fancy pigeons from Europe and Australia continued, for the most part without any quarantine measures, until 1996 when concerns over the importation of larger numbers of meat pigeons in recent years resulted in MAF withdrawing the import health standard (IHS). As there is currently no IHS for the importation of pigeons into New Zealand, homing pigeon owners have no way to legally import new blood-lines to improve the performance of their birds.

This risk analysis examines the biosecurity risks to animals, humans, and the environment, associated with importing domestic pigeons from Australia.

2. Scope

This analysis is limited to the description of the risks due to disease-causing organisms associated with the importation of live domestic pigeons (*Columba livia*) from Australia.

The risk analysis does not consider speculative events that could occur in the future, such as the possible establishment of disease vectors due to climate change. The Ministry of Agriculture and Forestry Biosecurity New Zealand (MAFBNZ) has the flexibility to modify any IHS based on this risk analysis if future events make this appropriate.

The risk analysis is qualitative.

3. Commodity Definition

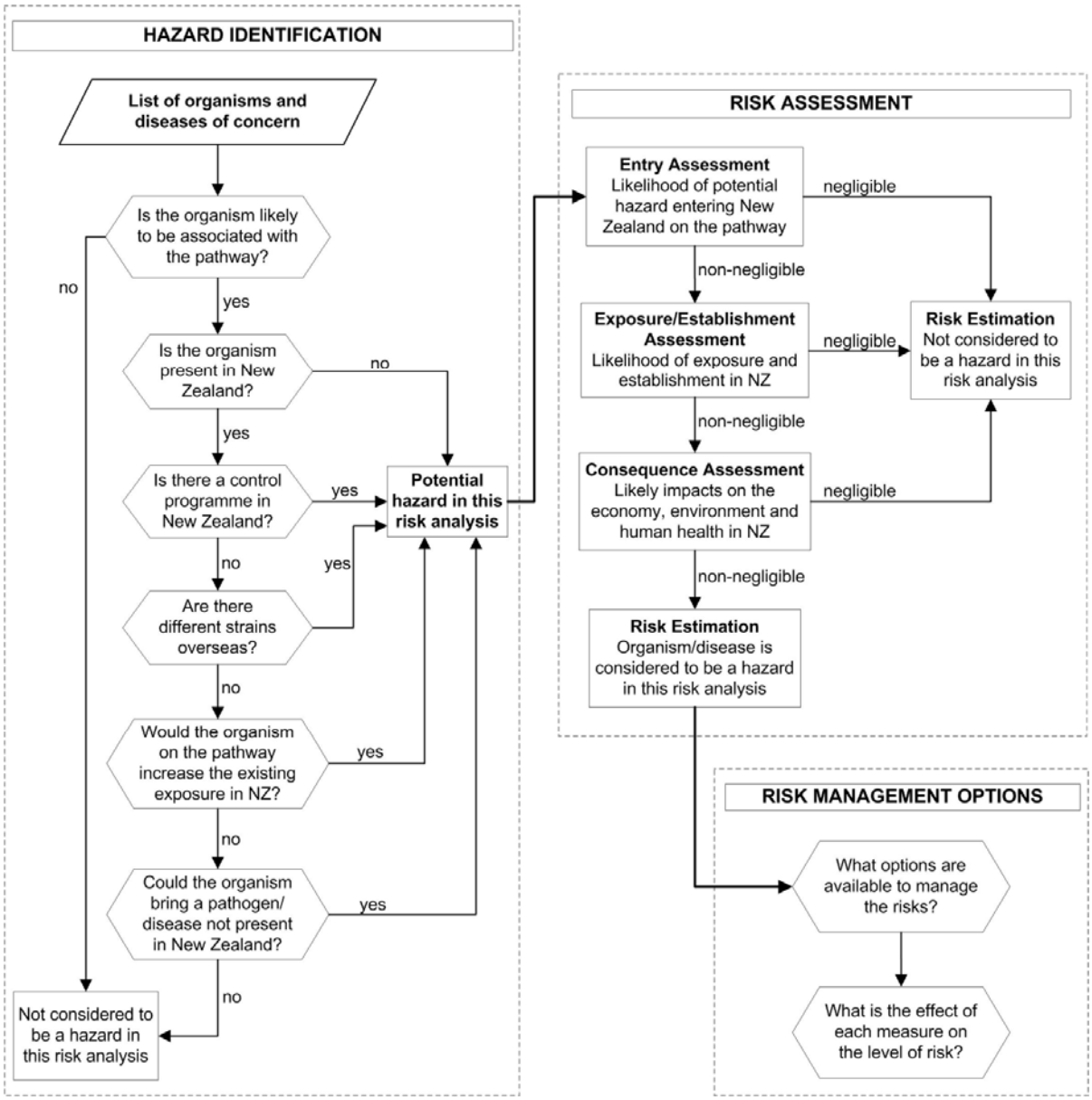
The commodity considered in this risk analysis is domestic pigeons (*Columba livia*) from Australia.

4. Risk Analysis Methodology

The methodology used in this risk analysis is described in MAF Biosecurity New Zealand's *Risk Analysis Procedures – Version 1* (Biosecurity New Zealand 2006) and is consistent with the guidelines in the OIE *Terrestrial Animal Health Code* ("the *Code*") and the OIE Handbook on Import Risk Analysis (OIE 2004).

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

Figure 1. The risk analysis process.



4.1. PRELIMINARY HAZARD LIST

The hazard identification process begins with the collation of a list of organisms likely to be associated with the commodity. Table 1 shows these organisms, together with some of the key information considered. This list was compiled from the following sources:

Simpson BS (2006). *Import Risk Analysis: Passerine Hatching Eggs from the European Union.* Ministry of Agriculture and Forestry, Wellington.

Ritchie BR, Harrison GJ, and Harrison LR (1994). *Avian Medicine: Principles and Application*. Wingers Publishing, Inc, Lake Worth, Florida.

Polyomavirus infection was added to the list as it was considered in the Chicken Meat Import Risk Analysis (MAF 1999) at the request of the Department of Conservation (DOC).

Table 1. Preliminary hazard list

Pathogen	Occurs in NZ	Occurs in Australia	Recorded in pigeons	Strain variation NZ vs Aus	Comments	Requires further consideration
Orthomyxoviruses						
Highly pathogenic avian influenza virus (HPAI) H5/H7 strains	No (OIE 2007)	No (OIE 2007; Peroulis O'Riley 2004)	Yes		Notifiable	No
Low pathogenic avian influenza viruses	Yes (Stanislawek et al 2002)	Yes	Yes	Yes	Some strains in NZ	Yes
Paramyxoviruses						
Virulent Newcastle disease virus, avian paramyxovirus 1 (APMV-1)	No (OIE 2007)	No (OIE 2007; Peroulis and O'Riley 2004)	Yes		Notifiable	No
Low virulence Newcastle disease virus (APMV-1)	Yes (Stanislawek et al 2001)	Yes	Yes	Yes	Some strains in NZ	Yes
Other avian paramyxoviruses 2-9) (APMV 2-9)	Some (Stanislawek et al 2001)	Some	No			Yes
Pneumovirus (turkey rhinotracheitis, swollen head)	No (MAF 2007)	No (Buckley 2007)	No*			No
Herpesviruses						
Duck virus enteritis virus	No (OIE 2007)	No(OIE 2007)	No*		Notifiable	No
Infectious laryngotracheitis virus	Yes****	Yes (OIE 2007)	No (Cover 1996)			No
Marek's disease virus	Yes****	Yes (OIE 2007)	No*			No
Pigeon herpesvirus	Yes (Thompson et al 1977)	Yes	Yes	No		No
Pigeon herpes encephalitis virus	No@	No@	Yes (Ritchie 1995)			No
Pacheco's disease virus	No (Loth 2003; Thornton & Stanislawek 2003)	Yes	No (Vindevogel & Pastoret 1993)			No
Coronaviruses						
Coronavirus enteritis virus	Yes	Yes	Yes (Cavannah 2005)			No
Infectious bronchitis virus and group 3 coronaviruses	Yes****	Yes	Yes		Various strains	Yes
Adenoviruses						
Group I adenoviruses	Yes	Yes	Yes	No		No

Pathogen	Occurs in NZ	Occurs in Australia	Recorded in pigeons	Strain variation NZ vs Aus	Comments	Requires further consideration
	(Christensen & Saifuddin 1989)					
Group II adenoviruses	No*	Yes	No (Domeruth et al 1977)			No
Group III egg drop syndrome	Yes(Howell 1992)	Yes	No(McFerran & Adair 2003)			No
Pigeon Pox virus	Yes**	Yes	Yes**	No		No
Circoviruses						
Chicken infectious anaemia virus	Yes****	Yes	No (Schat 2003)			No
Pigeon circovirus	Yes (Christensen 2007)	Yes (Woods et al 1993)	Yes			No
Psittacine beak and feather disease virus	Yes (Black & Orr 1997; Fraser et al 1999)	Yes	No*			No
Birnaviruses						
Birnavirus (Infectious bursal disease)	No (Bingham et al 2006)	Yes (OIE 2007)	Yes (Kasanga et al 2008)		Notifiable	Yes
Papovaviruses						
Polyoma virus	Yes (Jakob-Hoff 2003)	Yes	No*			No
Papilloma virus	No@	Unknown	Unknown			Yes
Parvoviruses						
Derzsy's disease of geese	No (MAF 2007)	No (Biosecurity Australia 2000)	No			No
Australian Arboviruses	No	Yes	??			Yes
Flaviviruses						
West Nile virus	No	No (Australian Biosecurity 2003)	Yes			No
Louping ill virus	No (MAF 2007)	No (Animal Health Australia. 2005b)	No*			No
Reoviruses						
Rotavirus	?	?	Yes (McNulty 2003)	No		Yes
Orbivirus	No*	Yes	No*			No
Other reoviruses	Yes (Saifuddin et	Yes	Yes*	No (McFerran		No

Pathogen	Occurs in NZ	Occurs in Australia	Recorded in pigeons	Strain variation NZ vs Aus	Comments	Requires further consideration
	al 1989)			et al 1976)		
Bunyaviruses						
Nairovirus (Crimean-Congo haemorrhagic fever)	No (MAF 2007)	No (Swanepoel & Burt 2004)	No*			No
Borna virus group						
Borna disease virus	No (MAF 2007)	No (Animal Health Australia 2007)	No*			No
Picornavirus group						
Avian encephalomyelitis virus	Yes****	Yes (OIE 2007)	Yes	No		No
Avian nephritis virus	Yes (Biosecurity Australia 2000)	Yes (Biosecurity Australia 2000)	No (Imada & Kawamura 2003)			No
Duck hepatitis 1&3 (DVH 1 & 3) virus	No (MAF 2007)	No (Animal Health Australia 2005a)	No		See also astrovirus	No
Astrovirus group						
Astrovirus (turkey astrovirus)	No (MAF 2007)	No (Buckley 2007)	No			No
Astrovirus (duck hepatitis complex DVH2)	No (MAF 2007)	No (Buckley 2007)	No		See also picornavirus	No
Hepatitis B virus						
Hepadnavirus (duck virus hepatitis)	No	Yes (Trivatni et al 2001)	No (Fernholz et al 1993)			No
Retrovirus Group						
Avian leucosis virus	Yes (Stanislawek 2001)	Yes (OIE 2007)	No*			No
Lymphoproliferative disease virus (LPDV)	No	No	No (Payne 2002)		Disease of turkeys	No
Reticuloendotheliosis virus	Yes (Howell 1992)	Yes	Yes	No		No
Unknown aetiology						
Macaw wasting disease/proventriculitis	No (MAF 2007)	Yes	No (Gregory 1995)			No
Big liver/spleen disease and/or hepatitis splenomegaly syndrome	No (MAF 2007)	Yes	No (Payne 2003)		BLS not in Australia in recent years	No

Pathogen	Occurs in NZ	Occurs in Australia	Recorded in pigeons	Strain variation NZ vs Aus	Comments	Requires further consideration
Transmissible spongiform encephalopathy	No (MAF 2007)	No (Animal Health Australia 2004)	No*			No
Young bird sickness	No	No@	Yes (Scullion & Scullion 2007)			No
Bacteria						
Chlamydophila spp. (ornithosis)	Yes (Motha et al 1995)	Yes	Yes			No
Salmonella Gallinarum	No (MAF 2007)	No (OIE 2007)	Yes			No
Salmonella Pullorum	No (MAF 2007)	Yes (OIE 2007)				Yes
Salmonella Arizonae	No (MAF 2007)	No (Biosecurity Australia 2006)	No*			No
Exotic Salmonellae (numerous types and subtypes)	No	Yes	Yes	Yes (numerous)		Yes
Salmonella Enteritidis	Yes	Yes	Yes	Yes		Yes
Escherichia coli						
Avian pathogenic *E coli*	Yes ***	Yes	Yes	No		No
Vero toxigenic *E Coli*						
Campylobacter spp. (e.g. *C. jejuni*)	Yes ****	Yes	Yes	No		No
Other enteric bacteria *(Enterobacteriaceae)*	Yes ****	Yes	Yes		Universal	No
Pasteurella multocida	Yes (OIE 2007)	Yes (OIE 2007)	Yes	No		No
Riemerella anatipestifer	Yes (Anonymous 1974; Orr 1990)	Yes	Yes			No
Riemerella columbina	No	No@	Yes(Vancanneyt et al 1999)			No
Pasteurella gallinarum	Yes	Yes	No*			No+
Ornithobacterium rhinotracheale	No (MAF 2007)	No (Biosecurity Australia 2000)	Yes	No		No
Bordetella avium	No(MAF 2007)	Yes (Blackall & Farrah 1985)	No*			No
Haemophilus paragallinarum	No (MAF 2007)@	Yes (Blackall & Matsumoto 2003)	No (Blackall & Matsumoto 2003)			No
Mycoplasma gallisepticum	Yes****	Yes	Yes	No		No
Mycoplasma iowae	No (MAF 2007)	No	No(Bradbury & Kleven 2003)			No
Other *Mycoplasma*	Some	Yes	Yes	Yes		Yes

Pathogen	Occurs in NZ	Occurs in Australia	Recorded in pigeons	Strain variation NZ vs Aus	Comments	Requires further consideration
spp.						
Mycobacterium tuberculosis	Yes (OIE 2007)	Yes	No*			No
Mycobacterium avium/intracellulare	Yes (OIE 2007)	Yes	Yes	No		No
Francisella tularensis	No (MAF 2007)	No (OIE 2007)	No*			No
Megabacterium spp.	Yes (Johnstone & Cork 1993)	Yes	No*			No
Streptococci/ Staphylococci	Yes	Yes	Yes		Occur universally	No
Yersinia spp. *Y. pseudotuberculosis* *Y. enterocolitica*	Yes(Cork et al 1995)	Yes				No
*Borrelia anserina (*avian spirocaetosis)	No (MAF 2007)	Yes (Commonwealth of Australia 2001)	No information			Yes
Borrelia burgdorferi (Lyme disease)	No (MAF 2007)	No (Doggett et al 1997)	No (Fabbi et al 1995)			No
Intestinal spirochetes	Yes (Midwinter 1999)	Yes	Yes	No		No
Coxiella burnetii	No (MAF 2007)	Yes (OIE 2007)	Yes		Notifiable	Yes
Ehrlichia ruminantium (Cowdria ruminantium)	No (MAF 2007)	No (OIE 2007)	No			No
Aegyptianella pullorum	No	No	Yes		Vector not in NZ	No
Protozoa						
Blood parasites *Haemoproteus* spp. *Leucocytozoon* spp. *Plasmodium* spp. *Trypanosoma* spp.	Some	Some	Some	Not known		Yes
Intestinal protozoa *Eimeria* spp. *Trichomonas* spp. *Sarcocystis* spp.	Some	Some	Some	Not known		Yes
Toxoplasma gondi	Yes(Hartley & Marshall 1957)	Yes				No
Fungi and Yeasts						
Examples: *Histoplasma* spp., *Cryptococcus* spp.	Some species (MAF 2007)	Yes		Numerous, some exotic		Yes
Exotic mycoses	No	?	?			Yes
Internal Parasites	Yes (McKenna 1998)	Yes	Yes	Variety of species		Yes
External parasites	Yes (Bishop &	Yes	Yes			Yes

Pathogen	Occurs in NZ	Occurs in Australia	Recorded in pigeons	Strain variation NZ vs Aus	Comments	Requires further consideration

Heath 1998)

* Extensive review of the literature revealed no evidence of occurrence of the disease in pigeons or reports that pigeons act as reservoirs of the agent.

** Pigeon pox virus vaccine is registered for use in New Zealand.

*** *E coli* are universally distributed and both enterotoxigenic and verotoxigenic species have been found in New Zealand.

**** The organism is commonly isolated or antibodies to it are commonly demonstrated by veterinary or poultry industry laboratories. Surveillance data is regularly reported in the MAF *Surveillance* magazine.

@ Review of the literature revealed no evidence of the occurrence of the organism in the country.

The poultry industry has reported no clinical signs of the disease – see data reported in *Surveillance.*

+ A rare disease of no economic significance (Gooderham 1996).

4.2. HAZARD IDENTIFICATION

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

- Whether the imported commodity could act as a vehicle for the introduction of the organism?
- If the organism requires a vector, whether competent vectors might be present in New Zealand?
- Whether the organism is exotic to New Zealand but likely to be present in exporting countries?
- If it is present in New Zealand,
  - 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
  - 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.

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

4.3. RISK ASSESSMENT

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

Risk Assessment

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 release 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 release is non-negligible but the exposure assessment concludes that the likelihood of exposure to susceptible species in the importing country is negligible, or where both release and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

4.4. RISK MANAGEMENT

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

4.5. RISK COMMUNICATION

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

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

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

References

References marked * were seen as abstracts in electronic databases

Animal Health Australia (2004). Australia's case for TSE freedom boosted by new programme. http://www.animalhealthaustralia.com.au/aahc/index.cfm?350835DA-0592-7798-2CCA-7FD174059A94, downloaded 6/11/07.

Animal Health Australia (2005a). Duck virus hepatitis. http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/ Duck virus hepatitis, downloaded 6/11/07.

Animal Health Australia (2005b). Louping ill (encephalitides- tick borne). http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/ Louping ill, downloaded 6/11/07

Animal Health Australia (2007). Borna disease. http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/ Borna disease, downloaded 6/11/07.

Anonymous (1974). Lincoln Animal health laboratory. *Surveillance*, 1(4), 20-4.

Australian Biosecurity CRC (2003). 2.014R West Nile virus susceptibility and transmission studies in Australian avifauna. http://www1.abcrc.org.au/Pages/project.aspx?projectid=81, downloaded 6/11/07.

Bingham P, Christensen N, Stanislawek W (2006). Investigation into bursal disease seropositivity on two commercial free-range layer properties. *Surveillance*, 33(1), 3-6.

Biosecurity Australia (2000). The importation of non-viable eggs and products containing egg. Technical issues paper., Australian Department of Agriculture Fisheries and Forestry.

Biosecurity Australia (2006). Draft Generic Import Risk Analysis. Report for Chicken Meat. http://www.daff.gov.au/__data/assets/pdf_file/0017/10655/2006-18a.pdf, downloaded 5/11/07.

Biosecurity New Zealand (2006). Risk analysis Procedures. Version 1, Ministry of Agriculture and Forestry, Wellington, New Zealand.

Bishop DM, Heath ACG (1998). Checklist of ectoparasites of birds in New Zealand. *Surveillance*, 25(Special Issue), 13-31.

Black A, Orr M (1997). Review of veterinary diagnostic cases -January to March (1997). *Surveillance*, 24(2), 22-4.

Blackall PJ, Farrah JG (1985). The isolation of *Bordetella avium* from poultry. *Australian Veterinary Journal*, 62(11), 370-2.

Blackall PJ, Matsumoto M (2003). Infectious Coryza. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 691-703. Iowa State Press.

Bradbury J, Kleven SH (2003). *Mycoplasma iowae* infection. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of poultry, 11th edition*. Pp. 766-71. Iowa State Press.

Buckley D (2007). Personal commnication. AFFA.

Cavannah D (2005). Coronaviruses in poultry and other birds. *Avian Pathology*, 34(6), 439-48.

Christensen NH, Saifuddin MD (1989). A primary epidemic of inclusion body hepatitis in broilers. *Avian Diseases*, 33, 622-8.

Christensen NH (2007). Personal communication. E-mail to Howard Pharo, 27 October 2007

Commonwealth of Australia (2001). Generic import risk analysis (IRA) for uncooked chicken meat. Issues paper2. http://www.daffa.gov.au/__data/assets/pdf_file/11292/2001-16a.pdf, downloaded 6/11/07.

Cork SC, Marshall RB, Madie P, Feniwick SG (1995). The role of birds and the environment in the epidemiology of Yersiniae in New Zealand. *New Zealand Veterinary Journal*, 42(5), 169-74.

Cover MS (1996). Early history of ILT. *Avian Diseases*, 40, 494-500.

Doggett SL, Russell RC, Lawrence R, Dickeson D (1997). Lyme disease. http://medent.usyd.edu.au/fact/lyme%20disease.htm#history, downloaded 5/11/07.

Domeruth CH, Forrester DJ, Trainer DO, Bigler WJ (1977). Serologic examination of wild birds for hemorrhagic enteritis of turkey and marble spleen disease of pheasants. *Journal of Wildlife Diseases*, 13, 405-8.*

Fabbi M, Sambri V, Marangoni A, Magnino S, SolariBasano F, Cevenini R, Genchi C (1995). *Borrelia* in pigeons: no serological evidence of *Borrelia burgdorferi* infection. *Zentralblatt fur Veterinarmedizin B*, 42(8), 503-7.*

Fernholz D, Wetz H, Will H (1993). Hepatitis B viruses in birds. In: McFerran JB, McNulty JS (eds). *Virus Infections of Birds*. Elsevier Science Publications, Amsterdam.

Fraser C, Julian AF, Hill FI, Thompson J (1999). Quarterly review of diagnostic cases. *Surveillance*, 26(2), 14-6.

Gooderham KR (1996). Avian pasteurellosis and Pasteurella-like organisms. In: Jordan FTW, Pattison M (eds). *Poultry Diseases*. Pp. 44-51. W. B. Saunders Company Ltd., London.

Gregory CR (1995). Proventricular dilation disease. In: Ritchie BW (ed). *Avian Viruses: Function and Control*. Pp. 439-48. Wingers Publishing Inc., Fort Worth, Florida.

Hartley WJ, Marshall SC (1957). Toxoplasmosis as a cause of ovine perinatal mortality. *New Zealand Veterinary Journal*, 5, 119-24.

Howell J (1992). Viral diseases of New Zealand Poultry. *Surveillance*, 19(2), 17-8.

Imada T, Kawamura H (2003). Avian Nephritis. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 379-83. Iowa State Press.

Jakob-Hoff R (2003). Report to Ministry of Agriculture and Forestry, Biosecurity Authority on the avian animal health surveillance project (contract Number BAH/51/2001), Wildlife Health and Research Centre, Auckland Zoo, Private Bag Grey Lynn, Auckland.

Johnstone AC, Cork SC (1993). Diseases of aviary and native birds of New Zealand. *Surveillance*, 20(3), 35-6.

Kasanga CJ, Yamaguchi T, Wambura PN, Munang'andu HM, Ohya K, Fufushi H (2008). Detection of infectious bursal disease (IBDV) in free-living pigeon and guninea fowl in Africa suggests invovement of wild birds in the epidemiology of IBDV. *Virus Genes*, 36(3), 521-9.*

Loth L (2003). Pacheco's disease ruled out in a Goffin cockatoo. *Surveillance*, 30(4), 13-4.

MAF (1999). Import Risk Analysis for the importation of chicken meat and chicken meat products, Section 2.1 Diseases reported to infect avian species, Ministry of Agriculture and Forestry, Wellington.

MAF (2007). Unwanted Organisms. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, downloaded 5/11/07.

McFerran JB, Adair BM (2003). Egg drop syndrome. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 227-37. Iowa State Press.

McFerran JB, Connor TJ, McCracken RM (1976). Isolation of adenoviruses and reoviruses from avian species other than domestic fowl. *Avian Diseases*, 33, 622-8.

McKenna PB (1998). Checklist of helminth and protozoan parasites of birds in New Zealand. *Surveillance*, 25(Special issue), 3-12.

McNulty MS (2003). Rotavirus infections. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 308-20. Iowa State Press.

Midwinter A (1999). Spirochaetes in New Zealand. *Surveillance*, 26(3), 10-2.

Motha J, Reed C, Gibbons A (1995). The prevalence of Chlamydia in feral pigeons and native psittacines. *Surveillance*, 22(4), 20-2.

OIE (2004). Handbook on Import Risk Analysis for Animals and Animal Products Pp. OIE, Paris.

OIE (2007). Handistatus.http://www.oie.int/hs2/report.asp, downloaded 5/11/07.

Orr MB (1990). Animal Health Laboratory Network. Review of diagnostic cases - April to June 1990. *Surveillance 1*, 7(3), 29-31.

Payne CJ (2003). Big liver and spleen disease. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 1183-6. Iowa State Press.

Payne LM (2002). Lymphoproliferative disease virus of turkeys. In: Jordan FW, Pattison M, Alexander DJ, Fragher T (eds). *Poultry Diseases 5th edition*. Pp. 134-5. WB Saunders, London.

Peroulis I and O'Riley K (2004) Detection of avian paramyxoviruses and influenza viruses amongst wild bird populations in Victoria. *Australian Veterinary Journal* 82, 79-82.

Ritchie BR (1995). Herpesviridae. In: Ritchie BR (ed). *Avian Viruses: Function and Control*. Pp. 171-97. Wingers, Fort Worth, Florida.

Ritchie BR, Harrison GJ, Harrison LR (1994). Avian Medicine: Principles and Application. Pp. 1201. Wingers Publishing, Inc, Lake Worth, Florida.

Saifuddin MD, Wilks CR, Christensen NH, Rice M (1989). Isolation of a reovirus from a broiler chicken flock with high early mortality. *New Zealand Veterinary Journal*, 37, 102-6.

Schat KA (2003). Chicken Infectious Anaemia. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of poultry, 11th edition*. Pp. 182-202. Iowa State Press.

Scullion FT, Scullion MG (2007). Pathologic findings in racing pigeons (*Columba livia domestica*) with "young bird sickness". *Journal of Avian Medicine and Surgery*, 21(1), 1-7.*

Simpson BS (2006). Import Risk Analysis : Passerine Hatching Eggs from the European Union, Ministry of Agriculture and Forestry, Wellington.

Stanislawek W (2001). Avian leucosis subgroup J in New Zealand. *Surveillance*, 28(4), 11-2.

Stanislawek WL, Meers J, Wilks C, Horner GW, Morgan C, Alexander DJ (2001). A survey for paramyxoviruses in caged birds, wild birds and poultry in New Zealand. *New Zealand Veterinary Journal*, 49, 18-23.

Stanislawek WL, Wilks CR, Meers J, Horner GW, Alexander DJ, Manvell RJ, Kattenbelt JA, Gould AR (2002). Avian paramyxoviruses and influenza viruses isolated from mallard ducks (*Anas platyrhynchos*) in New Zealand. *Archives of Virology*, 147(7), 1287-302.

Swanepoel R, Burt FJ (2004). Crimean-Congo haemorrhagic fever. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock.* Pp. 1077-85. Oxford University Press, Oxford.

Thompson EJ, Gumbrell RC, Watson PR (1977). Herpesvirus infection of pigeons. *New Zealand Veterinary Journal*, 25, 24.

Thornton R, Stanislawek W (2003). Pacheco's disease ruled out in at-risk smuggled parrots. *Surveillance*, 30(4), 10-2.

Trivatni M, Ey P, Tran T, Le Mire M, Qiao M, Burrell CJ, Jilbert AR (2001). Sequence comparison of an Australian duck hepatitis B virus strain with other avian hepadnaviruses. *Journal of General Virology*, 82, 373-8.*

Vancanneyt M, Vandamme P, Segers p, Torek U, Coopman R, Kersters K, Hinz KH (1999). *Riemerella columbina* sp. nov., a bacterium associated with respiratory disease in pigeons. *International Journal of Systematic Bacteriology*, 49(1), 289-95.*

Vindevogel H, Pastoret PP (1993). Herpesvirus infections of pigeons and wild birds. In: McFerran JB, McNulty MS (eds). *virus infections of birds*. Elsevier Science Publishers, Amsterdam, London, New York, Tokyo.

Woods LW, Latimer KS, Niagro FD (1993). A retrospective study of circovirus infections in pigeons (1986-1993). *Journal of Veterinary Diagnostic Investigation*, 5, 609-12.

5. Avian Paramyxovirus-1

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Avulavirus*, Species: *Avian paramyxovirus* 1 (APMV-1) (Lamb et al 2005). Strains vary from apathogenic to extreme virulence. Pigeon variant strains of the virus are now generally referred to as pigeon paramyxovirus 1 (PPMV-1).

5.1.2. OIE list

Listed.

5.1.3. New Zealand status

Apathogenic and mildly pathogenic (ICPI < 0.2) strains of APMV-1 occur (Pharo et al 2000; Stanislawek et al 2001; Stanislawek et al 2002). Strains of higher pathogenicity are considered to be exotic notifiable organisms (MAF 2007).

5.1.4. Epidemiology

Newcastle disease (ND) is defined by the *Code* as being caused by APMV-1 viruses above a certain level of pathogenicity in chickens, as measured by either the mean death time of embryonated eggs inoculated with the virus, the intracerebral pathogenicity index (ICPI) in chickens, or the amino acid sequence of a precursor glycoprotein, which acts as a molecular marker of pathogenicity. Virulent strains of the virus cause catastrophic disease and mortalities in chickens, but their virulence may vary for other bird species (Alexander 2003; Alexander 2004).

The domestic pigeon was shown to be relatively resistant to challenge with the virulent strains of ND virus isolated from chickens and other birds from the 1930s to 1980. Disease in pigeons occurred sporadically and was usually associated with outbreaks of ND in other bird species. In the mid 1970s, the so-called pigeon variant of APMV-1 emerged in the Middle East, and spread across Europe. Pigeon APMV-1 isolates can be differentiated from classical NDV strains by the titres obtained in HI tests, by the failure of mouse monoclonal antibodies directed against an HN epitope of NDV Ulster 2C strain to inhibit their HA activity, and by a unique binding pattern of monoclonal antibodies. ICPI determinations on some 50 pigeon isolates showed that they were virulent for chickens (Alexander 2003; Alexander et al 1985). Pigeon isolates may therefore be typical ND strains or the pigeon variant of the virus.

The incubation period for Newcastle disease varies from 2-15 days (Alexander 2003). The *Code* states that for ND the incubation period for international trade purposes is 21 days. Pigeons infected with PPMV-1 have been shown to excrete virus in laryngeal secretions from 2 to 9 days post infection, and in the faeces from 2 to 14 days (Vindevogel and Duchatel 1993).

APMV-1 of low virulence (apathogenic strains with ICPI <0.2) occurs in New Zealand (Pharo et al 2000), chiefly in broiler breeders. Haemagglutination inhibition titres of between 1:16 and 1:1024 were detected in poultry, caged and wild birds (Stanislawek et al 2001). No titres to APMV-1 were detected in 4 species of columbiform birds sampled

(Stanislawek et al 2001).
The pigeon variant of APMV-1 has never been recorded in New Zealand.

Lentogenic APMV-1 of a virulence similar to that in New Zealand has been isolated from birds in Australia (Alexander 2000; Westbury 2001). Five outbreaks of the disease occurred in poultry in Australia between 1998 and 2002 and virulent NDV with ICPI of 1.92 was isolated from poultry in western Sydney in 1998, and ND affected poultry in central New South Wales in 2000 (East et al 2006; Westbury 2001). The last recorded outbreak of ND in Australia was in Victoria in 2002. The Australian veterinary services instituted vigorous measures to eliminate the disease. Since 2002 no further isolations of virulent virus have been made, and Australia is recognised as being free from virulent Newcastle disease (Animal Health Australia 2007). A recent survey of poultry in all states failed to recover any virulent isolates (East et al 2006). Interpretation of serological data is complicated by the variable use of vaccination, but data from states where vaccination was not allowed shows that avirulent viruses continue to circulate.

Diagnostic tests include antibody detection by haemagglutination inhibition or ELISA and virus isolation (Alexander 2004), or PCR methods for detection of viral RNA (Gohm et al 2000; Pham et al 2005; Wise et al 2004).

5.1.5. Hazard identification conclusion

Avirulent to mildly pathogenic strains of APMV-1 continue to circulate in Australia, and surveys have been restricted to commercial poultry. Therefore avirulent APMV-1 strains are considered to be potential hazards in the commodity.

5.2. RISK ASSESSMENT

5.2.1. Entry assessment

Since there have been no reports of PPMV-1 in Australia, the entry assessment for PPMV-1 is negligible. Since precursor virulent strains of virus continue to circulate in Australia, the entry assessment for “precursor virus” strains of APMV-1 is non-negligible.

5.2.2. Exposure assessment

Given that extensive and uncontrolled contact between imported racing pigeons and other birds is inevitable, the likelihood of exposure for New Zealand birds is non-negligible.

5.2.3. Consequence assessment

A precursor strain of non-virulent APMV-1 that mutated to virulence is believed to have been responsible for outbreaks of Newcastle disease in Australia. If a non-virulent strain introduced via imported pigeons were to mutate to virulence, it is likely that serious outbreaks of Newcastle disease would result in exposed avian populations. Therefore the consequences of introduction and establishment of new strains of virus are considered to be non-negligible.

The consequences of introducing Australian strains of APMV-1 for native and wild birds are unknown, but it is assumed that native birds would be susceptible to the introduced viruses.

Virulent APVM-1 is reported to cause rare cases of conjunctivitis in humans that have close contact with infected birds or due to laboratory accidents. Infections are transient and the cornea is not affected. Spread from human to human has not been described (Alexander and Gough 2003). However, there are no reports of low virulence strains of APVM-1

causing disease in humans.
Therefore the consequences for human health would be negligible.

5.2.4. Risk estimation

Entry, exposure and consequence assessments are all non-negligible. As a result the risk estimate for APVM-1 is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified.

5.3. RISK MANAGEMENT

5.3.1. Options

The following points should be considered when drafting risk management options for low virulence strains of APMV-1:

- Freedom from disease signs would not be useful since infections are likely to be sub-clinical.
    1. Serological testing of the flock of origin could be used to demonstrate flock-freedom from infection. However, positive serological tests may indicate previous infection and does not imply that the birds are presently infected.

1) Quarantine alone would not be useful, but quarantine and PCR or virus isolation after they have been in quarantine for 3 weeks could be used to demonstrate freedom from infection.

a) Seroconversion in quarantine would indicate that the virus may be circulating in birds in quarantine.

1. Positive PCR or virus isolation tests would indicate infection, but a positive serological test and negative virus isolation or PCR indicates previous but not present infection.

Virus isolation may take up to 14 days and is therefore less useful than PCR for testing birds in quarantine

1. Vaccination is undesirable because vaccinated birds could harbour low virulence vaccine strains of the virus which do not occur in New Zealand and interfere with the interpretation of diagnostic tests.

The *Code* recommendations are designed to exclude Newcastle disease but not the low virulence strains of APMV-1 which probably circulate in all countries.

The relevant *Code* recommendations are:

> **Recommendations for the importation of live birds other than poultry**
>
> Regardless of the ND status of the country, zone or compartment of origin, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:
>
> - the birds showed no clinical sign suggestive of ND on the day of shipment;
>
> - the birds were kept in isolation approved by the Veterinary Services since they were hatched or for at least the 21 days prior to shipment and showed no clinical sign of infection during the isolation period;

- the birds were subjected to a diagnostic test within 14 days prior to shipment to demonstrate freedom from infection with NDV;

- the birds are transported in new or appropriately sanitized containers.

If the birds were vaccinated against ND, the nature of the vaccine used and the date of vaccination should also be attached to the certificate.

The following options, given in order of ascending stringency, could be considered to effectively manage the risk:

Option 1.

Birds to be imported could:

- be kept isolated from other birds in isolation premises since they were hatched or for at least for the 21 days prior to export; and
- be subjected to a diagnostic test (serology, virus isolation or PCR) for APMV-1, on samples taken at least 14 after entry into quarantine, with negative results; and:
- have not been vaccinated against Newcastle disease;

This option is essentially the 2008 Code recommendation for Newcastle disease, but whereas the *Code* refers to testing only for ND, this option includes APMV type 1 viruses in general.

It is important to note that in this option testing takes place before the birds have been in isolation for the full incubation period of 21 days.

Option 2.

Birds to be imported could:

- be kept isolated from other birds in isolation premises since they were hatched or for at least for the 28 days prior to export; and
- be subjected to a diagnostic test (serology, virus isolation or PCR) for APMV-1, on samples taken at least 21 days after entry into quarantine, with negative results; and:
- have not been vaccinated against Newcastle disease.

This option is similar to option 1, but here testing takes place after the birds have been in isolation for the full 21 day incubation period, thereby achieving a higher level of sensitivity over option 1. The birds would remain in quarantine a further 7 days to allow time for the samples to be tested.

Option 3.

A further option is to use sentinel birds in pre-export quarantine. The number of these would have to be determined according to the size of the shipment, but they would have to be found negative by appropriate diagnostic testing prior to entry into quarantine. Sentinels would be subjected to the same testing regime as the birds intended for export, and any positives among sentinels would disqualify the entire shipment. Choice of sentinel birds might include SPF chickens.

Option 4.

A final option to maximise the likelihood of detecting any viruses in imported birds is, in addition to pre-export testing and isolation, to import the birds into post-arrival quarantine where they would be held for 21-28 days (with or without sentinel birds) and tested as for the previously discussed options.

References

References marked * were seen as summaries in electronic databases.

Alexander DJ (2000). Newcastle disease and other avian paramyxoviruses. *Revue Scientifique et Technique, OIE*, 19(2), 443-62.

Alexander DJ (2003). Newcastle Disease. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 64-87. Iowa State Press.

Alexander DJ (2004). Newcastle disease. In: OIE (ed). *OIE manual of diagnostic tests and vaccines, fifth edition*. Pp. 270-82. OIE, Paris.

Alexander DJ, Gough RE (2003). Avian paramyxoviruses 2-9. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 88-92. Iowa State Press.

Alexander DJ, Russell PH, Parsons G, Abu Elzein EME et al (1985). Antigenic and biological characteristics of avian paramyxovirus type 1 isolates from pigeons - an international collaborative study. *Avian Pathology*, 14, 365-76.

Animal Health Australia (2007). Newcastle disease http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/ Newcastle disease, downloaded 6/11/07.

East I, Kite V, Daniels P, Garner G (2006). A cross sectional survey of Australian chicken farms to identify risk factors associated with seropositivity to Newcastle-disease virus. *Preventive Veterinary Medicine*, 77(3-4), 199-214.

Gohm DS, Thur B, Hoffman MA (2000). Detection of Newcastle disease virus in organs and faeces of experimentally infected chickens using RT-PCR. *Avian Pathology*, 29, 143-52.

Lamb RA, Collins PL, Kolakofsky D, Melero JA, Nagai Y, Oldstone MBA, Pringle CR, Rima BK (2005). Genus *Avulavirus*. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (eds). *Eighth Report of the International Committee on Taxonomy of Viruses*. Pp. 661. Elsevier Academic Press, Amsterdam.

MAF (2007). Unwanted Organisms. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, downloaded 5/11/07.

Pham HM, Konnai S, Usui T, Chang KS, Murata S, Mase M, Ohasi K, Onuma M (2005). Rapid detection and differentiation of Newcastle disease virus by real-time PCR with melting curve analysis. *Archives of Virology*, 150(12), 2429-38.

Pharo H, Stanislawek WL, Thompson J (2000). New Zealand Newcastle disease status. *Surveillance*, 27(4), 8-13.

Stanislawek WL, Meers J, Wilks C, Horner GW, Morgan C, Alexander DJ (2001). A survey for paramyxoviruses in caged birds, wild birds and poultry in New Zealand. *New Zealand Veterinary Journal*, 49, 18-23.

Stanislawek W, Wilks C, Melero JA, Horner GW, Alexander D, Manvell RJ, Kattenbelt JA, Gould EA (2002). Avian paramyxoviruses and influenza viruses isolated form mallard ducks. *Archives of Virology*, 147, 1287-307.

Vindevogel H, Duchatel JP (1993). Paramyxovirus Type 1 infection in pigeons. In: McFerran JB, McNulty MS (eds). *Virus Infections of Birds*. Pp. 363-74. Elsevier Science Publishers, Amsterdam.

Westbury H (2001). Newcastle disease virus: an evolving pathogen. *Avian Pathology*, 30, 5-11.

Wise MG, Suarez DL, Seal BS, Pedersen JC, Senne DA, King DJ, Kapezynski DR, Speckman E (2004). Development of a real-time reverse transcription PCR for detection of disease virus RNA in clinical samples. *Journal of Clinical Microbiology*, 42(1), 329-38.

6. Other Avian Paramyxoviruses

There are eight other serotypes of avian paramyxovirus, APMV 2-9. The significance of these viruses has been reviewed by Alexander (Alexander 2003).

APVM-2 and APVM-3 have not been described in Australia and are not considered potential hazards in this risk analysis (Buckley 2007; Biosecurity Australia 2008).

APMV-4 and APMV-6 are present in New Zealand (Stanislawek et al 2001) and are not potential hazards in the commodity.

APMV-5 is restricted to a very small number of outbreaks of disease in budgerigars (Gough et al 1993; Nerome et al 1978) and is therefore not a potential hazard in pigeons.

APMV-7 has not been reported in Australia and is therefore not a potential hazard.

No reports of isolation of APMV-8 and APMV-9 from species other than healthy anseriformes could be located and no reports could be located of APMV-8 or 9 from Australia. On these grounds neither APMV-8 nor APMV-9 are considered hazards in the commodity.

References

Alexander DJ (2003). Avian paramyxovirues 2-9. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 88-92. Iowa State Press.

Biosecurity Australia (2008) Generic import risk analysis report for chicken meat. Final report. Part C. http://www.daff.gov.au/__data/assets/pdf_file/0004/872788/2008_33c.pdf. Last accessed 9/07/09.

Buckley D (2007). Personal commnication. AFFA.

Gough RE, Manvell RJ, Drury SEN, F. NP, Spackman D, Cooke SW (1993). Deaths in budgerigars associated with a paramyxovirus-like agent. *Veterinary Record*, 133(5), 123.

Nerome K, Nakayama M, Ishida M, Hukumi H (1978). Isolation of a new avian paramyxovirus from budgerigar (*Melopsittacus undulatus*). *Journal of General Virology*, 38, 293-301.

Stanislawek WL, Meers J, Wilks C, Horner GW, Morgan C, Alexander DJ (2001). A survey for paramyxoviruses in caged birds, wild birds and poultry in New Zealand. *New Zealand Veterinary Journal*, 49, 18-23.

7. Avian Influenza Virus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

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

7.1.2. OIE list

The *Code* lists avian influenza (AI) and defines the notifiable form as follows:

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

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

7.1.3. New Zealand status

Influenza type A virus (exotic avian strains) are listed as unwanted, notifiable organisms (MAF 2007). No influenza viruses have been recovered from pigeons in New Zealand and there have been no reported outbreaks of HPNAI in any birds. Viruses of types H1N3, H4N6, H6N4, H11N3 and H5N2 have been isolated from healthy wild mallard ducks (Austin and Hinshaw 1984; Stanislawek 1990; Stanislawek 1992; Stanislawek et al 2002). The following incompletely classified types have also been isolated; H2N?, H7N?, H10N? (Stanislawek 2008). The H5N2 virus was of low pathogenicity (Stanislawek et al 2001; Stanislawek et al 2002). In 2008 a H5N1 virus was isolated from mallards. However, this isolate is a low pathogenicity strain, unlike the high pathogenicity strain responsible for the world-wide pandemic of avian influenza (MAF 2008). A recent survey found no evidence of active infection with NAI viruses in 167 poultry farms. In one case there was evidence of an "historic local exposure event and that there was no evidence of ongoing circulating virus" (Tana et al 2007).

7.1.4. Epidemiology

The family *Orthomyxoviridae* contains four genera: *Influenzavirus A, Influenzavirus B, Influenzavirus C,* and *Thogovirus*. AI is caused only by influenza A viruses. Type A viruses are divided into subtypes according to the antigenic nature of their surface

glycoprotein haemagglutinins (H) and neuraminidases (N). There are currently 16 H types and 9 N types recognised. Virus isolates exhibiting many combinations of the H and N antigens have been found and due to the capacity of the influenza viruses to mutate and recombine the types of viruses circulating is constantly changing. All known highly pathogenic avian influenza (HPAI) isolates have either the H5 or H7 haemagglutinin but H5 and H7 isolates of low virulence are also known. Virulence of AI isolates is related to the presence of sites on the haemagglutinin that allow cleavage into two proteins HA1 and HA2 (Perdue and Suarez 2000). Strains can be classified as virulent based on the amino acid sequences at the cleavage site, but identification of pathogenicity is still primarily determined by measuring an intravenous pathogenicity index in chickens. According to the most recent OIE definition, notifiable AI includes both HPAI strains and LPAI strains of the H5 and H7 subtypes. For the purposes of this risk analysis all influenza A strains found in birds presented for export to New Zealand will be considered.

AI viruses have a world-wide distribution and a broad host range, with the majority of isolates having come from waterfowl. The virus can be transmitted by the oral or oronasal routes, particularly between birds that are in close contact. Long term carriers do not occur but the virus may be excreted for up to 30 days in ducks, 36 days in chickens and 72 days in turkeys. Circulation of the virus in a flock may result in long-term infection. The incubation period of the disease is from 3 days in naturally-infected individual birds, up to 14 days for a flock (Swayne and Suarez 2000; Swayne and Halvorson 2003). The *Code* gives the incubation period as 21 days for the purposes of international trade.

Pigeons have been shown to be resistant to infection with AI viruses (Panigrahy et al 1996), including experimental infection with the highly pathogenic H5N1 virus (Perkins and Swayne 2002) and several references report the absence of evidence of AI infection in surveys of pigeons (Black et al 2004; Toro et al 2000). However, with the emergence of the highly virulent H5N1 that has caused a global pandemic of AI in poultry, much attention has been focussed on this strain. There are reports indicating that pigeons can be infected experimentally with HPAI virus subtype H5N1(Klopfleish et al 2006). A recent review concluded that pigeons are only partially susceptible to influenza A viruses of the H7 subtype and even less susceptible to subtype H5 viruses. It was noted that “Current expert opinion, as supported by the European Food Safety Authority Expert Group report, suggests that pigeons have the potential to act as bridging species between waterfowl and poultry i.e. that they may transfer the disease from infective waterfowl to poultry”. This review concluded that pigeons may spread AI viruses biologically or mechanically (DEFRA 2006). However, no evidence has been found that pigeons are long-term carriers of AI viruses.

There can be no certainty about which AI types are present in any country because strains may change as migratory birds come and go and mutations and recombinations occur.

In Australia, outbreaks of HPAI were recorded in Victoria in 1985, in Queensland in 1995 and in New South Wales in 1997 (Swayne and Suarez 2000). In all cases vigorous action by the Australian veterinary authorities eradicated the virus and Australia has been free of HPNAI since 1997 (Animal Health Australia 2006).

Several diagnostic options are available. Since the nucleocapsid antigen of all Influenza A viruses is similar, the agar gel immunodiffusion test in which nucleocapsid antigen is used can be used as a group test to detect antibody to all Influenza A subtypes. For virus detection an antigen detection ELISA based on a monoclonal antibody to the group specific nucleoprotein that can detect all Influenza A viruses is available as a commercial kit (Alexander 2004). A matrix real time RT/PCR TaqMan is available at MAF’s

Investigation and Diagnostic Centre for detection of influenza A viruses and is preferred to the antigen detection ELISA because of greater sensitivity (Stanislawek 2008).

7.1.5. Hazard identification conclusion

Australia is considered free of HPAI. Furthermore, there can be no certainty about which other AI types are present in any country because strains may change as migratory birds come and go and mutations and recombinations occur. Therefore, reflecting this level of uncertainty, LPAI strains are considered to be potential hazards in the commodity.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

LPAI viruses occur endemically in Australia and the number and type occurring there are likely to be in continual flux. Pigeons are generally resistant to infection with AI viruses but infection with some types has been occasionally reported. Therefore, there is a low likelihood of introducing the viruses in the commodity.

7.2.2. Exposure assessment

Imported pigeons will be introduced into pigeon lofts in which they are in close contact with New Zealand pigeons. They will also be allowed to fly freely in the environment and take part in pigeon races. Therefore, the likelihood that introduced viruses will be transmitted to New Zealand birds is non-negligible.

7.2.3. Consequence assessment

Since influenza viruses are constantly mutating and recombining, the introduction of new viruses will increase the pool of genetic material available, and therefore increase the likelihood of emergence of a virulent strain of the virus. This would require the importation of a low pathogenic H5 or H7 virus that could become virulent through mutation or recombination. Emergence of a virulent strain of virus would have catastrophic consequences for the poultry industry due to massive mortalities and destruction of flocks to prevent the spread of the disease.

Although wild birds frequently carry AI viruses, outbreaks of mortality in wild birds have only been described in one outbreak in terns in South Africa (Hansen 2006). Since the beginning of the H5N1 pandemic, many mortalities have occurred in many species of wild bird, particularly in swans and ducks (Sabirovic et al 2006). However these mortalities have occurred as sporadic cases rather than major outbreaks. The susceptibility of native birds to LPAI or HPAI viruses is not known but swans and ducks at least would be susceptible to the H5N1 strain.

Infections of humans with AI strains occurred sporadically before 2003. As a consequence of the present pandemic in birds caused by the HPAI H5N1 strain, the World Health Organisation has reported that the cumulative number of cases in humans between 2003 and 5 November 2007 was 334 with 105 deaths (World Health Organisation 2007).

It is concluded that the consequence assessment for LPAI strains of virus is considered to be non-negligible for poultry, native birds and human health.

7.2.4. Risk estimation

Entry, exposure and consequence assessments are all non-negligible. As a result the risk estimate for avian influenza is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified.

7.3. RISK MANAGEMENT

7.3.1. Options

When considering options for effectively managing the risks posed by avian influenza, the following points should be considered:

1. Freedom from disease signs would not be useful since infections may be sub-clinical.
    1. Quarantine alone would not be useful in preventing export of sub-clinically infected birds, because LPAI could circulate amongst susceptible birds in quarantine without causing signs of disease.
1. Quarantine and testing birds after they have been in quarantine for 3 weeks could demonstrate recent infection of sero-converting pigeons.
    - As an alternative, virus could be detected in swabs from birds in quarantine using an antigen detection ELISA or a PCR test that is group specific for influenza A viruses.
    - The incubation period stated in the *Code* for international trade is 21 days.
    - Given the uncertainty regarding the existence of different strains of NAI in Australia and New Zealand, risk management measures more stringent than those described in the *Code* could be considered unjustified.

The section of the *Code* relating to live birds is given below:

> **Recommendations for the importation of live birds other than poultry**
>
> Regardless of the NAI status of the country, zone or compartment of origin, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:
>
> 1. the birds showed no clinical sign of infection with a virus which would be considered NAI in poultry on the day of shipment;
> 2. the birds were kept in isolation approved by the Veterinary Services since they were hatched or for at least the 21 days prior to shipment and showed no clinical sign of infection with a virus which would be considered NAI in poultry during the isolation period;
> 3. the birds were subjected to a diagnostic test within 14 days prior to shipment to demonstrate freedom from infection with a virus which would be considered NAI in poultry;
> 4. the birds are transported in new or appropriately sanitized containers.
>
> If the birds have been vaccinated, the nature of the vaccine used and the date of vaccination should be attached to the certificate.

Therefore the available risk management options, listed in ascending order of stringency, are:

Option 1.

Birds to be imported could:

1. be kept in an approved isolation station for at least the 21 days prior to shipment; and

2. be subjected to a diagnostic test (serology, virus isolation or PCR) for influenza A virus on samples taken during the 7 days prior to shipment, with negative results.

This is equivalent to the *Code* requirements, but testing of birds in quarantine occurs before the full incubation period (21 days) has elapsed;

Option 2.

Birds to be imported could:

1) be kept isolated from other birds in isolation premises since they were hatched or at least for the 28 days prior to export; and
2) be subjected to a diagnostic test (serology, virus isolation or PCR) for influenza A virus, on samples taken at least 21 days after entry into quarantine, with negative results.

This option is similar to option 1, but testing takes place after the birds have been in isolation for the full 21 day incubation period, thereby achieving a higher level of sensitivity over option 1. The birds would remain in quarantine a further 7 days to allow time for the samples to be tested.

Option 3.

A further option is to use sentinel birds in pre-export quarantine. The number of these would have to be determined according to the size of the shipment, but they would have to be found negative by appropriate diagnostic testing prior to entry into quarantine. Sentinels would be subjected to the same testing regime as the birds intended for export, and any positives among sentinels would disqualify the entire shipment. Choice of sentinel birds might include SPF chickens.

Option 4.

A final option to maximise the likelihood of detecting any viruses in imported birds is, in addition to pre-export testing and isolation, to import the birds into post-arrival quarantine where they would be held for 21-28 days (with or without sentinel birds) and tested as for the previously discussed options.

References

References marked * were seen as abstracts in electronic data bases.

Alexander D (2004). Highly pathogenic avian influenza. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (mammals, birds and bees).* Pp. 258-69. OIE, Paris.

Animal Health Australia (2006). Avian influenza http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/ Avian influenza, downloaded 5/11/07.

Austin FJ, Hinshaw VS (1984). The isolation of influenza A and paramyxoviruses from feral ducks in New Zealand. *Australian Journal of Experimental Biology and Medical Science*, 62, 355-60.*

Black H, Stanislawek W, Cooper C, Saunders W (2004). Avian virus survey in pigeons. *Surveillance*, 31(4), 20-1.

DEFRA (2006). Avian influenza (bird flu): Low Pathogenic H7N3 outbreak in Dereham, Norfolk April 2006. http://www.defra.gov.uk/animalh/diseases/notifiable/disease/ai/latest-situation/dereham.htm, downloaded 5/11/07.

Hansen W (2006). Avian influenza. In Field Manual of Wildlife Diseases: Birds. http://www.nwhc.usgs.gov/publications/field_manual/chapter_22.pdf, downloaded 5/11/07.

Kawaoka Y, Cox NJ, Haller O, Hongo S, Kaverin N, Klenk H-D, Lamb RA, McCauley J, Palese P, Rimstad E, Webster RG (2005). Influenza A. In: Fauquet CM MM, Maniloff J, Desselberger U, Ball LA (ed). *Eighth Report of the International Committee on Taxonomy of Viruses*. Pp. 685-7. Elsevier Academic Press, Amsterdam.

Klopfleish R, Werner O, Mundt E, Harder T, Teifke JP (2006). Neurotropism of highly pathogenic avian influenza virus A/chicken/Indonesia/2003 (H5N1) in experimentally infected pigeons (*Columbia livia* f. *domestica*). *Veterinary Pathology*, 43, 463-70.

MAF (2007). Unwanted Organisms. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, downloaded 5/11/07.

MAF (2008). Low pathogenic virus no cause for concern. *Biosecurity* 87, 13.

Panigrahy ML, Senne DA, Pedersen JC, Shafer AL, Pearson JE (1996). Susceptibility of pigeons to avian influenza. *Avian Diseases*, 40, 600-4.

Perdue ML, Suarez DL (2000). Structural features of avian influenza virus hemagglutinin that influence virulence. *Veterinary Microbiology*, 47, 77-86.

Perkins LE, Swayne DE (2002). Pathogenicity of a Hong Kong H5N1 pathogenic avian influenza virus for emus, geese, ducks and pigeons. *Avian Pathology*, 46, 43-63.

Sabirovic M, Wilesmith J, Hall S, Coulson N, Landeg F (2006). Outbreaks of HPAI H5N1 in europe during (2005)/(2006). http://www.defra.gov.uk/animalh/diseases/monitoring/pdf/hpai-europe300606.pdf, downloaded 5/11/07.

Stanislawek W (1990). Avian influenza survey of wild ducks. *Surveillance*, 17(2), 13-4.

Stanislawek W (1992). Survey of wild ducks for evidence of avian influenza. *Surveillance*, 19(1), 21-2.

Stanislawek W (2008). Personal communication.

Stanislawek WL, Meers J, Wilks C, Horner GW, Morgan C, Alexander DJ (2001). A survey for paramyxoviruses in caged birds, wild birds and poultry in New Zealand. *New Zealand Veterinary Journal*, 49, 18-23.

Stanislawek WL, Wilks CR, Meers J, Horner GW, Alexander DJ, Manvell RJ, Kattenbelt JA, Gould AR (2002). Avian paramyxoviruses and influenza viruses isolated from mallard ducks (*Anas platyrhynchos*) in New Zealand. *Archives of Virology*, 147(7), 1287-302.

Swayne DE, Halvorson DA (2003). Influenza. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 135-60. Iowa State Press.

Swayne DE, Suarez DL (2000). Highly pathogenic avian influenza. *Revue Scientifique et Technique OIE*, 19(2), 463-82.

Tana T, Rawdon T, Stanislawek W (2007). Avian influenza surveillance programme. *Surveillance*, 34(2), 11-3.

Toro H, Saucedo C, Borie C, Gough RE, Alcaino H (2000). Health status of free living pigeons in the city of Santiago. *Avian Pathology*, 28, 619-23.

World Health Organisation (2007). Cumulative human cases of avian influenza A (H5N1). http://www.who.int/csr/disease/avian_influenza/country/en/ downloaded 5/11/07.

8. Coronavirus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family *Coronaviridae*: Genus: *Coronavirus*, there are three groups of coronaviruses. Group 3 includes *Infectious bronchitis virus* (IBV) (Spaan et al 2005). *Pigeon coronavirus* is proposed as a new species in this group (Cavanagh 2005a).

8.1.2. OIE list

Listed.

8.1.3. New Zealand status

Infectious bronchitis virus is endemic. The status of *Pigeon coronavirus* is not known.

8.1.4. Epidemiology

Infectious bronchitis occurs universally. IBV is endemic in New Zealand. However, many serotypes and genotypes of the virus occur and some of these may not be present in New Zealand. Serotypes can be differentiated by virus neutralisation tests but high and variable cross reactivity occurs with the haemagglutination inhibition test (Cavanagh 2007; Cavanagh and Naqi 2003).

Transmission of IBV occurs rapidly by contact with infected birds (Cavanagh and Naqi 2003), probably by the respiratory route. Faecal-oral transmission is also likely.

Signs of IBV infection in chickens include coughing and gasping, wet eyes, nasal discharge and other respiratory signs. Nephropathic viruses may cause signs of depression, ruffled feathers, wet droppings, and increased mortality (Cavanagh and Naqi 2003).

IBV, or viruses which have high sequence homology with IBV, have been isolated from healthy and diseased pigeons (Barr et al 1988; Wu et al 2005; Zhu et al 2007). In addition Group 3 coronaviruses that are distinct from IBV have been isolated from healthy pigeons (Cavanagh 2005b; Cavanagh 2007; Jonassen et al 2005). All viruses isolated from pigeons have been Group 3 viruses. Signs of IBV infection in pigeons include ruffled feathers, dyspnoea, and mucous discharge from the beak (Barr et al 1988). Distinct histological lesions in trachea, lungs and pancreas were seen after experimental challenge (Wu et al 2005). Pancreatitis has been described in sick pigeons and pigeons experimentally infected with the virus (Qian et al 2006).

Birds that recover from infectious bronchitis may remain carriers for long periods and excrete the virus periodically. IBV has been isolated from eggs up to 43 days after recovery from infectious bronchitis (Cavanagh and Naqi 2003). Group 3 viruses have been isolated from faeces or cloacal swabs of pigeons (Jonassen et al 2005).

There is no indication from the sparse literature on coronaviruses that they cause economically important diseases in pigeons.

The ELISA, immunofluorescence and immunodiffusion tests detect antibody to both group and type specific IBV antigens (Cavanagh and Naqi 2003). The ELISA test gives “earlier reactions and higher antibody titres” than other tests, but lacks type or strain specificity (Gelb 2008). Therefore, it is likely that the ELISA will detect antibody to most, if not all,

Group 3 coronaviruses that infect pigeons. A universal RT-PCR that detects pigeon coronavirus as well as coronaviruses of other species in cloacal swabs has been described (Jonassen et al 2005).

8.1.5. Hazard identification conclusion

Although IBV is endemic in New Zealand, some strains of the virus may not occur here. Since the emergence of SARS-coronavirus in humans in 2002 there has been an increased interest in coronaviruses in other species using modern molecular diagnostic techniques. Group 3 coronaviruses have been detected in pigeons but sequencing now suggests that these viruses are clearly not isolates of IBV (Cavanagh 2005; Cavanagh and Gelb 2008).

These pigeon Group 3 coronaviruses that are distinct from IBV have not been described in New Zealand or Australia, although intensive searches for them have not been conducted. Literature on Group 3 coronaviruses in pigeons is sparse and there is no indication that these viruses are economically important.

Since pigeons were imported into New Zealand from a number of countries for almost 150 years up to 1996, it could be considered likely that coronaviruses that are associated with pigeons in Australia have already been introduced into this country. There is no evidence that pigeons are a source of infection for poultry.

It is therefore considered that there is insufficient evidence to conclude that that Group 3 coronaviruses should be classified as potential hazards in live pigeons from Australia.

References

References marked * were sighted as abstracts in electronic databases.

Barr DA, Reece RL, O'Rourke D, Button C, Faragher JT (1988). Isolation of infectious bronchitis virus from a flock of racing pigeons. *Australian Veterinary Journal*, 65(7), 228.

Cavanagh D (2005a). Template for taxonomic proposal to the ICTV executive committee creating species in an existing genus. *http://talk.ictvonline.org/files/folders/vertebrate-2008/entry232.aspx*, downloaded 14/8/08

Cavanagh D (2005b). Coronaviruses in poultry and other birds. *Avian Pathology*, 34(6), 439-48.

Cavanagh D (2007). Coronavirus avian infectious bronchitis virus. *Veterinary Research*, 38, 281-97.

Cavanagh D, Naqi SA (2003). Infectious bronchitis. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry*, 11th edition. Pp. 101-19. Iowa State Press.

Cavanagh D and Gelb J (2008) Infectious bronchitis. In *Diseases of Poultry*, 12th Edition, Ed YM Saif, Blackwell Publishing, 117-135.

Gelb J (2008). Avian infectious bronchitis. *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. Pp. 443-55. OIE, Paris.

Jonassen CM, Kofstad T, Larsen IL, Lovland A, Handeland K, Follestad A, Lillehaug A (2005). Molecular identification and characterization of novel coronaviruses infecting graylag geese (*Anser anse*r), feral pigeons (*Columbia livia*) and mallards (*Anas platyrhynchos*). *Journal of General Virology*, 86(Pt 6), 1597-607.*

Qian DH, Zhu GJ, Wu LZ, Hua GX (2006). Isolation and characterization of a coronavirus from pigeons with pancreatitis. *American Journal of Veterinary Research*, 67(9), 1575-9.*

Spaan WJM, Brian D, Cavanagh D, De Groot RJ, Enjuanes L, Gorbalenya AE, Holmes KV, Masters P, Rottier P, Taguchi F, Talbot P (2005). Genus *Coronavirus*. In: Fauquet CM MM, Maniloff J, Desselberger U, Ball LA (ed). *Eighth Report of the International Committee on Taxonomy of Viruses*. Pp. 947-55. Elsevier Academic Press, Amsterdam.

Wu ZL, Qian HD, Yao CB (2005). Isolation and identification of pigeon coronaviruses. *Journal of Shanghai Jiaotong University*, 23(3), 275-9.*

Zhu JG, Qian HD, Zhang YL, Hua XG, Wu ZL (2007). Analysis of similarity of the s1 gene in infectious bronchitis virus isolates in Shanghai, China. *Archivos de Medicina Veterinaria*, 39(3), 223-8.*

9. Birnavirus (Infectious Bursal Disease)

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family *Birnaviridae*: Genus: *Avibirnavirus*, Species: *Infectious bursal disease virus* (Delmas et al 2005) Serotypes 1 and 2 are recognised.

9.1.2. OIE List

Listed.

9.1.3. New Zealand Status

Exotic, notifiable disease (MAF 2007).

9.1.4. Epidemiology

IBD is considered to be a serious disease of chickens. Chickens and turkeys are the natural hosts of IBDV (Lukert and Saif 2003). Serotype 1 virus is common in chickens throughout the world (Lukert and Saif 2003), with the exception of New Zealand (Chai et al 2001).

Serotype 1 occurs in numerous pathotypes ranging from avirulent, through "classic" and "variant", to "very virulent" (Lukert and Saif 2003). Serotype 1 primarily affects chickens, but natural infections in turkeys and ducks have been reported (McFerran et al 1980).

Serotype 2 infections occur in chickens, turkeys and ducks (McFerran et al 1980; Smyth and McNulty 1994). IBDV type 2 is believed to be non-pathogenic.

Both classic and variant strains of IBDV are present in Australia (Ignjatovic and Sapats 2002).

Infection with IBDV is rare in free flying birds and in the cases that are reported it is not clear (especially in earlier reports) which serotype is involved (Smyth and McNulty 1994; McFerran et al 1980).

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

9.1.5. Hazard identification conclusion

IBDV is present in Australia but not in New Zealand. As there are reports of the detection of IBDV in free-living pigeons, there is sufficient evidence to reasonably conclude that IBDV should be classified as a potential hazard in live pigeons from Australia.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Serotype 1 IBDV is widespread in chickens in Australia (Ignjatovic and Sapats 2002). It has been identified in pigeons (Kasanga et al 2008). Therefore, the likelihood of entry is assessed to be non-negligible.

9.2.2. Exposure assessment

The virus is transmitted by the faecal-oral route and is extremely resistant to the external environment (Lukert and Saif 2003). Therefore, it possible that the virus could be transmitted from pigeons to naïve New Zealand pigeons or poultry and the likelihood of exposure is assessed to be non-negligible

9.2.3. Consequence assessment

IBD is considered to be one of the most significant diseases of commercial poultry worldwide (Luckert and Saif 2003). Although the most pathogenic strains of the IBDV are not present in Australia, the strains that are present do cause significant economic losses due to disease, immunosupression, and vaccination costs. If these strains are introduced into New Zealand poultry flocks, the cost of eradication could be considerable. Therefore, the consequences of entry of Australian strains of IBDV are considered to be non-negligible for the poultry industry.

Since disease caused by IBDV occurs only in poultry, there would be no consequences to either humans or native animal species in New Zealand.

9.2.4. Risk estimation

Entry, exposure and consequence assessments are all non-negligible. As a result the risk estimate for IBDV is non-negligible and it is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

9.3. RISK MANAGEMENT

9.3.1. Options

When drafting options for effectively managing the risks the following points were considered:

- IBDV causes serious disease in poultry but not in other birds.
- There has been few reports of IBDV isolation from pigeons.
- The likelihood of the virus being present in pigeons imported from Australia is very low.
- Since imported pigeons are likely to have little contact with most commercial poultry, the likelihood of transmission from pigeons is low.
- The virus is not transmitted through the egg.
- OIE recommendations are for poultry and none relate directly to pigeons.
- It is unlikely that any pigeon lofts are regularly inspected by AQIS.

The section of the *Code* relating to domestic birds is as follows:

Recommendations for the importation of domestic birds

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

i. showed no clinical sign of infectious bursal disease on the day of shipment;
ii. come from an establishment which is regularly inspected by the Veterinary Authority;
iii. have not been vaccinated against infectious bursal disease and come from an establishment free from infectious bursal disease as demonstrated by the AGP test; or
iv. were vaccinated against infectious bursal disease (the nature of the vaccine used and the date of vaccination shall also be stated in the certificate).

Although the *Code* does not define "domestic birds", the wording of the chapter suggests that it applies only to chickens.

Therefore, available options for the effective management of the introduction of the virus in the commodity, given in ascending order of stringency are:

Option 1.

Allow unrestricted entry of pigeons based on the assessment that the likelihood of IBDV infection in pigeons is extremely low.

Option 2.

Submit all birds for importation to serological testing within 7 days of shipment, with disqualification of the any birds that test positive. A variant of this option would be to disqualify the entire shipment if any birds test positive.

Option 3.

Test a sample of pigeons from the loft of origin of the birds to be imported. The birds to be imported could be included in the sample and the sample could be big enough to ensure that there is a 95% probability of detecting an infected bird if the prevalence of infection is 5%. As there is no indication that such a prevalence level is likely in a flock of pigeons, there is not a strong basis for determining the sample size necessary and sampling as above would be very conservative.

References

References marked* were sighted as abstracts in electronic data bases.

Chai YF, Christensen NH, Wilks CR, Meers J (2001). Characterisation of New Zealand isolates of infectious bursal disease virus. *Archives of Virology*, 146, 1571-80.

Delmas B, Kibenge FSB, Leong JC, Mundt E, Vakharia VN, Wu JL (2005). Genus Abirnavirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (eds). *Eighth Report of the International Committee on Taxonomy of Viruses*. Pp. 566-7. Elsevier Academic Press, Amsterdam.

Ignjatovic J, Sapats S (2002). Confirmation of the existence of two distinct genetic groups of infectious bursal disease virus in Australia. *Australian Veterinary Journal*, 80(11), 689-94.

Kasanga CJ, Yamaguchi T, Wambura PN, Munang'andu HM, Ohya K, Fufushi H (2008). Detection of infectious bursal disease (IBDV) in free-living pigeon and guninea fowl in Africa suggests invovement of wild birds in the epidemiology of IBDV. *Virus Genes*, 36(3), 521-9.

Lukert PD, Saif YM (2003). Infectious bursal disease. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry*, 11th edition. Pp. 161-79. Iowa State Press.

MAF (2007). Unwanted Organisms. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, downloaded 5/11/07.

McFerran JB, McNulty MS, McKillop ER, Connor TJ, McCracken RM, Collins DS, Allan GM (1980). Isolation and serological studies with infectious bursal disease virus from fowl, turkey and ducks. Demonstration of a second serotype. *Avian Pathology*, 8, 395-404.

Ogawa M, Wakuda T, Yamaguchi T, Murata K, Setiyone A, Fukushi H and Hirai K (1998). Seroprevalence of infectious bursal disease virus in free-living wild birds in Japan. *J. Vet. Med. Sci*. 60 (11), 1277-9.

Smyth JA, McNulty MS (1994). A transmissible disease of the bursa of Fabricius of ducks. *Avian Pathology*, 23, 447-60.

10. Papillomavirus (Papillomatosis)

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Family: *Papillomaviridae* (De Villiers et al 2005).

10.1.2. OIE list

Not listed

10.1.3. New Zealand status

Papillomaviridae are not listed in the register of unwanted organisms. No report of *Papillomaviridae* in pigeons being confirmed in New Zealand birds could be located.

10.1.4. Epidemiology

Papillomaviruses are generally host specific (Ritchie 1995). Most papillomas are found in passerine species. Reports of papillomas or papillomaviruses in pigeons or other columbiform birds could not be located.

10.1.5. Hazard identification conclusion

Given the host-specific nature of papillomaviruses, and the lack of reports of infection of columbiform birds, papillomaviruses are not considered to be potential hazards in the commodity.

References

De Villiers EM, Bernard HU, Broker T, Delius H, Zur Hausen H (2005). Family *Papillomaviridae*. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (eds). *Eighth Report of the International Committee on Taxonomy of Viruses*. Pp. 239-53. Elsevier Academic Press, Amsterdam.

Ritchie BR (1995). Papovaviridae. In: Ritchie BR (ed). *Avian Viruses: Function and Control*. Pp. 127-70. Wingers, Fort Worth, Florida.

11. Arboviruses

HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Arthropod-borne viruses (arboviruses). The viruses concerned belong to the families *Togaviridae, Flaviviridae, Rhabdoviridae, Bunyaviridae* and *Orbiviridae.*

11.1.2. OIE list

Not listed.

11.1.3. New Zealand status

Whataroa virus is the only recognised New Zealand arbovirus of birds (Maguire et al 1967; Miles 1973).

11.1.4. Epidemiology

At least 65 arboviruses have been identified in Australia (Mackenzie et al 1994). Only a small number of these cause serious diseases. Most arboviruses are maintained in a reservoir host which often shows no clinical signs. They are transmitted by an arthropod vector. Several arboviruses cause disease when they are transmitted to a species that is not the natural host (Simpson 1988).

In Australia most species are transmitted by blood sucking flying insects such as mosquitoes and *Culicoides* spp. The School of Integrative Biology (SIB 2005) and the New South Wales Arbovirus Monitoring Programme (Anonymous 2007) have identified the arboviruses of concern in Australia and listed their maintenance hosts as follows:

- Dengue virus – no maintenance host identified
- Sindbis virus – birds are the maintenance hosts
- Ross River virus – maintenance hosts are native mammals
- Barmah Forest virus – maintenance hosts are possibly native mammals
- Japanese encephalitis virus – maintenance host Nankeen night heron and egrets. Cases are restricted to far North Queensland.
- Murray Valley encephalitis virus – maintenance hosts are water birds
- Kunjin virus – maintenance hosts are probably water birds

Dengue fever is maintained in a human/mosquito cycle. While monkeys may also be infected and act as a source of virus (World Health Organisation 2002), birds have not been identified as hosts.

In the USA pigeons infected with St Louis encephalitis and western equine encephalomyelitis developed viraemia for only 2-3 days and were considered unsuitable as sentinels for arboviral diseases (Reisen et al 1992). Sentinel chickens used to determine the risk to humans are known to seroconvert during spread of Murray Valley encephalitis in lowland disease areas of Australia during summer, and virus has been isolated from them (Campbell and Hore 1975). An assessment of the competence of *Culex quinquefasciatus*, a mosquito that seems to have recently expanded its range in New Zealand (Holder and Brown 1999), suggested that it was an unlikely vector of Murray Valley encephalitis and Kunjin virus (Kay et al 1982).

A search of three major electronic databases showed no reports in which pigeons have been identified as maintenance hosts of Australian arboviruses.

11.1.5. Hazard identification conclusion

As pigeons have not been identified as reservoir hosts for Australian arboviruses they are not considered to be potential hazards in the commodity.

References

References marked * were seen as summaries in electronic databases.

Anonymous (2007). NSW arbovirus surveillance and vector monitoring program. http/www.arbovirus.health.nsw.gov.au/areas/arbovirus/viruses/viruses.htm., downloaded 5/11/07.

Campbell J, Hore DE (1975). Isolation of Murray valley encephalitis virus from sentinel chickens. *Australian Veterinary Journal*, 51, 1-3.

Holder P, Brown G (1999). The Mosquitoes of New Zealand. *Surveillance*, 26(4), 12-5.

Kay BH, Fanning ID, Carley JG (1982). Vector competence of *Culex quinquefqsciatus* for Murray valley encephalitis, Kunjin and Ross river viruses in Australia. *American Journal of Tropical Medicine and Hygiene*, 31, 844-8.

Mackenzie JS, Lindsay MD, Coelen RJ, Broom AK, Hall RA, Smith DW (1994). Arboviruses causing human disease in Australian zoographic region. *Archives of Virology*, 136(3-4), 447-67.

Maguire T, Miles JA, Casals J (1967). Whataroa virus a group A arbovirus isolated in south Westland, New Zealand. *American Journal of Tropical Medicine and Hygiene*, 16(3), 371-3.

Miles JA (1973). The ecology of Whataroa virus, an alphavirus, in South Westland, New Zealand. *J Hyg (Lond)*, 71(4), 701-13.

Reisen WK, Hardy JL, Presser SB (1992). Evaluation of domestic pigeons as sentinels for arbovirus activity in southern California. *American Journal of Topical Medicine and Hygiene*, 46(1), 69-79.

SIB (2005). Arboviruses. School of Integrative Biology http/www.sib.uq.edu.au/arboviruses, downloaded 5/11/07.

Simpson DIH (1988). Toga and Flaviviruses. In: McFerran JB, McNulty MS (eds). *Virus Infections of Birds*. Pp. 321-8. Elsevier Science Publishers, Amsterdam, London, New York, Tokyo.

World Health Organisation (2002). Dengue and Dengue haemorrhagic fever. http.//www.who.int/mediacentre/factsheets/fs117/en/, downloaded 5/11/07.

12. Rotavirus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Family: *Reoviridae*, Genus: *Rotavirus*, Species: *Rotavirus A, Rotavirus B, Rotavirus C, Rotavirus D, Rotavirus E* are recognised. *Rotovirus F* and *Rotavirus G* are considered tentative species (Ramig et al 2005).

12.1.2. OIE list

Not listed

12.1.3. New Zealand status

Rotaviruses described as group A rotaviruses have been reported from pigs in New Zealand (Fu and Hampson 1987; Fu and Hampson 1989; Fu et al 1989; Fu et al 1990). Rotavirus has been found in several other species but the serotypes were not clearly identified (Holdaway et al 1982; Schroeder et al 1983).

12.1.4. Epidemiology

Viruses of different species are believed to be unable to reassort their genome segments and are antigenically distinct. Viruses within a species usually have less than 10% sequence variation in their genomes while those from different species have more than 30%. However, since the distinctions between species were not known information in the older literature on classification of species is often lacking.

Rotavirus species occur commonly in many countries. Rotavirus D, F and G have only been found in birds. Most information about rotaviruses in birds relates to chickens and turkeys. In chickens the virus results in mild diarrhoea or subclinical infections. The incubation period in experimentally infected birds is 2-5 days and horizontal infection occurs by direct or indirect contact. Young birds are most commonly infected but older birds that have escaped infection may be more susceptible. Infected chickens and turkeys excrete large numbers of viruses in their faeces. The virus can be readily demonstrated in the faeces of most birds in infected flocks. Peak virus excretion occurs 3 days post infection and sometimes continues for more than 16 days in turkey poults, but there is no evidence for the occurrence of carriers (McNulty 2003).

The virus has been isolated from pigeons and antibody has been demonstrated in populations of pigeons (Gough et al 1992; Minamoto et al 1988; Vindevogel et al 1981). Gough et al (1992) described the recovery of a rotavirus from young pigeons from a loft in which diarrhoea, lethargy and loss of appetite had been reported although the significance of the rotavirus isolate in relation to these signs was not determined.

12.1.5. Hazard identification conclusion

The virus has been found in pigeons and it is not known whether New Zealand pigeons are infected or what strains other than serotype A occur in New Zealand. However, since pigeons were imported into New Zealand from a number of countries for almost 150 years up to 1996, it could be considered likely that any rotaviruses that are associated with pigeons in Australia have already been introduced into this country. There is no evidence that pigeons are a source of infection for poultry.

It is therefore considered that there is insufficient evidence to conclude that rotaviruses should be classified as potential hazards in live pigeons from Australia.

References

References marked * were sighted as abstracts in electronic databases.

Fu ZF, Hampson DJ (1987). Group A rotavirus excretion patterns in naturally infected pigs. *Research in Veterinary Science*, 43(3), 297-300.

Fu ZF, Hampson DJ (1989). Natural transmission of group A rotavirus within a pig population. *Research in Veterinary Science*, 46(3), 312-7.

Fu ZF, Hampson DJ, Blackmore DK (1989). Detection and survival of group A rotavirus in a piggery. *Veterinary Record*, 125, 576-8.

Fu ZF, Hampson DJ, Wilks CR (1990). Transfer of maternal antibody against group A rotavirus from sows to piglets and serological responses following natural infection. *Research in Veterinary Science*, 48(3), 365-73.

Gough RE, Cox WJ, Devoy J (1992). Isolation and identification of rotavirus from racing pigeons. *Veterinary Record*, 130, 273.

Holdaway MD, Todd BA, Schroeder BA, Kalmakoff J (1982). Rotavirus infection in New Zealand. *New Zealand Medical Journal*, 95, 67-9.

McNulty MS (2003). Rotavirus infections. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 308-20. Iowa State Press.

Minamoto N, Oki K, Tomita M, Kinjo T, Suzuki Y (1988). Isolation and characterization of rotavirus from feral pigeon in mammalian cell cultures. *Epidemiology and Infection*, 100(3), 481-92.*

Ramig RF, Ciarlet M, Mertens PPC, Dermody TS (2005). Genus Rotavirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (eds). *Eighth Report of the International Committee on Taxonomy of Viruses*. Pp. 485-96. Elsevier Academic Press, Amsterdam.

Schroeder BA, Kalmakoff J, Holdaway D, Todd BA (1983). Isolation of rotavirus from calves, foals, dogs and cats in New Zealand. *New Zealand Veterinary Journal*, 31, 114-6.

Vindevogel H, Degainis L, Lansival B, Pastoret PP (1981). Incidence of rotavirus, adenovirus and herpesvirus in pigeons. *Veterinary Record*, 108, 285-6.

13. Mycoplasma spp. (Mycoplasmosis)

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Currently at least 26 avian mycoplasmas have been named. Most are recognized as non-pathogenic, whilst only four (*Mycoplasma gallisepticum, M. synoviae, M. meleagridis* and *M. iowae*) are recognized as pathogens in poultry. Three mycoplasmas (*M. columbinum, M. columborale* and *M. columbinasale*) are recognised as being associated with pigeons (Kleven 2003).

13.1.2. OIE status

Only *M. gallisepticum* infection is a listed disease of poultry.

13.1.3. New Zealand status

M. gallisepticum, M. synoviae and *M. meleagridis* have been diagnosed in New Zealand (Lohr 1975). *M. iowae* is considered exotic. Serological evidence of the occurrence of the three endemic species is regularly noted in disease surveillance data reported by the poultry industry to MAF and published in Surveillance magazine. The species found in pigeons have not been recognised but since investigations to detect them have not been reported the true status is unknown.

13.1.4. Epidemiology

Three mycoplasmas (*M. columbinum, M. columborale* and *M. columbinasale)* are recognised as being associated with pigeons, as they are most frequently isolated from pigeon sources, but their association with disease is unproven and they should be regarded as non-pathogenic (Jordan 1996; Vindevogel and Pastoret 1993). *M. columborale* and *M. columbinasale* are considered to be saprophytes (Vindevogel and Pastoret 1993). No reports were found of the *Mycoplasma* spp. that are pathogenic in poultry causing disease in pigeons.

13.1.5. Hazard identification conclusion

The *Mycoplasma* spp. of pigeons are not pathogens and *M. gallisepticum, M. synoviae* and *M. meleagridis* are already present in New Zealand. Therefore, *Mycoplasma* spp. are not considered to be potential hazards in the commodity.

References

Jordan FTW (1996). Avian Mycoplasmosis. In: Jordan FTW, Pattison M (eds). *Poultry Diseases*. Pp. 81-93. W B Saunders Company Ltd, London.

Kleven SH (2003). Mycoplasmosis. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry*. Pp. 719-21. Iowa State Press.

Lohr JE (1975). Mycoplasmosis in poultry. *New Zealand Veterinary Journal*, 14, 151.

Vindevogel H, Pastoret PP (1993). Aetiology of respiratory disease in pigeons. In: McFerran JB, McNulty MS (eds). *Virus Infections of Birds*. Pp. 547-53. Elsevier Science Publishers, Amsterdam, London, New York, Tokyo.

14. *Salmonella* spp, (Salmonellosis)

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agents

Salmonella are Gram-negative rods that ferment glucose and other sugars (but not lactose), and are oxidase negative. Modern nomenclature classifies the genus *Salmonella* into only two species: *S. enterica* and *S. bongori* (Davies 2004; Wray and Davies 2002).

Salmonella enterica is divided into six subspecies. Each subspecies is classified into a number of different serovars. The serovars most commonly causing infections in humans and food animals belong to subspecies 1. The other subspecies are common in reptiles, although some serovars of subspecies *arizonae* are associated with disease in poultry and sheep. According to this latest nomenclature *Salmonella typhimurium* is known as *Salmonella enterica* subspecies *enterica* serovar Typhimurium. For convenience, the earlier convention of writing the serovar name in roman type (Old 1992) is used here, e.g. *Salmonella* Typhimurium.

14.1.2. OIE list

Salmonella serotypes other than *S.* Gallinarum-Pullorum are not included in avian section of the OIE lists.

14.1.3. New Zealand status

S. Arizonae, *S.* Abortus ovis, *S.* Dublin, *S.* Enterititidis DT4, *S.* Typhimurium DT 44 and DT104, *S.* Gallinarum, *S.* Pullorum, and *Salmonella* spp. (exotic affecting animals) are unwanted organisms (MAF 2007).

All *Salmonella* isolates made in New Zealand are referred to the Institute of Environmental Science and Research (ESR) laboratory for typing. Records of all isolates made are available at the ESR Public Health Surveillance website (ESR 2007).

14.1.4. Epidemiology

Salmonellae cause both animal and human disease. In veterinary literature a distinction is usually made between infections caused by the two non-motile serovars, *S.* Pullorum (pullorum disease) and *S.* Gallinarum (fowl typhoid), which are host- adapted serovars of poultry, and the remainder referred to as paratyphoid salmonellae (Gast 2003a; Gast 2003b). The arizonae group of salmonellae, which primarily affect reptiles, mainly affects turkeys amongst avian hosts (Gast 2003a). Although there are over 2,400 serotypes of paratyphoid salmonellae (Davies 2004; Gast 2003a), only about 10% of these have been isolated from poultry (ESR 2007).

The major food borne serotypes are *S.* Typhimurium and *S.* Enteritidis. Although *S.* Typhimurium is common in New Zealand, the definitive phage type (DT) 104, which is of particular concern because of its multiple resistance to commonly-prescribed antibiotics, occurs rarely and its low prevalence has remained stable, as can be verified from the ESR databases (ESR 2007). This is in contrast to the situation in many other countries where *S.* Typhimurium DT104 occurs commonly (Hogue et al 1997; Jones et al 2002).

The most common *Salmonella* serotype of pigeons internationally is *S.* Typhimurium var *Copenhagen*, which causes enteritis and joint infections (Tudor 1991). It is a common

isolate from healthy pigeons (Methner and Lauterbach 2003). Isolates from pigeons are usually of phage types 2 or 99 and are host adapted variants that are less pathogenic for humans than other *S.* Typhimurium strains (Passmans et al 2004; Rabsch et al 2002). *S.* Typhimurium DT 99 has been isolated once from a bovine source in New Zealand (ESR 2007).

The incubation period of *S.* Typhimurium var *Copenhagen* in pigeons is 3-5 days in laterally-transmitted infections, and 2 days following vertical transmission. However since many *Salmonella* infections of pigeons are subclinical, a discussion of incubation period has little meaning. A variety of signs of *Salmonella* infection have been reported from columbiforms. Although *S.* Typihimurium causes septicaemia in squabs, of greater clinical significance is the presence of arthritis in pigeons chronically infected with *S.* Typhimurium O:1,4,12,: H:I:1,2. Many infections with this organism show no clinical signs. Infections with other types of *S.* Typhimurium and other serovars are also likely to be subclinical. Long term carriers of many *Salmonella* serotypes have been described in many animals species and it could be assumed that pigeons might be carriers of a wide variety of *Salmonella* serotypes.

A large number of *Salmonella* serotypes occur in Australia, including *S.* Pullorum, but Australia has been free of *S.* Gallinarum since 1952 (OIE 2006). *S.* Pullorum was last diagnosed in New Zealand in 1985, and *S.* Gallinarum and *S.* Arizonae have never been recorded in New Zealand (OIE 2006). Since these are poultry adapted strains, the probability of these serovars infecting pigeons is very low and they need not be considered differently from those posing a higher entry risk.

Diagnosis of subclinical *Salmonella* infections presents problems. Serological tests are available only for certain specific serotypes. In particular, agglutination tests are used for *S.* Pullorum and *S.* Gallinarum infections in poultry and an ELISA is available for *S.* Enteritidis. These methods are used as flock tests rather than for individual animals. *Salmonella* can be cultured from faeces or cloacal swabs but in the case of carriers, excretion of the organism may be intermittent and culturing should be done on more than one occasion. Flock testing is likely to be more sensitive than testing individual animals.

14.1.5. Hazard identification conclusion

Since *Salmonella* spp. are zoonotic agents and responsible for disease in all species of animals and birds, introduction of exotic and rare *Salmonella* serotypes should be avoided. Therefore, they are regarded as potential hazards in this risk analysis.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

Since many *Salmonella* serotypes occur in Australia and pigeons can be long-term carriers of the organisms the likelihood of entry is considered to be non-negligible.

14.2.2. Exposure assessment

Imported birds will be introduced into lofts and exposure of New Zealand birds to imported carriers is inevitable. Introduced pigeons and local pigeons infected by them are likely to be traded within New Zealand and transported to take part in races, at which they will be in contact with other birds. Therefore, the likelihood of exposure is non-negligible.

14.2.3. Consequence assessment

Since contact between New Zealand pigeons and introduced birds is inevitable, introduced *Salmonella* serotypes are likely to be spread widely and infect pigeons, other birds, other animals and people. The consequences are therefore non-negligible.

Since humans will be in contact with pigeons and their faeces, transmission to humans is likely to occur. The occurrence of new types of salmonellosis in humans is likely to cause loss of productivity, expenses for medical treatment and even in rare cases death. All animals are susceptible to infection with *Salmonella* spp., and therefore wild and feral birds and animals could become infected.

Introduction of new *Salmonella* serovars are likely to cause infections in humans and domestic, wild, and feral animals. The consequences of introduction are considered to be non-negligible.

14.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for exotic *Salmonella* serovars is non-negligible and they are classified as a hazard in the commodity. Therefore risk management measures can be justified.

14.3. RISK MANAGEMENT

14.3.1. Options

The following points should be considered when drafting options to prevent the introduction of exotic *Salmonella* spp.:

- Since long-term carriers may occur, quarantine on its own is unlikely to be effective.
- Suitable generic serological tests for a wide variety of *Salmonella* spp. are not available.
- The testing of cloacal swabs from a sample of birds could be used to detect infected flocks. Information is not available for the prevalence of many *Salmonella* spp. in pigeons and selection of sample size is therefore somewhat arbitrary. To compensate for the fact that cases of infection may occur sporadically or at low prevalence a sample that is large enough to detect at least one infected pigeon with a confidence of 95% if the prevalence is 5% could be tested.
- Samples for testing could include cloacal samples from a sample of birds in the flock and also swabs from the loft environment such as litter samples, swabs from the floor surface, perches and nest boxes.
- Flock freedom from *Salmonella* infection would provide a high level of assurance of freedom and would compensate for the decreased sensitivity due to intermittent excretion, when testing individual birds.

There are no *Code* recommendations relating to *Salmonella* spp. in birds other than poultry.

Therefore, available options in ascending order of stringency are:

Option 1.

- Within the 10 days prior to shipment, cloacal swabs from individual pigeons could be cultured; and

- all isolates of *Salmonella* spp. could be identified to serovar and in the case of Salmonella Enteritidis and Salmonella Typhimurium to phage type, and the results forwarded to MAFBNZ for consideration.

MAFBNZ could prohibit the importation of any birds from flocks infected with *Salmonella* serovars that are exotic to New Zealand. In the case of birds infected with *Salmonella* that occur in New Zealand the importer could decide whether to proceed with the importation.

Option 2.

1. Pigeons to be imported could originate from flocks where salmonellosis has not been diagnosed for the last 12 months; and
2. before transfer of individual birds into quarantine, cloacal swabs could be tested from a sufficient number of birds in the flock of origin to provide 95% confidence that the sample will detect at least one infected animal if the prevalence of Salmonella infection is 5% or higher. In addition, environmental swabs could be tested. At the discretion of MAFBNZ pooling of appropriate numbers of samples could be allowed. Only if all tests on the flock are negative for *Salmonella* spp. of significance as determined by MAFBNZ should individual birds be moved into quarantine for a minimum of 3 weeks; and
3. within 10 days of shipment swabs from individual birds could be cultured; and
4. all isolates of *Salmonella* spp. should be identified to serovar and in the case *S.* Enteritidis and *S.* Typhimurium to phage type, and the results forwarded to MAFBNZ for consideration. MAFBNZ could prohibit the importation of any birds from flocks infected with Salmonella serovars that are exotic to New Zealand. In the case of birds infected with Salmonella that occur in New Zealand the importer could decide whether to proceed with the importation.

Option 3.

A possible variation on option 2 is that swabs could be taken from individual birds before entry into quarantine and twice while in quarantine. All other requirements could be the same as in option 2.

References

References marked * were seen as summaries in electronic databases.

Davies Rl (2004). Salmonellosis. In: OIE (ed). *OIE Manual of Standards for Diagnostic Tests and Vaccines for Terrestrial Animals (mammals, birds and bees)*. Fifth Edition. Pp. 1018-32. OIE, Paris.

ESR (2007). Enteric reference laboratory reports http://www.surv.esr.cri.nz/enteric_reference/enteric_reference.php, downloaded 5/11/07.

Gast RK (2003a). Paratyphoid infections. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry*, 11th edition. Pp. 583-613. Iowa State Press.

Gast RK (2003b). Salmonella infections. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry*, 11th edition. Pp. 567-8. Iowa State Press.

Hogue A, Agula F, Johnson R, Petersen K, Saini P, Schlosser W (1997). Situation Assessment: Salmonella Typhimurium DT 104, United States Department of Agriculture, Food Safety and Inspection Service, Washington DC 20250. http://www.fsis.usda.gov/OPHS/stdt104.htm, downloaded 5/11/07.

Jones YE, Chappell S, McLaren IM, Davies RH, Wray C (2002). Antimicrobial resistance in Salmonella isolated from animals and their environment in England and Wales from 1988 to 1999. *Veterinary Record*, 150, 649-54.

MAF (2007). Unwanted Organisms. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, downloaded 5/11/07.

Methner U, Lauterbach L (2003). The detection of Salmonella typhimurium varatio copenhagen DT2 in purebred pigeons. *Deutsche Tierarzliche wochenschrift*, 110(6), 239-44.*

OIE (2006). Handistatus. http://www.oie.int/hs2/report.asp, downloaded 5/11/07.

Old DC (1992). Nomenclature of Salmonella. *Journal of Medical Microbiology*, 37, 361-3.

Passmans F, Van Immerseel F, Hermans K, Heyndricks M, Collard JM, Ducatelle R, Haesebrouck F (2004). Assessment of virulence of pigeon isolates of Salmonella enterica serovar Typhimurium variant Copenhagen for humans. *Journal of Clinical Microbiology*, 42(2), 2000-2.

Rabsch W, Andrews HL, Kingsley RA, Pragaer R, Tschape H, Adams LG, Baumler AJ (2002). Salmonella enterica Serotype Typhimurium and its host adapted variants. *Infection and Immunity*, 70, 2249-55.*

Tudor DC (1991). *Pigeon Health and Disease*. pp. 54-9. Iowa State University Press, Ames.

Wray C, Davies RH (2002). Enterobacteriaceae. In: Jordan FTW, Pattison M, Alexander DJ, Faragher T (eds). *Poultry Diseases.* Fifth Edition. Pp. 95-130. WB Saunders, London.

15. *Coxiella burnetii* (Q fever)

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Coxiella burnetii.

15.1.2. OIE status

Listed as a disease of multiple species but there is no chapter on the disease in the OIE *Code*.

15.1.3. New Zealand status

Notifiable organism (MAF 2007).

15.1.4. Epidemiology

C. burnetii is known to infect a wide number of vertebrate species including various pigeons and other birds (Campbell 1994; Martinov et al 2004; Sakai et al 1998). Infections in birds are sub-clinical. The organism is maintained in wild bird populations and transmitted by ticks (Lang 1990). Chickens have been found to excrete the organism in their faeces from 7 to 40 days post infection (Little 1983), but whether a similar period of excretion occurs in pigeons is unknown. In one study, experimentally infected birds remained infected for up to 3 months (Sethi et al 1978). Infection in birds is generally considered to be unimportant in the epidemiology of the disease. However, a case has been described in which pigeons were thought to have infected five members of a family (Stein and Raoult 1999). Lang (1990) has reviewed the literature and quotes evidence that suggests that Q fever can be transmitted to humans by infected poultry, through consumption of raw eggs or aerosolised fomites.

Q fever does not occur in New Zealand but it is endemic in most of the world. In Australia, there are approximately 600 annually reported cases of Q fever, 200 of which result in hospitalisation and about three fatalities. Ninety per cent of cases occur in New South Wales and southern Queensland. Workers in the meat industry are at greatest occupational risk (Anonymous 2002).

Antibody can be detected in birds using the complement fixation test, immunofluorescence or agglutination tests, or ELISA. The organism can be detected in tissues or faeces by PCR or the organism can be isolated by injection of mice or guinea pigs (Rousset et al 2004).

15.1.5. Hazard identification conclusion

Since the disease is exotic to New Zealand and occurs endemically in Australia the organism is considered to be a potential hazard in this risk analysis.

15.2. RISK ASSESSMENT

15.2.1. Entry assessment

Infection of pigeons with *C. burnetii* is rarely reported anywhere and has never been reported from Australia. However, the organism is endemic in Australia and pigeons are

susceptible to infection. Therefore, the likelihood of importing infected pigeons is low but non-negligible.

15.2.2. Exposure assessment

Imported pigeons will be integrated into New Zealand pigeon flocks. It has been suggested that the infection may be carried between pigeons by ticks. Although New Zealand pigeons are believed to be free from ticks, the possibility of other means of transmission between pigeons in close contact with each other cannot be ignored. The likelihood of transmission to New Zealand birds and the subsequent establishment of the organism is low but non-negligible.

Evidence that humans have been infected with *C. burnetii* by pigeons or other birds is rare, but since it has been suggested that it may occur, the likelihood is assessed as non-negligible. It is also likely that the risk of exposure of humans in New Zealand as a result of the importation of pigeons from Australia will be considerably lower than the risk posed by the hundreds of thousands of New Zealanders who travel to Australia each year. These include visitors to, and workers in, Australian meat plants, and people that have been exposed to ticks and tick faeces.

15.2.3. Consequence assessment

Since no clinical effects of *C. burnetii* infection have been demonstrated in birds, the consequence assessment for New Zealand's avian fauna is negligible.

C. burnetii can cause a serious disease in humans, therefore the consequences for humans from exposure to Q fever in imported pigeons is non-negligible.

15.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for *C. burnetii* is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified.

15.3. RISK MANAGEMENT

15.3.1. Options

The following points should be considered when drafting options to manage the risk of importing *C. burnetii* in the commodity:

- Birds infected with *C. burnetii* show no signs and remain carriers for at least 3 months. Therefore, quarantine without testing would not be useful.
- However, if animals are quarantined for a period that exceeds the incubation period and then tested, both chronically infected animals and newly infected animals would be detected. Therefore, pigeons could be quarantined in tick-free premises for 3 weeks and tested by a serological test within the 5 days prior to shipment.

The available options in ascending order of stringency are:

Option 1.

Since pigeons were imported into New Zealand from a number of countries for almost 150 years up to 1996, it may be likely that any *C. burnetii* associated with pigeons in Australia has already been introduced into this country. Given that Q-fever has not become established here, it could be considered that no restrictions are necessary.

Option 2.

Pigeons for export could:

i. be maintained tick-free and quarantined in tick free premises for at least 21 days; and
ii. be tested by an antibody detection ELISA within the 5 days prior to shipment, with negative results;

Option 3.

Pigeons for export could:

i. be maintained tick-free and quarantined in tick free premises for 21 day while in contact with a suitable number of serologically negative sentinel pigeons; and
ii. be tested by an antibody detection ELISA together with the sentinel pigeons within five days prior to shipment, with negative results.

References

References marked * were seen as summaries in electronic databases.

Anonymous (2002). Q fever register developed to address concern in the meat industry. *NAW Public health Bulletin*, 13(5), 113, http://www.health.nsw.gov.au/public-health/phb/phbmay02.pdf, downloaded 6/11/07.

Campbell RSF (1994). Pathogenesis and pathology of the complex rickettsial infections. *Veterinary Bulletin*, 64(1), 1-24.

Lang GH (1990). Coxiellosis (Q fever) in Animals. In: Marrie TJ (ed). *Q fever. Volume 1. The disease*. CRC Press, Boca Raton.

Little TWA (1983). Q fever: an enigma. *British Veterinary Journal*, 139, 277-83.*

MAF (2007). Unwanted Organisms. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, downloaded 5/11/07.

Martinov SP, Pandarov S, Popov GV (2004). Seroepizootology of Q fever in Bulgaria during the last 5 years. *European Journal of Epidemiology*, 54(4), 425-7.

Rousset E, Russo P, Pepin M, Aubert MF (2004). Q fever. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (mammals, birds and bees).* Pp. 387-98. OIE, Paris.

Sakai H, Shirota K, Kano C, Abe S, Sugimoto T, Morita K, Takashima I, Maruyama T, Yamaguchi T, Fukushi H, Hirai K (1998). Coxiellosis in domestic and wild birds in Japan. *Journal of Wildlife Diseases*, 34(2), 310-6.

Sethi MS, Bhupender S, Yadav MP (1978). Experimental infection of *Coxiella burnetii* in chicken: Clinical symptoms, serological response and transmission through egg. *Avian Diseases*, 22, 392-5.

Stein A, Raoult D (1999). Pigeon pneumonia in Provence. *Clinical Infectious Diseases*, 29(3), 617-20.

16. *Borrelia anserina* (Avian Spirochaetosis)

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

Borrelia anserina is a large spirochaete, labile in the environment, and sensitive to desiccation. As with other *Borrelia* spp., it is transmitted by arthropod vectors (Barnes 2003).

16.1.2. OIE List

Listed by OIE

16.1.3. New Zealand Status

B. anserina is listed on the register of unwanted organisms. Neither *B. anserina* nor *Argas* spp. ticks (the vector of avian spirochaetosis) have been recorded in New Zealand.

16.1.4. Epidemiology

Avian spirochaetosis is an acute disease of chickens, turkeys, pheasants, geese and ducks. Argasid ticks are the major vectors, especially *Argas persicus* (the fowl tick). The occurrence of the disease corresponds to the tropical and subtropical distribution of the fowl tick (Barnes 2003). There is evidence of specificity amongst the vectors of the various species of *Borrelia*. *B. duttonii* and *B. anserina* cannot be efficiently transmitted by each other's natural vectors, *Ornithodorus moubata* and *Argas persicus*, respectively (Barbour and Hayes 1986). *B. anserina* is not resistant to the environment outside the host, and the organisms disappear from tissues at the same time or shortly after they disappear from the circulation (Barnes 2003). It has been known since 1932 that pigeons are relatively resistant to experimental infection with *B. anserina* (Barnes 1991).

Argas persicus occurs in Australia (Hart 1985), as does *B. anserina* (Commonwealth of Australia 2001; Hart 1985; Petney et al 2004). No record could be located of its occurrence in pigeons in Australia.

16.1.5. Hazard identification conclusion

Since *B. anserina* is exotic to New Zealand and it is present in Australian birds, the organism is considered to be a potential hazard in the commodity.

16.2. RISK ASSESSMENT

16.2.1. Entry assessment

Since *Argas persicus* occurs in Australia it is considered that there is a non-negligible likelihood that pigeons arriving in pre-export isolation might be infected with *B. anserina.*

16.2.2. Exposure assessment

Since the vector (*Argas persicus*) is not present in New Zealand the disease could not be transmitted and the likelihood of exposure and establishment in New Zealand is considered to be negligible

16.2.3. Risk estimation

Since the exposure assessment is negligible, the risk estimate for *B. anserina* is negligible and it is not classified as a hazard in the commodity. Therefore risk management measures are not justified.

References

References marked * were seen as summaries in electronic databases.

Barbour AG, Hayes SF (1986). Biology of Borrelia species. *Microbiological Reviews*, 50(4), 381-400.

Barnes HJ (1991). Spirochetosis. In: Calnek BW (ed). *Diseases of Poultry. Ninthth editon*. Pp. 304-10. Iowa State University Press, Ames Iowa.

Barnes HJ (2003). Miscellaneous and sporadic bacterial infections. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry*. Pp. 845-62. Iowa State Press.

Commonwealth of Australia (2001). Generic import risk analysis (IRA) for uncooked chicken meat . Issues paper2. http://www.daffa.gov.au/__data/assets/pdf_file/11292/2001-16a.pdf., downloaded 5/11/07.

Hart L (1985). Spirochaetosis. In: Beveridge W HL (ed). *Animal health in Australia. Volume 7. Viral, Bacterial and Fungal Diseases of Poultry*. Pp. 131-3. Australian Government Publishing Service, Canberra.

Petney TN, Andrews RH, McDiarmid LA, Dixon BR (2004). *Argas perciscus* sensu stricto does occur in Australia. *Parasitology Research*, 93(4), 296-9.

17. Protozoal Blood Parasites

HAZARD IDENTIFICATION

17.1.1. Aetiological agents

Protozoal blood parasites of the genera *Haemoproteus, Leucocytozoon, Plasmodium* and *Trypanosoma*.

17.1.2. OIE list

Not listed.

17.1.3. New Zealand status

H. danilewsky, L. tawaki, L. fringillinarum, P. cathemerium, P. elongatum, P. relictum, and *Plasmodium* spp. have been recorded in New Zealand (McKenna 1998).

There are no species listed as unwanted or notifiable except *L. struthionis* which is listed as an "other exotic organism" (MAF 2007).

17.1.4. Epidemiology

Many species of blood parasites belonging to the genera *Haemoproteus, Leucocytozoon* and *Plasmodium* have been described in birds world-wide. These parasites have generally been identified in clinically normal birds (Rukhsana 2005). A large number have been described. For example, in a single investigation 112 new host-parasite associations were described (Valkiunas et al 2005).

Most haematozoa do not cause significant disease in their avian hosts. Some may be opportunistic pathogens, particularly when in combination with other infections. The pathogenicity of parasites has seldom been proven by experimental infection. In a study on the pathogenicity of *L. simondi*, a parasite that has often been associated with high mortality in ducks and geese, it was found that experimental infection did not cause clinical signs or affect the growth rates of infected American black ducks and mallards (Shutler et al 1999).

It appears that the majority of parasites described are non-pathogenic or mildly pathogenic in the species in which they have evolved. However, when transmitted to species that have not previously been exposed to the parasite they may be pathogenic. The best example of this is the introduction of the mosquito vector *Culex quinquefasciatus* and its associated parasite *P. relictum* into Hawaii. This event caused habitat restrictions and extinctions of several Hawaiian bird species (Atkinson and LaPointe 2005). Another example is the susceptibility of penguins to avian malaria (Duignan 2001). However, some species may adapt to the presence of new species of parasites. Infection with, and recovery from, avian malaria provides a reproductive advantage to the Hawaiian honeycreeper and adaptation of Hawaiian bird species to avian malaria may already be occurring (Kilpatrick et al 2005).

Large numbers of identified and unidentified haematozoa, belonging to the *Haemoproteus, Leucocytozoon, Plasmodium* and *Trypanosoma* genera, have been described in Australia (Mackerras and Mackerras 1960). These were present in Australian birds during the period when there were few or no restrictions on the importation of pigeons or other birds from Australia.

Haemoproteus spp.

More than 120 species of *Haemoproteus* have been reported from birds, but most species have low pathogenicity (Bermudez 2003). However, outbreaks of disease have been associated with the parasite (Resende et al 2001), but it is not known whether other infections were involved.

In pigeons, *H. columbae* occurs very commonly and has been reported at prevalences of 6-100% in normal pigeons in many locations (Gicik and Arslan 2001; Klei and De Giusti 1975; Mushi et al 1999). *H. sacharovi* also occurs in pigeons (Glass et al 2002).

Haemoproteus spp. have been recognised in 54 species of Australian birds (Harrigan 1981).

H. columbae has not been described in New Zealand, probably because the vector *Pseudolynchia canariensis* is not present. *H. danilewsky* has been described in blackbirds, skylarks and song thrushes (McKenna 1998).

Leucocytozoon spp.

L. marchouxi has been found in pigeons (Ozmen et al 2005) and is the only *Leucocytozoon* spp. occurring in Columbiformes. It occurs in pink pigeons, doves, pigeons, and rock pigeons (*Columba guinea*) (Pierce et al 1977; Earle 1997; Pierce and Greenwood 1997; Scullion and Scullion 2007). It is common in Mauritian pink pigeons and was probably introduced into Mauritius by exotic columbids. Juveniles are more likely to be infected than adults and are more susceptible to infection. Infection has been described as the primary cause of death in some cases, but "the pink pigeon has acquired sufficient immunity to now be a maintenance host of the parasite" (Swinnerton et al 2005).

Plasmodium spp.

About 65 species of *Plasmodium* from more than 1,000 species of birds have been described but 35 or fewer are considered valid. The parasites probably occur world-wide (Bermudez 2003). Pathogenicity of various species ranges from non-pathogenic to virulent. Pathogenic species can cause severe anaemia and death. *P. relictum* is a serious pathogen of Hawaiian birds that have not evolved with the parasite. At least three *Plasmodium* spp., including *P. relictum*, have been described in New Zealand, indicating that, for these species at least, suitable insect vectors are present. *P. relictum* has spread widely in New Zealand (Derraik 2006) and must be regarded as endemic. However, as New Zealand has only 16 identified species of mosquito (Holder and Brown 1999) and no *Culicoides* spp., the generally low prevalence of haematozoa could be due the paucity of suitable vectors. New Zealand native birds could be a naïve and highly susceptible population in relation to introduction of haematozoa in birds from the rest of the world. Haematozoon infections are generally diagnosed by the microcopic examination of blood smears. However, recently PCR tests have been developed for avian malaria and because of the similarity of DNA sequences in *Plasmodium*, *Leucocytozoon* and *Haemoproteus* spp., these tests detect a wide range of malarial parasites and are much more sensitive than blood smear examination (Cosgrove et al 2006; Hellgren et al 2004; Tomkins and Gleeson 2006).

17.1.5. Hazard identification conclusion

Haematozoa are seldom recognised as being of any economic significance and are not listed in import health standards of other countries as can be determined by perusal of the Overseas Market Access Requirements (OMARS) on the MAF website. The position with regard to the occurrence of these parasites in New Zealand is not certain and their ability to establish here is unknown. Since many of New Zealand's domestic pigeons originated

from minimally restricted importations from Australia prior to 1996, it seems likely that organisms occurring in racing pigeons in Australia may have previously been imported. However, several species of haematozoa occur in Australia that have not been described in New Zealand and it is not known whether New Zealand's native birds are susceptible to these parasites. Therefore, the parasites are assessed to be potential hazards.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

Since a variety of haematozoa occur in Australia the likelihood that imported pigeons could be carrying exotic haematozoa is non-negligible.

17.2.2. Exposure assessment

Imported pigeons would be mixed with New Zealand pigeons, but the haematozoa are not contagious and require the presence of a suitable vector for transmission. The main vector (*Pseudolynchia canariensis*) of the most common parasite of pigeons (*H. columbae*) is not present. However, several species of haematozoa have been described in New Zealand and therefore suitable vector systems for at least some haematozoa are present. The likelihood of transmission of protozoa to other bird species is assessed as non-negligible.

17.2.3. Consequence assessment

Pigeons have frequently been imported from Australia in the past with no adverse effects on New Zealand pigeons or native birds. Descriptions of disease caused by haematozoa in pigeons are rare. Therefore, the likelihood of importing parasites pathogenic to pigeons is low. However, the introduction of parasites to naïve bird populations had serious consequences for native birds in Hawaii (Atkinson and LaPointe 2005) and vector systems for at least some protozoal parasites are already present in New Zealand. The most pathogenic of the malarial parasites, *P. relictum*, is already endemic. Therefore, the consequences of introducing exotic haematozoan parasites is assessed to be non-negligible for pigeons and other birds.

There would be no consequences for human health since haematozoa of birds are not zoonotic.

17.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for haematozoa is non-negligible and they are classified as hazards in the commodity. Therefore, risk management measures can be justified.

17.3. RISK MANAGEMENT

17.3.1. Options

The following points should be considered when drafting options for preventing the importation of haematozoa:

- Since pigeons were imported into New Zealand from a number of countries for almost 150 years up to 1996, it could be considered likely that any haematozoa that may be associated with pigeons in Australia have already been introduced into this country.

- Birds are long term carriers of many haematozoa and quarantine would not be a useful measure to prevent introduction.
- Serological tests are not available that could be used to identify carriers of the many possible haematozoan parasites.
- Some avian malarial parasites can be diagnosed by sensitive PCR tests
- Microscopic examination of blood smears is the only available method of identifying all species of parasites but it lacks sensitivity.
- To improve the sensitivity of both blood smear examination and PCR, blood could be examined from a sample of birds from the flock of origin rather than individual birds. Since the prevalence of haematozoa in pigeons is unknown the sample size could be chosen assuming a low prevalence to provide high flock sensitivity e.g. the sample could be taken from a sufficient number of pigeons to provide 95% confidence that the sample will contain at least one infected bird if the prevalence of infection is 5%.
- Strict measures could be implemented to prevent the introduction of haematophagous arthropod parasites that could act as biological or mechanical vectors of protozoal parasites.

The *Code* does not contain any recommendations relating to haematozoa.

Available options for measures to effectively manage the risk of haematozoa, in order of ascending stringency, are:

Option 1.

Birds could be imported without restrictions, based on the assessment that since importation of pigeons was permitted for 150 years until 1996, haematozoa of pigeons in Australia are likely to have already been introduced into this country.

Option 2.

Blood smears from birds to be imported could be examined microscopically for haematozoa. Birds infected with exotic haematozoa could be prohibited from being imported.

Option 3.

PCR as primary test with further confirmatory tests on positives

1. Blood samples from birds to be imported could be examined by PCR for haematozoa; and
2. All birds positive to the PCR test could be further investigated to determine the species of parasite involved. All birds infected with exotic parasites could be disqualified.

If confirmatory testing is not possible (e.g. the species cannot be identified) then a decision could be based on the PCR results alone.

Option 4.

Use of blood smear examination and PCR in series, assuming that exotic species of Haematozoa can be identified by each of these tests (note, if this assumption is not fulfilled, then this option cannot be applied).

i. Blood smears from birds to be imported could be examined microscopically for haematozoa. Birds infected with exotic haematozoa could be disqualified; and

ii. Blood from birds to be imported could be examined by PCR for haematozoa. Birds infected with exotic haematozoa could be prohibited from being imported.

Option 5.

Flock of origin testing

i. Blood smears from number of birds in the flock of origin could be examined microscopically and by PCR. The sample size could be as large as is practical for the particular case and could include all the birds to be imported. Haematozoa present in smears could be identified to genus and species. MAFBNZ could reserve the right to refuse entry of any birds from flocks infected with exotic haematozoa.

References

References marked * were seen as abstracts in electronic databases.

Atkinson CT, LaPointe DA (2005). *Plasmodium relictum* (micro-organism) http://www.issg.org/database/species/ecology.asp?si=39&fr=1&sts=), downloaded 5/11/07.

Bermudez AJ (2003). Miscellaneous and sporadic protozoal infections. In Diseases of poultry. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 1010-23. Iowa State Press.

Cosgrove CL, Day KP, Sheldon KC (2006). Coamplification of *Leucocytozoon* by PCR diagnostic tests for malaria : a cautionary note. *Journal of Parasitology*, 92(6), 1362-5.

Derraik JGB (2006). Bitten birds. *Biosecurity*, 65, 16-7.

Duignan PJ (2001). Diseases of penguins. *Surveillance*, 28(4), 5-11.

Earle RA (1997). Haematozoa of feral rock doves and rock pigeons in mixed flocks. *South African Journal of Wildlife Research*, 23(4), 98-100.

Gicik Y, Arslan MO (2001). Blood parasites of wild pigeons in Ankara district. *Turkish Journal of Veterinary and Animal Science*, 25, 269-72.*

Glass JW, Fedynich AM, Small MF, Benn SJ (2002). Characteristics of the haemoproteid community in an expanding white-winged dove population. *Journal of Parasitology*, 88(1), 74-8*.

Harrigan K (1981). Bird parasites in aviary and caged birds, Proceedings 55 of Post Graduate Committee in Veterinary Science. University of Sydney, Sydney.

Hellgren O, Waldenstrom J, Bensch S (2004). A new PCR assay for simultaneous studies on *Leucocytozoon*, *Plasmodium*, and *Haemoproteus* from avian blood. *Journal of Parasitology*, 90(4), 797-802.

Holder P, Brown G (1999). The Mosquitoes of New Zealand. *Surveillance*, 26(4), 12-5.

Kilpatrick AM, Lapointe DA, Atkinson CT, Woodworth BL (2005). Effects of chronic avian malaria (*Plasmodium relictum*) infection on reproductive success of Hawaii amakihi (Hemignathus virens). http://findarticles.com/p/articles/mi_qa3793/is_200607/ai_n16629441, downloaded 5/11/07.

Klei TR, De Giusti DL (1975). Seasonal occurrence of *Haemaproteus columbae* Kruse and its vector *Pseudolynchia canariensis*. *Bequaert Journal of Wildlife Diseases.*, 11, 130-4.

MAF (2007). Unwanted Organisms. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, downloaded 6/11/07.

Mackerras MJ, Mackerras IM (1960). The haematozoa of Australian birds. *Journal of Zoology*, 8(2), 226-60.*

McKenna PB (1998). Checklist of helminth and protozoan parasites of birds in New Zealand. *Surveillance*, 25(Special issue), 3-12.

Mushi EZ, Binta MG, Chabo RG, Mathaio M, Ndebele RT (1999). *Haemoproteus columbae* in domestic pigeons in Sebele, Gaborone , Botswana. *Onderstpoort Journal of Veterinary Research*, 66(1), 29-32.

Ozmen O, Halgut M, Yukar BA (2005). *Leucocytozoon marchouxi* in Turkish pigeons. *Turkish Journal of Veterinary and Animal Science*, 29, 1273-8.***

Pierce MA, Cheke AS, Cheke RA (1977) A survey of parasites in the Mascarene Islands, Indian Ocean. *Ibis* 119: 451-461

Pierce MA, Greenwood AGS, K (1997) Pathogenicity of *Leucocytozoon marchoux*i in the pink pigeon. *Veterinary Record* 140

Resende JS, Martins NRS, Jorge MA (2001) An outbreak of malaria by Haemoproteus columbae in pigeons. http://www.scielo.br/scielo.php?pid=S0102-09352001000300015&script=sci_arttext&tlng=en)

Rukhsana Y (2005) Infections of haematozoan parasites found in birds in NWFP (Pakistan). *Pakistan Journal of Biological Sciences* 8: 1-5

Scullion FT, Scullion MG (2007). Pathologic findings in racing pigeons (*Columba livia domestica*) with "young bird sickness". *Journal of Avian Medicine and Surgery*, 21(1), 1-7.

Shutler D, Ankey CD, Mullie A (1999). Effects of the blood parasite *Leucocytozoon simondion* growth rates of anatid ducklings. *Canadian Journal of Zoology 77(10), 1573-8*, 77(10), 1573-8.*

Swinnerton KJ, Pierce MA, Greenwood AG, Chapman RE, Jones CG (2005). Prevalence of *Leucocytozoon marchouxi* in the endangered Pink Pigeon. *Ibis*, 147(4), 725-37.

Tomkins DM, Gleeson DM (2006). Relationship between avian malaria distribution and an exotic invasive mosquito in New Zealand. *Journal of the Royal Society of New Zealand*, 36(2), 51-62.

Valkiunas G, Sehgal RNM, Iezhou TA, Smith TB (2005). Further observations on blood parasites of birds in Uganda. *Journal of Wildlife Diseases*, 41(3), 580-7.*

18. Protozoa (Other than Haematozoa)

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

The section covers protozoan parasites relevant to the importation of pigeons from Australia to New Zealand. Blood parasites have been considered in Section 17. Relevant genera include *Eimeria*, *Sarcocystis, Trichomonas, Cryptosporidium* and *Giardia*.

18.1.2. OIE List

There are no protozoal diseases of birds listed by the OIE.

18.1.3. New Zealand status

Thirty species of protozoa from birds have been reported in New Zealand (McKenna 1998). *E. labbeana, T. gallinae,* and *Sarcocystis* spp. have been recorded in pigeons. None of these are from genera included in the list of unwanted organisms.

18.1.4. Epidemiology

Many enteric protozoa are present in Australia (Biosecurity Australia, 2002). *E. labbeana* is the most important coccidian of pigeons, and the only species recorded in Australia (Morrow 1986) and New Zealand (McKenna 1998). Control of coccidian infections is by development of immunity through continuous low grade infection (Tudor 1991).

At least 30 species of intestinal protozoal parasites already occur in birds in New Zealand (McKenna 1998). Many of these parasites do not cause significant disease.

Sarcocystis spp. are two-host species with the sexual cycle of the parasite generally occurring in the gastrointestinal tract of a carnivorous primary host and the cyst form of the parasite occurring in the muscles or organs of the secondary (prey) host (Bermudez 2003). Sarcocystis therefore cannot be established unless an infected pigeon is eaten by a competent primary host (carnivorous) animal. Since the likelihood of a valuable imported bird being eaten by a competent carnivore is remote, the likelihood of new species of sarcocysts establishing as a result of importation of pigeons is considered to be negligible.

18.1.5. Conclusion

Intestinal protozoal parasites are not regarded as potential hazards in pigeons imported from Australia since the relevant organisms are already in New Zealand or do not cause diseases of any significance.

References

Bermudez AJ (2003). Miscellaneous and sporadic protozoal infections. In Diseases of poultry. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 1010-23. Iowa State Press.

Biosecurity Australia (2002). Generic Import Risk Analysis (IRA) for African Crowned Cranes into Zoos in Australia. Technical Issues Paper. http://www.daffa.gov.au/__data/assets/word_doc/0019/11890/2002-48a.doc, downloaded 5/11/07.

McKenna PB (1998). Checklist of helminth and protozoan parasites of birds in New Zealand. *Surveillance*, 25(Special issue), 3-12.

Morrow CJ (1986). Host-parasite list for pigeon and quail, In Proceedings No. 92, Post Graduate Committee in Veterinary Science. University of Sydney, Sydney, pp. 698.

Tudor DC (1991). Coccidiosis. *Pigeon Health and Diseases*. Pp. 171-5. Iowa State University Press, Ames.

19. Exotic Fungi And Yeasts

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

Fungi and yeasts considered are

- Dermatophytes - *Microsporum* spp. and *Trichophyton* spp.
- *Histoplasma* spp.
- *Cryptococcus* spp.
- *Candida* spp.
- *Aspergillus* spp.
- Zygomycetes

1. *Absidia* spp.
2. *Mortierella* spp.
3. *Mucor* spp.
4. *Rhizopus* spp.

19.1.2. OIE List

Histoplasma farciminosum is listed by the OIE

19.1.3. New Zealand Status

Histoplasma farciminosum is listed in the register of unwanted organisms.

19.1.4. Epidemiology

The epidemiology of fungi and yeasts has been fully reviewed in relation to birds in the risk analysis for the importation of passerine hatching eggs (Simpson 2006).

19.1.5. Hazard identification conclusion

Simpson's (2006) conclusions that none of the above fungi and yeasts are hazards in birds, can be extended to pigeons sourced from Australia. Therefore these fungi and yeasts are not considered to be potential hazards in the commodity.

References

Simpson BS (2006). Import Risk Analysis : Passerine Hatching Eggs from the European Union, Ministry of Agriculture and Forestry, Wellington.

20. Internal Parasites

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

The section covers all Nematodes, Trematodes and Cestodes.

20.1.2. OIE List

There are no avian helminths listed by the OIE

20.1.3. New Zealand Status

Twelve nematodes (either species or genera) are listed in the register of unwanted organisms. Sixty one species of nematodes have been identified from birds in New Zealand. Three of these (*Capillaria obsignata*, *Ornithostrongylus quadriradiatus* and *Pelicitus* sp.) have been identified in rock pigeons (McKenna 1998). *Ascaridia columbae* was recorded in New Zealand in 2001 (McKenna 2001).

Four trematodes (either species or genera) are listed in the register of unwanted organisms. Thirty one species of trematodes have been identified from birds in New Zealand (McKenna 1998). *Echinostomum revolutum* from the rock pigeon and *Liperosomum megacotylosum* from the kiwi are the only trematodes from terrestrial birds. Others have come from aquatic birds.

Five species or genera of cestodes are listed in the register of unwanted organisms.

Twenty three species of cestodes have been identified from birds in New Zealand (McKenna 1998). None of these were from columbiform birds.

20.1.4. Epidemiology

A scan of literature databases reveals a number of nematode parasites of species and genera not recorded in New Zealand but present in pigeons in other parts of the world.

Nematodes from 11 families have been reported to infect pigeons (Tudor 1991). Only *Ascaridia columbae* and *Capillaria caudinflata* are recorded as being “more common”. The remainder are regarded as “occasional” or “incidental”. *Ascaridia columbae* is identified as the most common nematode parasite of doves in two recent surveys in the United States (Bean et al 2005; Glass et al 2002). An Australian host-parasite list reveals a close correlation between nematodes in Australian pigeons and those in New Zealand pigeons (Morrow 1986). This is not surprising given the unrestricted imports of pigeons from Australia in the recent past, and illustrates the incompleteness of New Zealand’s host-parasite records.

The epidemiology of nematode parasites of most birds is not well understood, but their importance in impairing racing performance has lead to a better understanding of nematodes in pigeons than in many species. *Ornithostrongylus quadriradiatus* and *Dispharynx nasuta* have been identified as the most serious roundworm pathogens of pigeons (Tudor 1991).

Trematodes invariably have indirect lifecycles. As the intermediate hosts, where these are known, are usually molluscs or tadpoles, it is not surprising that the majority of trematodes are recorded from aquatic birds (McDougald 2003). A scan of literature reveals a large

number of trematode parasites of species and genera not recorded in New Zealand but present in pigeons in the rest of the world (Tudor 1991). Records for trematodes in pigeons in Australia are as scant as those in New Zealand. Neither the epidemiology of most trematode parasites of birds nor their effect on bird health is well described. Varying degrees of host specificity are apparent.

Cestodes of birds have indirect lifecycles with snails, slugs, moths and especially weevils found in seeds and grain acting as vectors (Marshall 1999; McDougald 2003; Taylor et al 2007). Records in the scientific literature for cestodes in Australian pigeons are as scant as those in New Zealand. However, it has been stated in non-scientific literature that "tapeworms are a major problem in all parts of Australia" (Marshall 1999). Rations fed to pigeons must be free from potential vectors. Most cestodes are host specific for a single or a few closely related birds.

20.1.5. Hazard identification conclusion

Although pigeons have been imported recently from Australia, internal parasites must be considered a potential hazard in pigeons imported from Australia

20.2. RISK ASSESSMENT

20.2.1. Entry assessment

Given the frequent importation of pigeons from Australia until 1996, it is likely that any significant internal parasites of pigeons would likely have been transported to New Zealand. One of the most serious parasites (*Ornithostrongylus quadriradiatus)* is already present in New Zealand, and no record of the other (*Dispharynx nasuta)* occurring in Australian pigeons could be located. However, given the uncertainties in this area the entry assessment is considered to be non-negligible.

20.2.2. Exposure assessment

Imported pigeons will be introduced into pigeon lofts, and mixed in racing baskets in which they are in close contact with New Zealand pigeons. The exposure assessment for nematode parasites under these circumstances is non-negligible. Contact with intermediate hosts will be possible, so the exposure assessment for cestodes and trematodes is also non-negligible.

20.2.3. Consequence assessment

The consequences of importation of any internal parasites with pigeons from Australia are hard to assess as these birds have been imported freely until recently without any apparent adverse consequences for New Zealand birds. In view of the lack of exact information consequences are assessed to be low but non-negligible.

There are no public health consequences as a result of internal parasites that may be carried by pigeons imported from Australia.

20.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for internal parasites is non-negligible and they are classified as hazards in the commodity. Therefore risk management measures can be justified.

20.3. RISK MANAGEMENT

20.3.1. Options

The following points should be considered when drafting measures to prevent the introduction of parasites in the commodity:

- Since pigeons were imported into New Zealand from a number of countries for almost 150 years up to 1996, it could be considered that internal parasites associated with pigeons in Australia are likely to have already been introduced into this country.
- Anthelmintic treatment of the birds to be imported could be used to eliminate internal parasites. Treatment could be for nematodes (ivermectin or other) and cestodes and trematodes (praziquantel).
- Cleaning of the pre-export quarantine premises prior to entry of the birds would ensure that they were not re-infected. Measures to prevent contact with intermediate hosts of cestodes and trematodes could be implemented.
- The testing of faecal samples prior to export would ensure that adult worms were not present at the time of export. Two tests, one on entry into quarantine and one a week prior to shipment could be used to reduce the risk of importing parasites. Test procedures could include flotation, sedimentation and larval culture method in order to most effectively diagnose all forms of internal parasites.

Available options in ascending order of stringency are:

Option 1.

Birds could be imported without restrictions, based on the assessment that since importation of pigeons was permitted for 150 years up to 1996, internal parasites of pigeons in Australia are likely to have already been introduced into this country.

Option 2.

Within 7 days of export, birds could be treated with anthelmintics effective against a broad range of internal parasites.

Option 3.

Quarantine, treatment, testing.

i. Birds for export could be quarantined for a period of 3 weeks immediately before shipment; and
ii. The pre-export quarantine premises could have smooth, painted walls and impermeable floors and be regularly cleaned to remove all faeces and bedding materials. The premises could be cleaned to a standard that ensures that intermediate hosts of cestodes and trematodes are excluded and disinfected with a disinfectant effective against nematode eggs prior to birds entering quarantine; and
iii. Birds could be subjected to anthelmintic treatment immediately on entry to quarantine, and again 7-10 days later. The anthelmintic(s) used could be selected for their efficacy against a broad range of parasites; and
iv. Faeces samples from the birds could be examined for parasite eggs by flotation, sedimentation and larval culture methods and larvae, 5-7 days after the last treatment. If faeces samples are negative the birds could be accepted as suitable for importation. If faeces samples are not negative treatment could be repeated

until tests on faeces samples are negative. If necessary different anthelmintics could be used.

References

References marked * were seen as summaries in electronic databases.

Bean DL, Rojas-Flores GW, Foster GW, Kinsella JM, Forrester DJ (2005). Parasitic helmiths of Eurasian collared-doves (*Streptopelia decaocto*) from Florida. *Journal of Parasitology*, 91(1), 184-7.*

Glass JW, Feydenich AM, Small MF, Benn SJ (2002). Helminth community structure in expanding white winged dove (*Zenaida asiatica asiatica*) population. *Journal of Wildlife Diseases*, 38(1), 68-74.*

Marshall R (1999). Worms and worming the racing pigeon. *Pigeon medicine handbook.* *http://www.sahpa.asn.au/worms.htm*, downloaded 5/11/07

McDougald LR (2003). Cestodes and Trematodes. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition.* Pp. 961-71. Iowa State Press.

McKenna PB (1998). Checklist of helminth and protozoan parasites of birds in New Zealand. *Surveillance*, 25(Special issue), 3-12.

McKenna PB (2001). Register of new host parasite relationships. *Surveillance*, 28(1), 4-5.

Morrow CJ (1986). Host-parasite list for pigeon and quail, In Proceedings No. 92, Post Graduate Committee in Veterinary Science. University of Sydney, Sydney, pp. 698.

Taylor MA, Coop RL, Wall RL (2007). Parasites of exotics. Pigeons. *Veterinary Parasitology, 3rd edition,*. Pp. 652-9. Blackwell Publishing, Oxford.

Tudor DC (1991). Worm infestations. *Pigeon Health and Disease*. Pp. 157-70. Iowa State University Press, Ames.

21. External Parasites

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

The parasites considered in this section include mites (including feather mites and nasal mites), fleas, lice, ticks, and louse flies

21.1.2. OIE List

There are no avian mites, fleas, ticks, lice, or louse flies listed by the OIE.

21.1.3. New Zealand status

Seven species of mites are included in the register of unwanted organisms. A literature search failed to reveal reports of any of those mites from birds. Nineteen species of mite, nine species of nasal mite and 82 species of feather mite have been reported in New Zealand (Bishop and Heath 1998).

The following parasites of *Columbia livia* have been recorded in New Zealand (McKenna 2007).

Bonomiella columbae	pale feather eating louse
Campanulotes bidentatus compar	small pigeon louse
Colpocephalum turbinatum	pigeon louse
Columbicola columbae columbae	slender pigeon louse
Goniocotes gallinae	poultry fluff louse
Hohorstiella lata	pigeon body louse
Falculifer rostratus	feather mite
Megninia cubitalis	feather mite
Pterophagus strictus	feather mite
Tinaminyssus melloi	nasal mite
Nosopsyllus fasciatus	flea
Parapsyllus longicornis	flea (from Cape pigeon)

In addition, a further five feather mites have been reported from the New Zealand pigeon (*Hemiphaga novoseelandiae*). Two species of mite (*Dermanyssus gallinae* and *Picnemidocoptes laevis*) have been recorded from "columbiform species" in New Zealand (McKenna 1998).

Thirty species of flea, from ten genera, have been identified from birds in New Zealand. Two species of flea have been recorded in columbiform birds in New Zealand (McKenna 1998).

Five genera of ticks (*Amblyomma* spp., *Boophilus* spp., *Dermacentor* spp., *Ixodes* spp., and *Rhipicephalus* spp.) are included in the register of unwanted organisms.

Twelve species of tick have been reported from birds in New Zealand (McKenna 1998). Nine *Ixodes* species and *Ornithodoris capensis* have come almost exclusively from birds

from aquatic (mainly marine) habitats. The exceptions are findings of *Ixodes anatis* and *Ixodes eudyptis* on North Island brown kiwi, *Ixodes auritulus* from western weka and South Island kaka, and *Haemaphysalis longicornis* from the domestic fowl (McKenna 1998).

21.1.4. Epidemiology

The species *Dermanyssus gallinae, Ornithonyssus sylvarum* and *Falculifer rostratus* that commonly infect pigeons, and five species that occasionally infect pigeons have been recorded in New Zealand (McKenna 1998; McKenna 2007). A search of the Australian records of external parasites from columbiform birds (Morrow 1986a) showed that the list of external parasites that occur in Australia is similar to those that occur in New Zealand. However, *Echidnophaga, gallinacea* (the stick tight flea of poultry), and *Pseudolynchia canariensis* (the pigeon louse fly) occur in Australia but not in New Zealand.

Tudor (1991) indicates that only two (including *Echidnophaga gallinacea*) of the nine species of flea recorded as infecting pigeons are of occasional importance, the other seven being incidental findings only. *Echidnophaga gallinacea* occurs in Australia (Morrow 1986b). This flea is unique amongst poultry fleas in that adults become sessile and usually remain attached for days or weeks and cause feather irritation and blood loss, especially in young birds (Morrow 1986b). Arends (2003) considered only three poultry fleas worthy of note; *Echidnophaga gallinacea* was the only one causing adverse effects. Of the other two, *Ceratophyllus gallinae* occurs in New Zealand (McKenna 1998) and *Ceratophyllus niger* is not recorded as being present in Australia (CSIRO 2005a).

Avian hippoboscids (louse flies) are obligate bloodsucking parasites. They are, commonly, dorso-ventrally or laterally compressed and have specialised claws enabling them to move through and cling to plumage of their hosts. Some species are winged, others are wingless, and some lose their wings once the adults have found their hosts. The most significant hippoboscid fly present in Australia but absent from New Zealand is the pigeon louse fly *Pseudolynchia canariensis.* The adults live for about 45 days in the feathers of a pigeon, or more rarely a dove. The female lays four or five fully developed larvae, which pupate immediately after being laid and take 29-31 days before emerging as adults and a blood meal before reproducing (Tudor 1991).

Australian records show that there are 28 species of ixodid ticks (CSIRO 2005b) and eight species of argasid ticks (CSIRO 2005c). None of the ixodid ticks are primary parasites of terrestrial birds (except *Haemaphysalis longicornis*). Amongst the argasid ticks, only *Argas persicus* and the closely related *Argas robertsi* commonly infest terrestrial birds, including pigeons. Only larval *Argas persicus* attach to their hosts for periods up five days; nymphs and adults hide in cracks and crevices and attack their host and feed for periods of about 2 hours, mainly at night during warm dry periods (Morrow 1986b). In temperate localities nymphs remain inactive in crevices for long periods.

21.1.5. Conclusion

A large number of mites, including most of the important pathogenic parasites have been recorded in New Zealand. However, because there are very large numbers of species of mites that cannot be considered individually, mites are considered to be a potential hazard in the commodity.

As the significant lice of pigeons present in Australia are already present in New Zealand, these are not considered potential hazards.

As terrestrial birds are not primary hosts of ixodid ticks that occur in Australia, these ticks are not considered to be a potential hazard.

The external parasites that are considered potential hazards for the importation of pigeons from Australia are *Pseudolynchia canariensis, Echidnophaga gallinae, Argas persicus* and *Argas robertsi*.

21.2. RISK ASSESSMENT

21.2.1. Entry assessment

Pseudolynchia canariensis, *Echidnophaga gallinae* and various species of feather mites are present in Australia and could be imported on pigeons. *Argas persicus* and *Argas robertsi* nymphs and adults do not live on the host but reside in crevices and suitable hiding places. Since they are only on their hosts for short periods they are unlikely to be imported on birds, but larvae remain attached to the host for several days while feeding. Therefore, the likelihood that parasites could be imported from Australia is non-negligible.

21.2.2. Exposure assessment

If introduced into New Zealand the parasites could establish in pigeon lofts and spread by contact during pigeon races and transfer of birds from one loft to another. As they could also spread to feral and wild pigeons, the likelihood of exposure is non-negligible.

21.2.3. Consequence assessment

Feather mites have little economic impact in the poultry industry (Arends 2003), but the damage to feathers, resulting from some species of feather mites, is of concern in cage and aviary birds, particularly for owners of show-birds (Baker 1996; Greve 1996). Reports of feather mites having negative effects on survival or breeding capabilities of birds, including pigeons have not been found. These impacts are considered to be negligible.

The consequence of the introduction of *Pseudolynchia canariensis* include the capability to transmit *Haemoproteus columbae* (Sol et al 2000) and to cause blood loss in heavy infestations. These consequences are non-negligible.

Echidnophaga gallinae can cause blood loss, anaemia and blindness when they attach around the eyes, especially in young birds (Zajac and Conboy 2006b).

Argasid ticks, in addition to being the vector of *Borrelia anserina*, cause paralysis (Zajac and Conboy 2006a)

None of the parasites of birds are known to be parasites of humans. Therefore, the consequences for human health considered to be negligible,

The consequences for pigeons and other birds, of introducing parasites, are considered to be non-negligible.

21.2.4. Risk estimation

Since, entry, exposure and consequence assessments are all non-negligible, the risk estimate for external parasites is non-negligible and they are classified as hazards in the commodity. Therefore risk management measures can be justified.

21.3. RISK MANAGEMENT

21.3.1. Options.

The following points should be considered when drafting options for risk management:

- Since pigeons were imported into New Zealand from a number of countries for almost 150 years up to 1996, it could be considered likely that any external parasites that may be associated with pigeons in Australia have already been introduced into this country.
- Since there are hundreds of different species of mites, lice and ticks that cannot be considered individually, it is important to implement general methods that are applicable to preventing the importation of all external parasites.
- Treatment of the birds to be exported could be used to eliminate external parasites.
- Cleaning and treatment with insecticides of the pre-export quarantine premises prior to entry of the birds should be carried out to ensure that they are not infested with any parasites that have life cycle stages off the birds.
- Examination of birds for parasites should be done visually with the aid of a jeweller's headset and suitable lighting. In addition a feather ruffling technique using a suitable insecticide dust or aerosol, or anaesthetic and a dissecting microscope to examine the recovered debris for parasites, could be used. Methods of examining birds for parasites have been described in detail by (Clayton and Walther 1997).

Available options in order of ascending stringency are:

Option 1.

Birds could be imported without restrictions, based on the assessment that since importation of pigeons was permitted for 150 years up to 1996, external parasites of pigeons in Australia are likely to have already been introduced into this country.

Option 2.

Birds to be imported could be treated with a suitable insecticide within 7 days of shipment.

Option 3.

Quarantine, treatment, testing.

i. Birds for export could be quarantined for at least the 3 weeks immediately before shipment; and
i. Quarantine premises could have smooth painted walls and impermeable floors that do not provide shelter places for insects. Floors, walls and cages could be steam cleaned and sprayed with an insecticide effective against all stages of the relevant parasites prior to birds entering quarantine; and
ii. Birds to be imported could be subjected to treatment effective against external parasites immediately on entry to, and during pre-export quarantine. The compounds used should be effective against ticks, fleas, lice, louse flies and mites; and
iii. Birds for importation could be thoroughly inspected immediately prior to export to ensure that they are free from parasites. Inspections could be carried out visually using a jeweller's headset and suitable lighting as well a feather ruffling technique using a suitable insecticide dust or aerosol or anaesthetic and examination of the recovered debris with a dissecting microscope;

References

References marked * were seen as summaries in electronic databases.

Arends JJ (2003). External parasites and poultry pests. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds). *Diseases of Poultry, 11th edition*. Pp. 905-30. Iowa State Press.

Baker JR (1996). Survey of feather diseases of exhibition budgerigars in the United Kingdom. *Veterinary Record*, 139(24), 590-4.

Bishop DM, Heath ACG (1998). Checklist of ectoparasites of birds in New Zealand. *Surveillance*, 25(Special Issue), 13-31.

Clayton DH, Walther A, W (1997). Collection and quantification of arthropod parasites of birds. In. In: Clayton DH, Moore J (eds). *Host-parasite evolution: general principles and avian models*. Pp. 419-40. Oxford University Press, Oxford.http://www.bruno-walther.de/docs/ClaytonWaltherAppendixCsinglespaced.pdf, downloaded 6/11/07.

CSIRO (2005a). Entomology. *http://www.ento.csiro.au/aicn/name_s/b_749.htm*, downloaded 6/11/07.

CSIRO (2005b). Entomology. *http://www.ento.csiro.au/aicn/system/ixodidae.htm*, downloaded 6/11/07.

CSIRO (2005c). Entomology. *http://www.ento.csiro.au/aicn/system/argasida.htm*, downloaded 6/11/07.

Greve JH (1996). Parasites of the skin. In: Rosskopf W, Woerpel R (eds). *Diseases of cage and aviary birds*. Pp. 623-6. Williams and Wilkins, Baltimore.

McKenna PB (1998). Checklist of helminth and protozoan parasites of birds in New Zealand. *Surveillance*, 25(Special issue), 3-12.

McKenna PB (2007). Personal communication.

Morrow CJ (1986a). Host-parasite list for pigeon and quail, In Proceedings No. 92, Post Graduate Committee in Veterinary Science. University of Sydney, Sydney, pp. 698.

Morrow CJ (1986b). Poultry parasites, In Proceedings No. 92, post graduate committee in veterinary science. University of Sydney, Sydney, pp. 683.

Sol D, Jovani R, Torres J (2000). Geographical variation in blood parasites in feral pigeons: the role of vectors. *Ecography*, 23(3), 307-14.

Tudor DC (1991). External parasite infestations. *Pigeon Health and Disease*,. Pp. 194-213. Iowa State University Press, Ames.

Zajac AM, Conboy GA (2006a). Veterinary Clinical ParasitologyPp. 222. Blackwell Publishing, Oxford.

Zajac AM, Conboy GA (2006b). Veterinary Clinical ParasitologyPp. 246. Blackwell Publishing, Oxford.