Import Risk Analysis: Psittaciformes

DRAFT FOR PUBLIC CONSULTATION

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Ministry for Primary Industries
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6011
New Zealand
Tel: 64 4 894 0100
Fax: 64 4 894 0731

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Contributors to this risk analysis

1. Author

Helen Smith	Senior Adviser, Risk Analysis (Animals and Aquatic)	MPI, Wellington
Stephen Cobb	Principal Adviser, Risk Analysis (Animals and Aquatic)	MPI, Wellington

2. Internal Peer Review

Lucy Johnston	Senior Adviser, Animal Imports	MPI, Wellington
Erik Van Eyndhoven	Principal Adviser, Conservation	MPI, Wellington
Stuart C. MacDiarmid	Prinicpal Adviser, Risk Analysis, International Policy	MPI, Wellington
Kylee Walker	Incursion Investigator, Surveillance and Incursion Investigation (Animals and Marine)	MPI, Wellington

3. External Scientific Review

Simon Girling	Head of Veterinary Services, Royal Zoological Society of Scotland.	Edinburgh, Scotland
Brett Gartrell	Associate Professor in Avian and Wildlife Health, Massey University	Palmerston North

1. Executive summary

Live psittacine imports from Australia and England have previously been permitted into New Zealand. In 1997 these imports ceased although aviculturists and individual pet owners have continued to submit requests for psittacine imports and bird smuggling remains an ongoing concern. Illegal importations have no sanitary controls and therefore represent an unmanaged biosecurity risk to New Zealand.

Historically, large numbers of exotic birds have been imported into New Zealand with little consideration of biosecurity issues. These importations have resulted in the establishment of free-living populations, as well as many other species kept in captivity. It is likely that some of the potential hazards considered in this risk analysis already entered New Zealand with these imports and that the low level of disease surveillance allows them to remain undetected.

This import risk analysis examines the biosecurity risks posed by infectious or parasitic agents when importing live psittacine birds from any country. Consideration of a list of 194 preliminary hazards (Table 1) has identified 24 organisms or diseases of concern which require further consideration. Of these, 16 have been identified as potential hazards and subject to a risk assessment. As a result of this, a non-negligible risk is identified with the following hazards:

- Newcastle disease virus and avian paramyxoviruses 2 and 5
- Highly pathogenic avian influenza viruses
- Psittacine herpesvirus
- Psittacinepox virus
- Avian reovirus
- West Nile virus
- Avian bornavirus
- *Bordetella avium*
- *Coxiella burnetii*
- *Salmonella* Gallinarum-Pullorum and *S. arizonae*
- *Plasmodium* spp. and *Leukocytozoon* spp.
- Helminths
- Ectoparasites

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

2. Introduction

The order Psittaciformes includes parrot species as well as budgerigars, cockatoos, corellas, cockatiels, conures, lories, lorikeets, galahs, guaiaberos, racket-tails, rosellas, bluebonnets, ringnecks, parakeets, lovebirds, and macaws (Gill and Donsker 2011).

Live psittacine imports from Australia and England have previously been permitted into New Zealand. In 1997 these imports ceased as it was recognised that knowledge of psittacine bird diseases was incomplete at that time and there was a lack of validated methods for testing imported birds.

Aviculturists and individual pet owners have continued to submit requests for psittacine imports and bird smuggling remains an ongoing concern. Illegal importations have no sanitary controls and therefore represent an unmanaged biosecurity risk. Anecdotal evidence suggests that allowing the importation of psittacine birds will decrease smuggling (AQIS 1999) although others have argued that allowing a greater diversity of exotic parrot species into New Zealand may increase opportunities for illegal smuggling to thrive (Gartrell 2012).

Risk analyses already exist for the importation of domestic budgerigars (*Melopsittacus undulatus*) from the United Kingdom and for hatching eggs of birds of the Order Psittaciformes from all countries.

New Zealand has a number of endemic psittacine species and several of these are considered to be either nationally critical or nationally endangered. Very little information is available on the susceptibility of New Zealand parrots to many of the diseases considered in this risk analysis and it is necessary to extrapolate using information from other parrot species. As they have evolved in relative isolation, it is argued that the New Zealand parrots could be particularly susceptible to new disease agents (Jackson et al 2000).

Historically, large numbers of exotic birds were imported into New Zealand with little consideration of biosecurity issues. These importations have resulted in the establishment of free-living populations of sulphur crested cockatoos, galahs, and eastern and crimson rosellas (Miskelly et al 2008), as well as many other species kept in captivity. It is likely that some of the potential hazards considered in this risk analysis already entered New Zealand with these imports and that the low level of disease surveillance allows them to remain undetected.

3. Scope

This qualitative analysis examines the biosecurity risks associated with the importation of psittacine birds from any country. The risk analysis is limited to infectious and parasitic disease risks and does not consider genetic diseases or ecological impacts resulting from species naturalisation.

The Ministry for Primary Industries (MPI) has the flexibility to modify any import health standard (IHS) based on this risk analysis if future events make this appropriate.

4. Commodity definition

The commodity considered in this risk analysis is defined as healthy live birds of any species of the order Psittaciformes. The birds to be imported must have been born in, and

continuously resident in, an aviary or zoological garden. The commodity does not include wild captured birds.

To be eligible for import, the Biosecurity Act 1993 requires MPI to be satisfied that the imported birds do not harbour potentially harmful organisms. The birds must be certified by an Official Veterinarian on the day of travel to be showing no clinical signs of infectious or parasitic diseases. Birds for import must also be certified as free from obvious contamination with weeds and weed seeds.

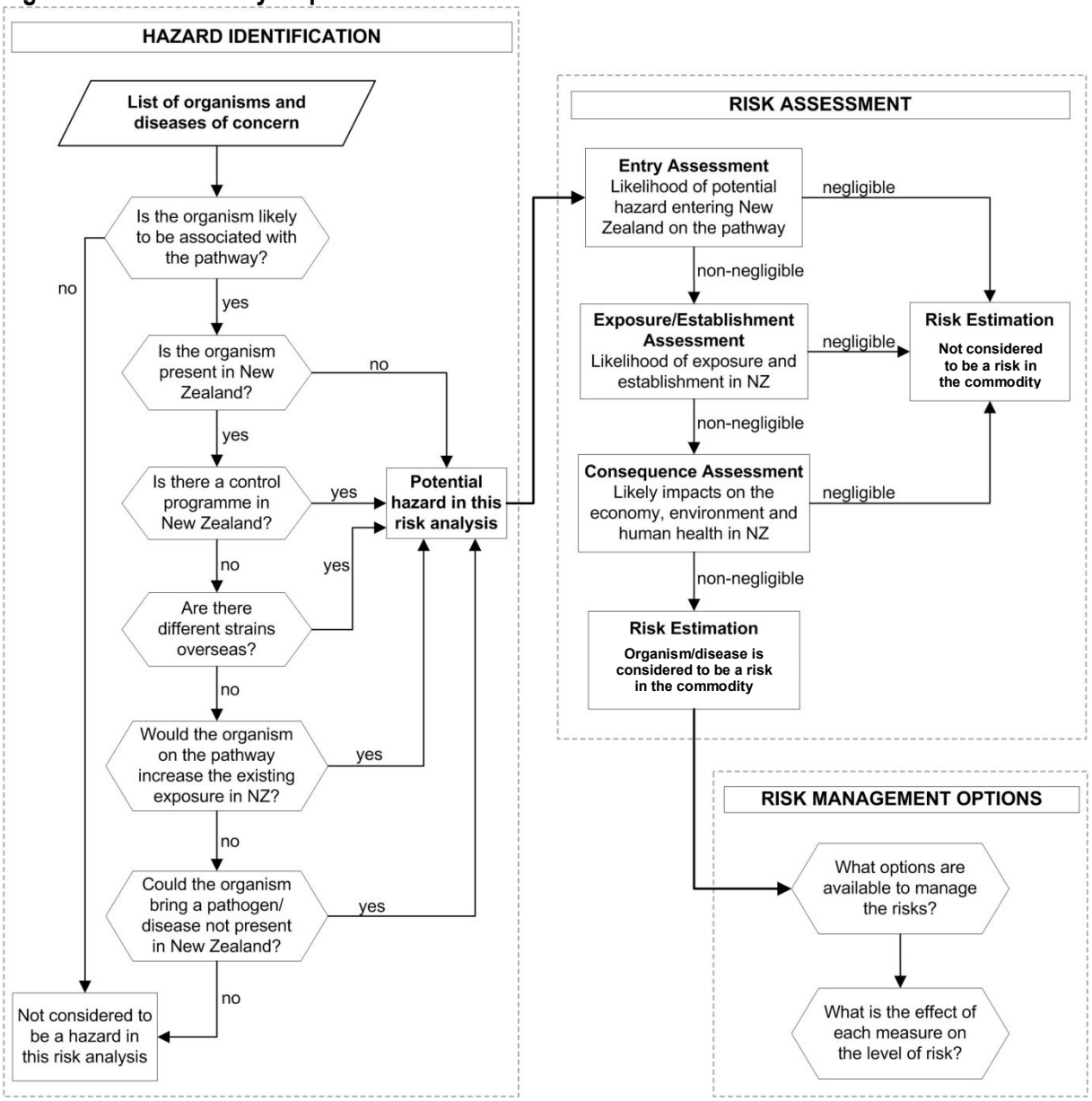
While the commodity definition and the contents of this risk analysis consider the entire Order Psittaciformes, this does not subvert the requirement for approval by the Environmental Protection Authority (EPA) of the importation of any "new organism", as defined in the Hazardous Substances and New Organisms Act 1996 (HSNO).

5. Risk analysis methodology

The methodology used in this risk analysis follows the guidelines as described in *Biosecurity New Zealand Risk Analysis Procedures- Version 1* (MAFBNZ 2006) and the *Terrestrial Animal Health Code* of the World Organisation for Animal Health (OIE 2011). The procedures provide a framework which adheres to the requirements set out under the World Trade Organization's Agreement on the Application of Sanitary and Phytosanitary (SPS) measures, 1995 and of the Biosecurity Act, 1993.

The risk analysis process is summarised in Figure 1.

Figure 1. The risk analysis process



5.1. PRELIMINARY HAZARD LIST

The first step in the risk analysis process is hazard identification. This requires consideration of all organisms of potential concern (i.e. those that could be transmitted in live psittacines and could infect New Zealand's domestic, feral, or wild animals, or humans) and involves assembling a list of organisms that may be associated with the commodity.

In this risk analysis, the preliminary hazard list was compiled from the diseases listed in the risk analyses for "Budgerigars (*Melopsittacus undulatus*) from the United Kingdom 2009" and "Psittacine Hatching Eggs 2010" as well as diseases reported in CAB biological abstracts and in the following textbooks:

- *Virus Infections of Birds*, edited by McFerran JB, and McNulty MS. Elsevier, Amsterdam, published 1993.

- *Avian Viruses: Function and Control,* edited by BW Ritchie. Wingers Publishing, Lake Worth, Florida, published 1995.
- *Avian Medicine: Principles and Application*, edited by BW Ritchie, GJ Harrison, and LR Harrison. Wingers Publishing, Lake Worth, Florida, published 1994.
- *Diseases of Cage and Aviary Birds*, edited by W Rosskopf and R Woerpel. Williams and Wilkins, Baltimore, published 1996.
- *Diseases of Poultry* (12th ed), edited by YM Saif, Blackwell Publishing, Ames, Iowa, published 2008.

Where more extensive epidemiological information on a preliminary hazard is available from other avian species, or organisms very closely related to a preliminary hazard, this information is used in assessing the likely behaviour of the organism in psittacines. Although it is expected that many organisms will have limited host ranges within Psittaciformes, species-specificity of pathogens is not proposed unless there are multiple reports of the organisms from only one species or one genus within the Psittaciformes. The organisms identified in the preliminary hazard list are shown in Table 1.

Table 1. Preliminary hazard list.

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
BACTERIA					
Actinomyces spp.	Yes	Present	No	No	Young 2000
Arcanobacterium pyogenes	No	Present	No	No	Young 2000
Aerococcus spp.	Yes	Present	No	No	Young 2000
Aeromonas spp.	Yes	Present	No	No	Young 2000
Alcaligenes spp.	Yes	Present	No	No	Young 2000
Bordetella avium (Turkey coryza, BART)	Yes	Exotic	Yes	No	Young 2000; Tüerkyilmaz et al 2009; Clubb et al 1994
B. bronchiseptica, *B. parapertussis*, *B. pertussis* are present in NZ					
Alcaligenaceae (*Pelistega* spp.)	No	Exotic	No	No	
Aegyptianella spp.	Yes	Exotic	No	No	MAF 2011
Argas tick vector is exotic to New Zealand					
Bacillus spp.	Yes	Present	No	No	Young 2000
Bacteroides spp.	Yes	Present	No	No	Young 2000
Brachyspira spp. (Avian intestinal spirochaetosis)	No	Some species	No	No	
Campylobacteraceae (*Arcobacter* spp.)	No	Present	No	No	
Campylobacteraceae (*Campylobacter* spp.)	Yes	Present	No	No	Young 2000

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
BACTERIA (continued)					
Chlamydia psittaci (Avian chlamydiosis, psittacosis, parrot fever)	Yes	Some species	Yes	Yes	Kuo et al 2010
Clostridium botulinum	No	Present	No	No	Young 2000
Clostridium colinum (Ulcerative enteritis, quail disease)	Yes	Present	No	No	
Clostridium perfringens type A and type C (Necrotic enteritis)	Yes	Present	No	No	Young 2000
Corynebacterium spp.	Yes	Present	No	No	Young 2000
Coxiella burnetii (Q fever)	Yes	Exotic	Yes	Yes	Porter 2011; OIE 2010
Desulfovibrionaceae (*Lawsonia intracellularis*)	No	Present	No	No	
Enterobacteriaceae (*Citrobacter* spp.)	Yes	Present	No	No	Young 2000
Enterobacteriaceae (*Enterobacter aerogenes*, *Aerobacter aerogenes*, *Klebsiella aerogenes*)	Yes	Present	No	No	Young 2000
Enterobacteriaceae (*Enterobacter* spp.)	Yes	Present	No	No	Young 2000
Enterobacteriaceae (*Escherichia coli*, Colibacillosis)	Yes	Some strains	No	No	MAF 2011; Young 2000
There is no evidence to suggest that strains of APEC found overseas are any more virulent than the strains encountered in this country					
Enterobacteriaceae (*Hafnia* spp.)	Yes	Present	No	No	Young 2000
Enterobacteriaceae (*Klebsiella* spp.)	Yes	Present	No	No	Young 2000
Enterobacteriaceae (*Plesiomonas* spp.)	No	Present	No	No	Young 2000
Enterobacteriaceae (*Proteus* spp.)	Yes	Present	No	No	Young 2000
Enterobacteriaceae (*Salmonella enterica* including *Salmonella* Gallinarum-Pullorum; Pullorum disease and fowl typhoid)	Yes	Exotic	Yes	Yes	OIE 2008b
Enterobacteriaceae (*Salmonella arizonae*, Arizonosis)	Yes	Exotic	Yes	No	OIE 2008b
Enterobacteriaceae (Salmonella spp., Paratyphoid infections)	Yes	Some species	Yes	No	Young 2000; OIE 2008b
S. choleraesuis, *S.* enteritidis, *S.* typhi, *S.* typhimurium are present in New Zealand					

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
BACTERIA (continued)					
Enterobacteriaceae (*Serratia* spp.)	Yes	Present	No	No	Young 2000
Enterobacteriaceae (*Yersinia* spp.)	Yes	Present	No	No	Young 2000
Enterococcaceae (*Enterococcus* spp.)	Yes	Present	No	No	Young 2000
Erysipelothrix rhusiopathiae	Yes	Present	No	No	Young 2000
Flavobacteriaceae (*Flavobacterium* spp.)	Yes	Present	No	No	
Flavobacteriaceae (*Ornithobacterium rhinotracheale*)	No	Exotic	No	No	
Flavobacteriaceae (*Riemerella anatipestifer*, Exudative septicaemia)	Yes	Some species	Yes	No	Hinz et al 1998; Saif 2008
Francisellaceae (*Francisella tularensis*, Tularaemia)	No	Exotic	No	No	
Fusobacteriaceae (*Streptobacillus moniliformis*)	No	Present	No	No	
Helicobacteraceae (*Helicobacter* spp.)	Yes	Present	No	No	
Lactobacillaceae (*Lactobacillus* spp.)	Yes	Present	No	No	Young 2000
Listeriaceae (*Listeria monocytogenes*)	Yes	Present	No	No	Young 2000
Moraxellaceae (*Acinetobacter* spp.)	Yes	Present	No	No	Young 2000
Moraxellaceae (*Moraxella* spp.)	No	Present	No	No	Young 2000
Mycobacteriaceae (*Mycobacterium avium* subsp. *Avium,* Avian tuberculosis)	Yes	Present	No	No	MAF 2010
There is no evidence that the strains of *M. avium* associated with avian tuberculosis in New Zealand are less virulent than strains found overseas.					
Mycobacteriaceae (*Mycobacterium intracellulare,* Avian tuberculosis)	Yes	Present	No	No	
Mycobacteriaceae (*Mycobacterium avium* subsp. *Paratuberculosis,* Paratuberculosis)	Yes	Present	No	Yes	
Mycobacteriaceae (*Mycobacterium* spp.)	No	Some species	No	No	
Mycobacteriaceae (*Mycobacterium bovis*)	Yes	Present	No	No	Bargalló et al (2009)
Mycobacteriaceae (*Mycobacterium smegmatis)*	No	Present	No	No	

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
BACTERIA (continued)					
Mycobacteriaceae (*Mycobacterium gallisepticum)*	No	Present	No	No	
Mycobacteriaceae (*Mycobacterium tuberculosis)*	Yes	Present	No	No	
Mycobacteriaceae (*Mycobacterium genavense)*	Yes	Exotic	Yes	No	Montali 2008
Mycoplasmataceae *(Mycoplasma gallisepticum,* Avian mycoplasmosis)	Yes	Some strains	Yes	Yes	MAF 2010; Gomes et al 2010
Exotic strains of *Mycoplasma gallisepticum* may be more virulent than those present in this country					
Mycoplasmataceae (*Mycoplasma iowae)*	Yes	Exotic	Yes	No	Gomes et al 2010
Mycoplasmataceae (*Mycoplasma meleagridis)*	No	Exotic	No	No	
Mycoplasmataceae (*Mycoplasma synoviae,* Avian mycoplasmosis)	Yes	Present	No	Yes	
Mycoplasmataceae (*Mycoplasma* spp.)	No	Some species	No	No	
Neisseriaceae (*Neisseria* spp.)	No	Present	No	No	Young 2000
Nocardiaceae (*Nocardia* spp.)	Yes	Present	No	No	Young 2000
Pasteurellaceae (*Actinobacillus* spp.)	Yes	Present	No	No	Young 2000
Pasteurellaceae (*Avibacterium paragallinarum,* Infectious coryza; *Haemophilus paragallinarum*)	No	Exotic	No	No	
Pasteurellaceae (*Avibacterium gallinarum, Pasteurella gallinarum*)	No	Exotic	No	No	
Pasteurellaceae (*Avibacterium paragallinarum*, Infectious coryza)	No	Exotic	No	No	
Pasteurellaceae (*Gallibacterium anatis*, Actinobacillus salpingitidis, avian *Pasteurella haemolytica*-like)	Yes	Present	No	No	
Pasteurellaceae *(Pasteurella gallinarum)*	No	Exotic	No	No	
Pasteurellaceae (*Pasteurella multocida*, Fowl cholera)	Yes	Present	No	Yes	Young 2000
Peptostreptococcaceae (*Peptostreptococcus* spp.)	No	Present	No	No	Young 2000

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
BACTERIA (continued)					
Planococcaceae (*Planococcus* spp.)	No	Exotic	No	No	
Pseudomonadaceae (*Pseudomonas* spp.)	Yes	Present	No	No	Young 2000
Rickettsiaceae (*Rickettsia* spp.)	Yes	Present	No	No	
Spirochaetaceae (*Borrelia anserina*, Avian spirochaetosis)	Yes	Exotic	No	No	MAF 2011
Argas tick vector is exotic to New Zealand					
Spirochaetaceae (*Borrelia burgdorferi*, Lyme disease)	Yes	Exotic	Yes	No	Ehrsam 1977
Spirochaetaceae (*Borrelia* spp.)	No	Exotic	No	No	
Staphylococcaceae (*Staphylococcus* spp., Staphylococcosis)	Yes	Present	No	No	Young 2000
Streptococcaceae (*Lactococcus* spp.)	No	Present	No	No	Young 2000
Streptococcaceae (*Streptococcus* spp., Streptococcosis)	Yes	Present	No	No	Young 2000
Vibrionaceae (V*ibrio* spp.)	No	Present	No	No	Young 2000
VIRUSES					
Adenoviridae (Duck adenovirus A, Egg drop syndrome virus)	No	Present	No	No	Howell 1992, Christensen 1998, Watts 2010, 2011
Adenoviridae (Fowl adenovirus, Group I avian adenoviruses)	Yes	Some species	Yes	No	Saifuddin et al 1992, Saifuddin 1990; Raue et al 2005.
FAdV-1, 8, and 12 are recognised as present in New Zealand. FAdV-2, 3, 4, and 8 have been demonstrated in psittacines					
Adenoviridae (Duck adenovirus B)	No	Exotic	No	No	
Adenoviridae (Goose adenovirus)	No	Exotic	No	No	
Adenoviridae (Turkey adenovirus B)	No	Exotic	No	No	
Adenoviridae (Pigeon adenovirus)	No	Exotic	No	No	
Adenoviridae (Psittacine adenovirus 1)	Yes	Exotic	Yes	No	Lüschow et al 2007
Adenoviridae (Meyer's parrot adenovirus 1)	Yes	Exotic	Yes	No	Wellehan et al 2005; Lüschow et al 2007
Adenoviridae (Raptor adenovirus A)	No	Exotic	No	No	Fauquet et al 2005
The natural host range of adenovirus types is usually confined to one species.					

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
VIRUSES (continued)					
Adenoviridae (Turkey adenovirus A)	No	Exotic	No	No	Gomez-Vilamandos et al 1995; Wellehan et al 2009.; Lüschow et al 2007
There is only one report to date suggestive of the presence of TAdV in psittacines and as the virus was not isolated it is likely that is was a (Group II) psittacine siadenovirus.					
Adenoviridae (Budgerigar adenovirus 1)	Yes	Exotic	Yes	No	Katoh et al 2009; Lüschow et al 2007
Adenoviridae (Great tit adenovirus 1)	No	Exotic	No	No	
Adenoviridae (Psittacine adenovirus 2, Plum headed parakeet adenovirus 1)	Yes	Exotic	Yes	No	Wellehan et al 2009; Benko 2011
Astroviridae (Turkey astrovirus)	No	Exotic	No	No	
Astroviridae (Duck astrovirus)	No	Exotic	No	No	
Astroviridae (Chicken astrovirus, Avian nephritis virus)	No	Exotic	No	No	
Astroviridae (Duck astrovirus, Duck virus hepatitis 2, 3)	No	Exotic	No	Yes	
Birnaviridae (Infectious bursal disease virus, Gumboro disease)	No	Exotic	No	Yes	
Bornaviridae (Borna disease virus)	No	Exotic	No	No	
Bornaviridae (Avian bornavirus, Proventricular dilatation disease; Macaw wasting disease)	Yes	Exotic	Yes	No	Staeheli 2010
Circoviridae (Beak and feather disease virus, Psittacine beak and feather disease)	Yes	Present	No	No	
Circoviridae (Pigeon circovirus)	No	Present	No	No	
Circoviridae (Duck circovirus)	No	Uncertain	No	No	
Circoviridae (Circovirus spp.)	No	Present	No	No	
Circoviridae (Chicken anaemia virus)	No	Present	No	No	
Coronaviridae (Infectious bronchitis virus, Avian infectious bronchitis)	No	Some strains	No	Yes	
Coronaviridae (Turkey Coronavirus, Coronavirus enteritis virus)	No	Exotic	No	No	
Coronaviridae (Psittacine coronavirus)	Yes	Exotic	Yes	No	Culver et al 2008
Coronaviridae (Coronavirus spp.)	No	Some strains	No	No	

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
VIRUSES (continued)					
Flaviviridae (Israel turkey meningoencephalitis virus)	No	Exotic	No	No	
Flaviviridae (West Nile virus)	Yes	Exotic	Yes	Yes	OIE 2008a
Flaviviridae (Kunjin virus)	No	Exotic	No	No	
Flaviviridae (Japanese encephalitis virus)	No	Exotic	No	Yes	
Flaviviridae (Flavivirus spp.)	No	Exotic	No	No	
Hepadnaviridae (Hepatitis B virus, Duck hepatitis B virus)	No	Exotic	No	No	
Hepeviridae (Avian hepatitis E virus, Hepatitis-splenomegaly syndrome)	No	Exotic	No	No	
Herpesviridae (Anatid herpesvirus 1, Duck plague virus, Duck enteritis virus)	No	Exotic	No	No	
Herpesviridae (Gallid herpesvirus 1, Avian infectious laryngotracheitis)	No	Present	No	Yes	
Herpesviridae (Psittacid herpesvirus, Pacheco's disease, Amazon tracheitis virus, Budgerigar herpesvirus)	Yes	Uncertain	Yes	No	Styles et al 2004, 2005; Tomaszewski et al 2006.
Thought to be exotic, however it is associated with internal papillomatosis disease (IPD) which is known to occur in New Zealand.					
Herpesviridae (Gallid herpesvirus 2,3, Marek's disease virus)	No	Some strains	No	Yes	
Herpesviridae (Meleagrid herpesvirus 1, Turkey herpesvirus)	No	Some strains	No	Yes	
Orthomyxoviridae (Highly pathogenic avian influenza (HPAI) virus)	Yes	Exotic	Yes	Yes	Senne et al 1983; Saif et al 2008
HPNAI (HP notifiable AI) encompasses all HPAI					
Orthomyxoviridae (Low pathogenic avian influenza (LPAI) virus)	Yes	Some strains	Yes	Yes	Saif et al 2008
LPNAI (LP notifiable AI) encompasses only H5 and H7 LPAI					
Papillomaviridae (*Fringilla coelebs* Papillomavirus, Chaffinch Papillomavirus)	No	Exotic	No	No	
Papillomaviridae (*Psittacus erithacus timneh* Papillomavirus, Cutaneous papillomas)	Yes	Uncertain	Yes	No	Phalen 2007

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
VIRUSES (continued)					
Paramyxoviridae (Newcastle disease virus, Avian paramyxovirus 1; Avian parainfluenza virus 1)	Yes	Some strains	Yes	Yes	Senne et al 1983
Paramyxoviridae (Avian paramyxovirus 2,3,5)	Yes	Exotic	Yes	No	Stanislawek et al 2001
The presence of APMV-2 in New Zealand cannot be excluded; there is no conclusive evidence of the presence of APMV-3.					
Paramyxoviridae (Avian paramyxovirus 4,6,7,8,9)	No	Some species	No	No	
Paramyxoviridae (Avian metapneumovirus, Turkey rhinotracheitis, swollen head syndrome)	No	Exotic	No	Yes	
Parvoviridae (Goose parvovirus, Derzsy's disease virus)	No	Exotic	No	No	
Picornaviridae (Duck hepatitis A virus, Duck virus hepatitis 1)	No	Exotic	No	Yes	
Picornaviridae (Avian encephalomyelitis virus)	No	Present	No	No	
Picornaviridae (Turkey hepatitis virus)	No	Exotic	No	No	
Polyomaviridae (Budgerigar fledgling disease polyomavirus, Avian polyoma virus)	Yes	Present	No	No	Anonymous 2004
Polyomaviridae (Goose haemorrhagic polyomavirus, Haemorrhagic nephritis enteritis of geese)	No	Exotic	No	No	
Polyomaviridae (Finch polyomavirus)	No	Exotic	No	No	
Polyomaviridae (Crow polyomavirus)	No	Exotic	No	No	
Poxviridae (Psittacinepox virus)	Yes	Uncertain	Yes	No	Senne et al 1983
Poxviridae (Fowlpox virus)	No	Present	No	No	
Poxviridae (Avipoxvirus spp.)	No	Some species	No	No	Fauquet et al 2005
Avipoxvirus has a narrow host range					
Reoviridae (Avian orthoreovirus, Viral arthritis)	Yes	Present	No	No	
Reoviridae (Avian orthoreovirus, Avian reovirus, psittacine reovirus)	Yes	Some strains	Yes	No	Hollmén and Docherty 2007; van den Brand et al 2007; Perpinan et al 2010
Reoviridae (Chicken rotavirus D)	No	Present	No	No	

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
VIRUSES (continued)					
Retroviridae (Avian leukosis virus, Leukosis/sarcoma)	No	Present	No	No	Simova-Curd et al 2010
Retroviridae (Avian sarcoma virus, Leukosis/sarcoma)	No	Present	No	No	
Retroviridae (Lymphoproliferative disease virus)	No	Exotic	No	No	
Retroviridae (Reticulo-endotheliosis virus)	No	Present	No	No	
Although some authors have considered lymphoproliferative diseases in psittacines to be associated with avian leukosis viruses (ALV) (Girling 2003), reports of psittacine disease have either not investigated aetiology (Palmer and Stauber 1981; Paul-Murphy et al 1985; Messonier 1992; de Wit et al 2003) or failed to detect the presence of exogenous retrovirus (Ramos-Vara et al 1997). The largest serological study of renal neoplasia in budgerigars found no correlation between the expression of ALV antigen and the presence of tumours (Neumann and Kummerfeld 1983). A more recent study using product-enhanced reverse transcriptase (PERT) assays similarly found no evidence of an exogenous replicating retrovirus being associated with renal neoplasia in budgerigars (Simova-Curd et al 2010).					
Togaviridae (Whataroa virus, Arbovirus A group)	No	Present	No	No	
Togaviridae (Highlands J virus, Arbovirus A group)	No	Exotic	No	No	
Togaviridae (Western equine encephalitis virus, Arbovirus A group)	No	Exotic	No	Yes	
Togaviridae (Eastern equine encephalitis virus, Arbovirus A group)	No	Exotic	Yes	Yes	Deem et al 2005
Togaviridae (Alphavirus spp., Arbovirus A group)	No	Exotic	No	No	
FUNGI AND YEASTS					
Arthrodermataceae (*Microsporum* spp., Favus, Dermatophytosis)	Yes	Present	No	No	
Arthrodermataceae (*Trichophyton* spp., Favus, Dermatophytosis)	Yes	Present	No	No	
Orbiliaceae (*Dactylaria gallopava*, Dactylariosis)	No	Present	No	No	
Saccharomycetaceae (*Candida albicans*, Candidiasis, thrush)	Yes	Present	No	No	
Saccharomycetales (*Macrorhabdus ornithogaster*, Megabacteriosis, avian yeast gastritis, proventricular disease)	Yes	Present	No	No	Johnstone and Cork 1993
Tremellaceae (*Cryptococcus neoformans*, *Filobasidiella neoformans*)	Yes	Present	No	No	González-Hein et al 2010
Trichocomaceae (*Aspergillus fumigatus*)	Yes	Present	No	No	

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
FUNGI AND YEASTS (continued)					
Trichocomaceae (*Aspergillus* spp.)	Yes	Present	No	No	Johnstone and Cork 1993
Unikaryonidae (*Microsporidia* spp., Microsporidiosis)	Yes	Exotic	Yes	No	Ladds 2009; Doneley 2009; Didier et al 2004
Microsporidia are single-celled, obligate intracellular parasites that were recently reclassified from protozoa to fungi.					
PARASITES and PROTOZOA					
Haematozoa					
Babesiidae (*Babesia* spp.)	No	Some species	No	No	McKenna 2010
Hepatozoidae (*Hepatozoon* spp.)	No	Some species	No	No	McKenna 2010
Hepatozoon albatrossi and *H.kiwii* are recorded in New Zealand.					
Plasmodiidae (*Akiba caulleryi*, *Leucocytozoon caulleryi*)	No	Exotic	No	No	Rae 1995
Plasmodiidae (*Haemoproteus* spp.)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
H. danilewsky and *Haemoproteus* sp. are present in New Zealand.					
Plasmodiidae (Leucocytozoon spp.)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
L. fringillinarum, L. tawaki and *Leucocytozoon* sp. are present in New Zealand.					
Plasmodiidae (*Plasmodium* spp., Avian malaria)	Yes	Some species	Yes	No	Tompkins and Gleeson 2006; McKenna 2010
P. cathermerium, P. elongatum, P. relictum, and *Plasmodium* sp. are present in New Zealand.					
Plasmodiidae (*Trypanosoma avium*)	Yes	Exotic	Yes	No	Coles 2007
Coccidia					
Cryptosporidiidae (*Cryptosporidium* spp.)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
Cryptosporidium sp. has been isolated from many avian species in New Zealand.					
Eimeriidae (*Atoxoplasma* spp.)	Yes	Present	No	No	Johnstone and Cork 1993; McKenna 2010; Levine 1982
Atoxoplasma paddae and *Atoxoplasma* sp. have been found in New Zealand including in a Kaka.					
Eimeriidae (*Caryospora* spp.)	No	Exotic	No	No	Muller 2010
Eimeriidae (*Eimeria* spp.)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
More than 13 species of Eimeria have been reported in New Zealand.					
Eimeriidae (*Isospora* spp.)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
I. turdi and *Isospora* sp. are present in New Zealand.					

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
Coccidia (continued)					
Hepatozoidae (*Hepatozoon* spp.)	No	Some species	No	No	Peirce 2005
Sarcocystidae (*Sarcocystis falcatula*)	Yes	Some species	Yes	No	Cray et al 2005; Johnstone and Cork 1993; McKenna 2010
Sarcocyst observed in a pigeon. *S. falcatula* limited to North America and opposum is the definitive host. No suitable definitive host in New Zealand.					
Sarcocystidae (*Toxoplasma gondii*)	Yes	Yes	No	No	Coles 2007; McKenna 2010
Toxoplasma gondii is frequently found in birds in New Zealand.					
Flagellated protozoa					
Dientamoebidae (*Histomonas meleagridis*, Blackhead)	No	Present	No	No	McKenna 2010
Hexamitidae (*Giardia* spp.)	Yes	Some species	Yes	No	McKenna 2010
Hexamitidae (*Spironucleus* spp., Hexamita)	Yes	Exotic	Yes	No	Saif et al 2008; McKenna 2010
Spironucleus sp. was recorded in an ostrich in New Zealand.					
Trichomonadidae (*Cochlosoma* spp.)	Yes	Exotic	Yes	No	Ladds 2009; Coles 2007
Trichomonadidae (*Trichomonas* spp., Canker; Roup)	Yes	Some species	No	No	McKenna 2010; Greiner and Ritchie 1994
T. gallinae is responsible for Trichomoniasis in psittacines and is widespread in New Zealand.					
Internal parasites					
Acanthocephala (Spiny-headed worms)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
Cestoda (Tapeworms)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
Nematoda (Roundworms)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
Trematoda (Flukes)	Yes	Some species	Yes	No	Coles 2007; McKenna 2010
External parasites					
Siphonaptera (Fleas)	Yes	Some species	Yes	No	Blank et al 2007
Euhirudinea (Leeches)	No	Some species	No	No	Davies et al 2008
Amblycera (Chewing lice)	Yes	Some species	Yes	No	Dik 2010
Ischnocera (Chewing lice)	Yes	Some species	Yes	No	Dik 2010
Acarina (Mites)	Yes	Some species	Yes	No	Pence 2008
Diptera (*Cochliomyia homnivorax*, New World screwworm fly (NWS), myiasis)	No	No	No	Yes	Little 2008

Table 1 (continued)

Organism	Recorded in psittacines	NZ status	Consider further?	OIE	Reference
External parasites (continued)					
Diptera (*Chrysomyia bezziana*, Old World screwworm fly (NWS), myiasis)	No	No	No	Yes	Little 2008
Argasidae (Soft ticks)	Yes	Some species	Yes	No	Pence 2008
Ixodidae (Hard ticks)	Yes	Some species	Yes	No	Pence 2008
OTHER					
Internal papillomatous disease (IPD), Cloacal papillomatosis	Yes	Present	No	No	Gartrell et al 2009

IPD may be a result of persistent herpesvirus infection but has been found in birds in NZ with no evidence of herpesvirus.

5.2. HAZARD IDENTIFICATION

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

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

For any organism, if the answer to question 1 is “yes” (and the answer to question 2 is “yes” in the cases of organisms requiring a vector) and the answers to either questions 3 or 4 are “yes”, it is identified as a potential hazard requiring risk assessment.

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

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

5.3. RISK ASSESSMENT

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

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

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

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 risk.

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

5.4. RISK MANAGEMENT

For each organism classified as a hazard, a risk management step is carried out which identifies the options available for managing the risk. Where the *Code* lists recommendations for the management of a hazard, these are described and unrestricted entry or prohibition may also be considered. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when an import health standard (IHS) is drafted.

As obliged under Article 3.1 of the WTO Agreement on Sanitary and Phytosanitary Measures (the SPS Agreement) the measures adopted in IHSs will be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3 (where measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate following a risk assessment).

5.5. RISK COMMUNICATION

After an import risk analysis has been written, the Animal Imports team of MPI analyse the options available and propose draft measures for the effective management of identified risks. These are then presented in a draft IHS which is released together with a risk management proposal summarising the options analysis, the rationale for the proposed measures and a link to the draft risk analysis. The package of documents is released for a six-week period of

stakeholder consultation. Stakeholder submissions in relation to these documents are reviewed before a final IHS is issued.

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6. Adenovirus

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

The family *Adenoviridae* contains five genera; *Mastadenovirus (*mammalian adenoviruses), *Ichtadenovirus* (fish adenoviruses), *Aviadenovirus* (group I avian adenoviruses), *Siadenovirus* (group II avian adenoviruses), and *Atadenovirus* (group III avian adenoviruses) (Harrach et al 2011).

The genus *Aviadenovirus* contains seven species: *Fowl adenovirus A-E* (FAdV), *Goose adenovirus*, and *Falcon adenovirus A* (FaAdV-1); and six tentative species: *Duck adenovirus B* (DAdV-2), *Meyer's parrot adenovirus 1*, *Pigeon adenovirus 1* (PiAdV-1), *Psittacine adenovirus 1* (PsAdV-1), and *Turkey adenovirus 1* and *2* (TAdV) (Harrach et al 2011).

The genus *Siadenovirus* contains three species: *Frog adenovirus* (FrAdV-1), *Raptor adenovirus A* (RAdV-1) and *Turkey adenovirus A* (TAdV-3); and five unclassified Siadenoviruses: *Budgerigar adenovirus 1* (BuAdV-1), *Great tit adenovirus 1* (GTAdV-1), *Psittacine adenovirus 2* (PsAdV-2) (also known as *Plum headed parakeet adenovirus 1* (Wellehan et al 2009)), *Sulawesi tortoise adenovirus 1* (STAdV-1), and *South Polar skua adenovirus 1* (Harrach et al 2011).

6.1.2. OIE list

Not listed

6.1.3. New Zealand status

Serological surveys consistently demonstrate that adenoviruses are widespread in New Zealand poultry (Howell 1992; Anonymous 2001; Poland 2002; Poland 2004) and in domestic and wild pigeons (Black et al 2004).

Disease in a Jardine's parrot (Stone 2005), rainbow lorikeet, cockatiel, red-tailed black cockatoo, and an unspecified species of parrot (MAF 2010) in New Zealand have each been associated with adenovirus infection. A causative organism was not however isolated and there remains no conclusive record of adenovirus infection in psittacines in this country.

6.1.4. Epidemiology

Genus: *Aviadenovirus*

Infections with avian adenoviruses are widespread throughout the world and have been described in a wide range of avian species (Monreal 1992; Adair and Fitzgerald 2008). Aviadenoviruses infect birds only (Fauquet et al 2005) and have no known public health significance. Aviadenoviruses have been reported in Galliformes, Columbiformes, Anseriformes, and Psittaciformes (Gerlach 1994). Infections with adenovirus (Ramis et al 1994; Capua et al 1995; Weissenböck and Fuchs 1995; Soike et al 1998; Hess et al 2000) or adenovirus-like particles (Scott et al 1986, Pass 1987, Mori et al 1989, Gomez-Villamandos et al 1995; Mackie et al 2003) have been described in a variety of psittacine birds.

The pathogenic role of most of the group I adenoviruses is questionable (Hess 2000), with the exceptions of FAdV strains which cause quail bronchitis and hydropericardium syndrome (Adair and Fitzgerald 2008). Different serotypes and different strains of the same serotype vary with regard to their ability to cause disease (Cook 1974; Dhillon and Winterfield 1984; McCracken and Adair 1993; El-Attrache and Villegas 2001) and it has been suggested that many avian adenoviruses cause disease only in the presence of a secondary agent (Adair and Fitzgerald 2008).

Lesions associated with adenoviruses in psittacine birds include hepatitis, conjunctivitis, interstitial pneumonia, enteritis, and splenic lymphoid depletion (Wellehan et al 2009). Although avian adenoviruses have been detected in psittacines many times by electron microscopy, only limited data exist on their classification (Raue et al 2005). Most infections have been associated with different serotypes of FAdV, and evidence of FAdV-1 (Capua et al 1998), FAdV-2 (McFerran et al 1976), FAdV-3 (Capua et al 1995), FAdV-4 (Soike et al 1998), and FAdV-8 (McFerran et al 1976) infections in psittacines have been reported.

Most of the group I avian adenoviruses are excreted via the faeces and transmission occurs by the faecal-oral route (Balamurugan and Kataria 2004). Vertical transmission and horizontal spread through contaminated faeces and mechanical vectors has also been reported (Cowen 1992; Akhtar 1994; Ashraf et al 2000; Ganesh and Raghavan 2000; Mazaheri et al 2003; Balamurugan and Kataria 2004).

Quail bronchitis (QB) virus and chicken embryo lethal orphan (CELO) virus are considered to be the same agent and are the type strain of group I, serotype 1 avian adenovirus (FAdV-1) (DuBose and Grumbles 1959; Reed and Jack 2008; Smyth and McNulty 2008a). There is only one report of psittacines infected with adenoviruses similar to FAdV-1 (Capua et al 1998). FAdV-1 is recognised as present in New Zealand (Saifuddin 1990; Saifuddin et al 1992).

Hydropericardium syndrome (HPS) is associated with group I, serotype 4 avian adenovirus (FAdV-4) (Hess et al 1999; Ganesh and Raghavan 2000; Schonewille et al 2008). The disease principally affects 3-6 week old broilers (Cowen 1992; Asrani et al 1997; Ashraf et al 2000) and can induce disease in the absence of immune suppression (Naeem et al 1995; Hess et al 1999; Kim et al 2008). In broilers, natural outbreaks of HPS are characterised by the sudden onset of the disease with few or no clinical signs other than sudden heavy mortality (Cowen 1992; Chandra et al 2000), although mortality in layers or breeders is low (10-20%) (Ashraf et al 2000; Kim et al 2008). FAdV-4 has been isolated from two species of parrot in Germany (Soike et al 1998), however the isolate was found to be different from the highly pathogenic strains responsible for HPS and experimental inoculation of chickens with the psittacine isolate resulted in no mortality (Hess et al 2000).

A novel group I avian adenovirus was detected in Senegal parrots (*Piocephalus senegalus*) with a hepatopathy (Raue et al 2005). The virus was described on the basis of molecular characterisation and later isolated (Lüschow et al 2007) and has been designated as *Psittacine adenovirus 1* (PsAdV-1) (Raue et al 2005). Lüschow et al (2007) have established a real-time PCR for rapid and sensitive detection of PsAdV-1. A further psittacine adenovirus was identified in a Meyer's parrot (*Piocephalus meyeri*) with characteristic inclusion body hepatitis by Wellehan et al (2005) using molecular methods. The new virus was classified as a group I avian adenovirus and designated as *Meyer's parrot adenovirus 1*. The lesions seen in these cases were consistent with lesions associated with adenoviral infection in other psittacine species and with experimental aviadenoviral disease in poultry.

There have been no subsequent reports of either PsAdV-1 or *Meyer's parrot adenovirus 1* infections in any avian species.

Genus: *Siadenovirus*

A variety of avian species are reported to be susceptible to experimental or natural infection with strains of TAdV-3 (Cowen et al 1988; Gomez-Villamandos et al 1995; Pierson and Fitzgerald 2008; Shivaprasad 2008). Avian adenovirus splenomegaly virus (AASV) of chickens, turkey haemorrhagic enteritis virus (THEV), and marble spleen disease virus (MSDV) of pheasants are all considered to be strains of TAdV-3 (Smyth and McNulty 2008b). A serologic survey of 42 wild bird species indicates that infection of wild birds is uncommon (Domermuth et al 1977). Mammals are not susceptible to infection with TAdV-3 and the virus poses no threat to public health (Smyth and McNulty 2008b).

Serological evidence indicates that TAdV-3 is widespread in many countries (Domermuth et al 1980). Despite this, clinically affected birds are rare (Fitzgerald et al 1994) and TAdV-3 is thought by some to be not a primary pathogen (Hess 2000).

Horizontal transmission of TAdV-3 occurs via the oral-faecal route. There is no evidence of vertical transmission (Chandra and Kumar 1998; Pierson and Fitzgerald 2008).

Gomez-Villamandos et al (1995) report a disease outbreak in psittacine birds which resembled haemorrhagic enteritis of turkeys. Immunohistochemical staining showed positive immunoreaction to TAdV-3 antigens in the intranuclear inclusion bodies. However, no genetic investigation was carried out. Subsequently, Wellehan et al (2009) identified a novel adenovirus from two psittacine birds (a plum-headed parakeet (*Psittacula cyanocephala*) and an umbrella cockatoo (*Cacatua alba*)) displaying non-specific clinical signs including lethargy and weight loss. Phylogenetic and comparative sequence analysis suggest that the virus is a *Siadenovirus* and it has been designated as *Psittacine adenovirus 2* (Wellehan et al 2009). In the same year, Katoh et al (2009) detected adenoviral DNA from budgerigars displaying ruffled feathers or death without clinical signs. Sequencing and phylogenetic analysis of the hexon gene revealed that the virus is a *Siadenovirus* and it has been designated as *Budgerigar adenovirus 1* (BuAdV-1). There have been no subsequent reports of either BuAdV-1 or *Psittacine adenovirus 2* infections in any avian species and their clinical significance is not known.

6.1.5. Hazard identification conclusion

Genus: *Aviadenovirus*

Aviadenoviruses are widespread in poultry and pigeons in New Zealand and several psittacine birds have demonstrated clinical signs consistent with adenovirus infection. McFerran and Smyth (2000) suggested that there are no trade implications for conventional avian adenovirus infections except for highly virulent viruses associated with hydropericardium syndrome (which is exotic to New Zealand but has not been described in psittacines) or inclusion body hepatitis (which is recognised in New Zealand). There is only limited evidence to support a pathogenic role for group I avian adenoviruses in psittacines and there is no evidence that exotic serotypes or strains likely to be present in imported psittacines are more pathogenic than those in New Zealand. Aviadenoviruses are not identified as a potential hazard in live psittacines.

Genus: *Siadenovirus*

A variety of avian species are reported to be susceptible to infection with strains of TAdV-3. However, the pathogenic role of this virus in species other than turkeys, chicken and pheasants is questionable. There are no reports of TAdV-3 isolation and only three instances of *Siadenovirus* infections in psittacine species. The associated clinical signs of these cases were non-specific and the clinical significance of the virus in psittacines is not known. *Siadenovirus* infections in psittacine species are extremely rare and are associated with non-specific clinical signs. Therefore Siadenoviruses are not identified as a potential hazard in live psittacines.

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7. Avian paramyxovirus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Nine avian paramyxovirus (APMV) species are recognised (Wang et al 2012), which are differentiated on serological grounds. Newcastle disease (ND) is caused by viruses belonging to serogroup APMV-1.

APMV-1, APMV-2, APMV-3, and APMV-5 have been demonstrated in Psittaciformes but most often infections are associated with APMV-3 (Alexander 1993; Szeleszczuk and Ledwon 2005; Leighton and Heckert 2007; Jung et al 2009). The psittacine species considered most susceptible to infection are Amazon parrots (*Amazona* spp.), eclectus parrots (*Eclectus roratus*), *Neophema* spp., and lovebirds (*Agapornis* spp.) (Szeleszczuk and Ledwon 2005).

7.1.2. OIE list

Newcastle disease is included in the OIE list of notifiable diseases. Strains of APMV-1 vary greatly in pathogenicity. Newcastle disease (ND) is defined by the *Code* (OIE 2011) as an infection of poultry caused by a virus (NDV) of avian paramyxovirus serotype 1 (APMV-1) that meets one of the following criteria for virulence:

a) the virus has an intracerebral pathogenicity index (ICPI) in day-old chicks (*Gallus gallus*) of 0.7 or greater; or
b) multiple basic amino acids have been demonstrated in the virus (either directly or by deduction) at the C-terminus of the F2 protein and phenylalanine at residue 117, which is the N-terminus of the F1 protein. The term 'multiple basic amino acids' refers to at least three arginine or lysine residues between residues 113 and 116. Failure to demonstrate the characteristic pattern of amino acid residues as described above would require characterisation of the isolated virus by an ICPI test.

7.1.3. New Zealand status

Apathogenic and mildly pathogenic (ICPI < 0.2) strains of APMV-1 occur in New Zealand, which have a precursor glycoprotein cleavage site containing no more than two basic amino acids (Pharo et al 2000; Stanislawek et al 2001; Stanislawek et al 2002). Exotic strains of APMV-1 of higher pathogenicity are notifiable organisms (Tana et al 2011). Newcastle disease has never been diagnosed in New Zealand. APMV-1 has been isolated from mallard ducks, chickens and one parrot, and all isolates have been demonstrated to be avirulent (Pharo et al 2000).

In addition to the APMV-1 isolations, Stanislawek et al (2002) recovered APMV-4 from healthy ducks and found serological evidence for APMV-1, 2, 3, 4, 6, 7, 8, and 9. However, because of cross-reactions and non-specific reactions, the authors were only prepared to claim their serology findings indicated the presence of APMV-6. A study of caged birds, wild birds, and poultry in New Zealand was unable to find any evidence of APMV-2 or APMV-3 in poultry or APMV-3 in wild birds, and the results of this study did not provide conclusive evidence for the presence of APMV-2 in wild birds (Stanislawek et al 2001).

Thus, although the presence of APMV-2 and -3 in New Zealand cannot be excluded, there is no conclusive evidence of their presence, and only APMV-1, 4 and 6 can be concluded to be present.

7.1.4. Epidemiology

APMV-1

Newcastle disease has been eradicated in several countries but NDV strains of low virulence are still found in poultry throughout the world. Velogenic NDV is endemic in areas of Mexico, Central and South America, Asia, the Middle East, and Africa, and in double-crested wild cormorants in the US and Canada (OIE 2009).

NDV has been shown to infect over 240 species of bird (Dortmans et al 2011) and it is likely that all birds are susceptible to infection, but the disease seen with any specified strain of virus may vary considerably with host (Kaleta and Baldauf 1988; Alexander and Senne 2008). For example, chickens are highly susceptible to disease, turkeys do not generally develop severe signs, and game birds, Passeriformes, and Psittaciformes are variable in their susceptibility. Wild birds and waterfowl (order Anseriformes) may harbour virus subclinically but some isolates within certain genotypes have caused epornitics within these species (OIE 2009).

NDV has been isolated from psittacine birds (Brunning-Fann et al 1992; Panigrahy et al 1993; Clavijo et al 2000). However infection and disease due to APMV-1 are uncommon (Szeleszczuk and Ledwon 2005).

The type and severity of clinical signs seen in birds infected with APMV-1 vary widely and depend on the viral strain and host species. Based on the disease produced in chickens, five pathotypes have been described (Dortmans et al 2011). However, pathotype groupings are rarely clear-cut and the less pathogenic strains may also induce severe disease if exacerbated by co-infections or by adverse environmental conditions. Experimental infection of a range of psittacine species with virulent NDV produced signs of depression, diarrhoea and deaths but the severity of signs differed between species (Erickson et al 1977; April and Pearson 1985).

NDV may cause a transient and non-life threatening infection in humans. Disease is manifested by excessive lachrymation, oedema of the eyelids, conjunctivitis and sub-conjunctival haemorrhage, and occasionally fever (OIE 2009). Reports of human to human transmission have not been identified.

Virus is shed in the respiratory secretions and faeces of infected birds and then spread by direct contact. Some psittacine birds have been demonstrated to shed NDV intermittently for over 1 year (Erickson et al 1977). Transmission occurs principally by the faecal-oral route or inhalation. However, fomites and contamination of waterways are also possible means of spread (McFerran et al 1968; Alexander 1988). Recovered birds do not remain long-term carriers of the virus.

Live vaccines derived from low virulence and moderate virulence APMV-1 strains, or inactivated vaccines, are used in many countries to control clinical disease (OIE 2009). Vaccination may protect birds exposed to pathogenic virus from clinical disease although it does not prevent infection and subsequent viral excretion (Parede and Young 1990; Alexander et al 1999).

There is limited information on the epidemiology of avian paramyxoviruses other than APMV-1. Given the similarities between APMV-1 and other avian paramyxoviruses in infection and replication, it has been suggested that the same mechanisms of introduction and spread would apply (Alexander 2000).

APMV-2

APMV-2 (also called Yucaipa virus) was first isolated from chickens and turkeys in America and Europe, but is more commonly isolated from passerine birds (Alexander and Senne 2008), in which most infections are subclinical or result in mild respiratory disease only (Ritchie 1995). APMV-2 isolations are rarely reported from psittacine birds (Alexander 1993; Szeleszczuk and Ledwon 2005; Leighton and Heckert 2007) and African grey parrots may be more susceptible than other psittacine species (Kaleta and Baldauf 1988).

APMV-2 infection of poultry leads to shedding from the respiratory and intestinal tracts (Alexander and Senne 2008). The large numbers of isolations of APMV-2 from passerine birds compared to psittacine birds in United Kingdom quarantine facilities is considered to point to psittacine infections being related to close contact in transit or quarantine with passerine birds (Alexander and Senne 2008). Similar conclusions were drawn from a study of birds imported into the USA (Senne et al 1983).

APMV-3

Two sub-groups of APMV-3 are recognised based on structural polypeptide analysis and monoclonal antibody serotyping. Group 1 isolates are mainly found in turkeys and group 2 isolates in psittacines (Anderson et al 1987). APMV-3 is isolated relatively frequently from caged and quarantined birds, mainly psittacines but also passerines (Shortridge et al 1991; Alexander 1993; Shihmanter et al 1998; Leighton and Heckert 2007; Alexander and Senne 2008). There have been no reports of isolation of APMV-3 from wild birds and infections in caged birds are likely to be associated with stress.

Infection may result in a mild respiratory disease (Alexander and Senne 2008) and neurological signs such as ataxia, torticollis, opisthotonos, and unilateral or bilateral paralysis of wings or legs (Shihmanter et al 1998; Szeleszczuk and Ledwon 2005; Jung et al 2009).

APMV-5

APMV-5 (Kunitachi virus) causes a rare disease of budgerigars that is characterised by very high mortality. The virus was reported from pet budgerigars in Japan between 1974 and 1976 (Yoshida et al 1977) and similar viruses have been identified from diseased budgerigars in Australia and the United Kingdom (Mustaffa-Babjee et al 1974; Gough et al 1993).

7.1.5. Hazard identification conclusion

APMV-4 and -6 are recognised as present in New Zealand, and are not identified as potential hazards in the commodity.

Psittacines are susceptible to infection with APMV-1, 2, 3, and 5. Importation of birds has been implicated in the introduction of NDV to the United States (Senne et al 1983; Brunning-Fann et al 1992). Although APMV-1 has been recovered in New Zealand, all isolates have

been shown to have an ICPI<0.7 and a precursor glycoprotein cleavage sequence (residues 113 to 116) containing no more than two basic amino acids.

NDV, APMV-2, 3 (psittacine strains), and 5 are therefore identified as potential hazards in live psittacines.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

NDV

Surveys of free-living psittacines have found few birds with evidence of paramyxovirus infection (Garnett and Flanagan 1989; Gilardi et al 1995; Karesh et al 1997; Stanislawek et al 2001; Deem et al 2005; Stone et al 2005). However, psittacines are considered to be common reservoir hosts for APMV-1 (Ritchie 1995). There are numerous reports of APMV-1, including velogenic strains, in captive psittacine birds, especially following international transport or during quarantine (Ashton and Alexander 1980; Senne et al 1983; Panigrahy et al 1993; Clavijo et al 2000).

The virulence of isolates of APMV-1 from psittacines varies, as does the susceptibility of different species of birds, and subclinical infections are possible. Subclinically-infected birds are unlikely to be detected during routine clinical examination. The likelihood of entry of NDV with live psittacines is therefore assessed to be non-negligible.

APMV-2

Reports of APMV-2 in passerine birds are relatively common (Nymadava et al 1977; Fleury and Alexander 1979; Tumova et al 1979; Senne et al 1983; Goodman and Hanson 1988) and passerines are considered to be the primary hosts for APMV-2 (Alexander 1986). Many surveys of free-living psittacines have failed to provide evidence of APMV-2 infection (Karesh et al 1997; Deem et al 2005; Stone et al 2005). However, APMV-2 has been isolated from a variety of captive psittacine species (Ritchie 1995). Psittacines are thought to become infected following close contact with infected passerines (Alexander 1986). Infection with APMV-2 may be associated with mild respiratory signs and infected birds may not be detected during routine clinical examination. The likelihood of entry of APMV-2 with live psittacines is therefore assessed to be low.

AMPV-3 (psittacine strains)

Surveys of free-living psittacines have failed to provide evidence of APMV-3 infection (Karesh et al 1997; Deem et al 2005; Stone et al 2005). However, APMV-3 is frequently isolated from pet psittacines in quarantine. Isolations of the virus from cage birds have almost exclusively been made from birds in quarantine. The likelihood of entry of APMV-3 with live psittacines is therefore assessed to be low.

APMV-5

APMV-5 is extremely rare and has only ever been reported in budgerigars (Mustaffa-Babjee et al 1974; Nerome et al 1978; Gough et al 1993). Therefore the likelihood of entry of APMV-5 with budgerigars is assessed to be extremely low. The likelihood of entry of APMV-5 with other species is assessed to be negligible.

7.2.2. Exposure assessment

Although little is known about the epidemiology of avian paramyxoviruses other than NDV, they are all expected to behave in a similar manner. Some birds may shed NDV for extended periods. Imported psittacines may be integrated into aviaries or may contact other birds at shows or pet shops. The Department of Conservation exercises control over private holdings of native psittacine birds, which will reduce the likelihood of introduced exotic species mixing with captive native birds. However, native birds kept in captivity and in contact with imported birds would be at risk. The likelihood of exposure to NDV, APMV-2, and APMV-3 from infected imported psittacines is therefore assessed to be non-negligible. Similarly, the likelihood of exposure to APMV-5 from infected imported budgerigars is assessed to be non-negligible.

Wild birds could have contact with imported birds or the waste products from their enclosures and contact through illegal releases and escapes may occur. Transmission of infection will depend on the host specificity of the strain of virus and the host range that might be exposed. For example, an outbreak of the velogenic strain of APMV-1 in psittacine birds throughout much of the United States did not spread to other avian orders (Panigrahy et al 1993). Wild birds have previously been implicated in the introduction and spread of NDV to poultry flocks (Lancaster 1966; Alexander 1988). There is a non-negligible likelihood of commercial poultry being exposed to NDV through infected wild birds.

7.2.3. Consequence assessment

NDV

The introduction of NDV would have serious consequences for the poultry industry and could result in substantial mortalities in wild or caged birds.

Reports indicate that both velogenic and vaccine strains of APMV-1 may cause transient conjunctivitis or fever in humans. However, disease is mild and reports are rare.

Therefore the consequences of introduction and establishment of NDV are assessed to be significant.

APMV-2

APMV-2 is most commonly isolated from passerine birds in which infections are usually mild and self-limiting. APMV-2 has been isolated from poultry in many parts of the world but reports of disease are scarce. Although there have been reports of severe respiratory disease in turkeys in the United States and Israel (Alexander and Senne 2008), an epidemiological study of the relationship between APMV-2 infection and acute respiratory disease syndrome in the United States did not indicate an aetiological connection and experimental infections of both turkeys (Bankowski et al 1981) and chickens (Bankowski and Corstvet 1961) resulted in mild disease only. There have been no reports of APMV-2 infecting humans.

The introduction of APMV-2 in the commodity would be associated with non-negligible consequences for the New Zealand poultry industries and negligible consequences for other species including humans.

APMV-3 (psittacine strains)

APMV-3 is isolated relatively frequently from caged and quarantined psittacines. There have been no reports of isolation of APMV-3 from wild birds and infections in caged birds are likely to be associated with stress. Infection may result in a mild respiratory disease and neurological signs. However, there are no documented reports of imported birds being responsible for disease in poultry or outbreaks of disease in cage birds. There is no evidence that the disease is of any economic importance anywhere in the world. For these reasons the consequences for poultry and caged birds that could result from importing the virus are assessed to be negligible. Since there have been no reports of the virus causing disease in wild birds it is unlikely that the virus would have any deleterious effects on New Zealand native birds. There have been no reports of APMV-3 infecting humans.

The likelihood that there would be any significant consequences for domestic or wild birds or humans is assessed to be negligible.

APMV-5

APMV-5 is extremely rare and has only been recognised in budgerigars in which high mortality rates may occur. The introduction of APMV-5 in the commodity would be associated with extremely low consequences for budgerigars and negligible consequences for other species including humans.

7.2.4. Risk estimation

Since entry, exposure, and consequence assessments are all non-negligible, the risk estimate for NDV, APMV-2, and APMV-5 (only in budgerigars) is non-negligible, and these viruses are classified as risks in the commodity. Therefore, risk management measures can be justified.

Since the consequence assessment for APMV-3 (psittacine strains) is negligible, the risk estimate is negligible and risk management measures cannot be justified.

7.3. RISK MANAGEMENT

7.3.1. Options

The following factors were considered when drafting options for the effective management of avian paramyxoviruses in live psittacines:

- 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 do not imply that the birds are presently infected.

- Quarantine and testing birds after they have been in quarantine for 3 weeks could be used to demonstrate freedom of individual birds from infection.

- Since some operators find bleeding of small psittacines difficult, birds could be tested by virus isolation or PCR instead of serological tests (Wise et al 2004; Pham et al 2005; OIE 2009). While serological tests require blood samples, PCR and virus isolation can be done using cloacal and tracheal swabs.

- The *Code* contains no recommendations for sanitary measures appropriate to manage the risk of APMV-2 or APMV-5. However, recognition of country, zone, or compartment freedom from NDV could be extended to include freedom from these strains and species.

Article 10.9.5. of the current OIE *Code* (OIE 2011) recommends that, 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 infection by NDV 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.

According to the *Code*, a country, zone, or compartment may be considered free from ND when it has been shown that NDV infection has not been present for the past 12 months, based on surveillance in accordance with Articles 10.9.22 to 10.9.26. If infection has occurred in a previously free country, zone, or compartment, ND free status can be regained three months after a stamping-out policy (including disinfection of all affected establishments) is applied, providing that surveillance in accordance with Articles 10.9.22 to 10.9.26 has been carried out during that three-month period.

Article 10.9.25 of the *Code* makes provisions for the recognition of ND-freedom in countries, zones, or compartments that practise vaccination against NDV. New Zealand could recognise APMV-1 freedom in a country, zone, or compartment practising vaccination using a lentogenic virus strain with an ICPI < 0.7 or an inactivated APMV-1 vaccine.

Although the OIE *Manual* only describes tests for APMV-1, the samples taken and methods involved for the isolation of other avian paramyxoviruses are identical (Alexander and Senne 2008). Virus isolation can be performed by egg inoculation with cloacal or tracheal swabs taken from live birds, followed by testing of haemagglutinating activity with monospecific antiserum to APMV-1, 2, and 5.

Haemagglutination, haemagglutination inhibition tests and ELISAs may be used for the serological diagnosis of ND (OIE 2009).

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

Option 1

Imported birds could be derived from a country, zone or compartment free from NDV, APMV-2, and APMV-5 (budgerigars only) since they were hatched or for at least the past 21 days. Freedom could be based on surveillance in accordance with Articles 10.9.22 to 10.9.26 of the *Code*.

Vaccination could be permitted using an inactivated APMV-1 vaccine or a live lentogenic virus strain which is shown to have an ICPI < 0.7.

Option 2

Birds to be imported could:

i. 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

ii. be subjected to a diagnostic test (serology, virus isolation or PCR) for NDV, APMV-2, and APMV-5 (budgerigars only) on samples taken at least 14 after entry into quarantine, with negative results; and;

iii. have not been vaccinated against ND;

This option is essentially the *Code* recommendation for ND, but whereas the *Code* refers to testing only for NDV, this option also includes APMV-2 and APMV- 5 (budgerigars only).

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 3

Birds to be imported could:

i. 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

ii. be subjected to a diagnostic test (serology, virus isolation or PCR) for NDV, APMV-2, and APMV-5 (budgerigars only), on samples taken at least 21 days after entry into quarantine, with negative results; and;

iii. have not been vaccinated against ND.

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 4

A further option is to use sentinel birds in pre-export quarantine, which would have to be tested 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 consignment. Choice of sentinel birds might include SPF chickens.

Option 5

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.

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8. Coronavirus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

The *Coronaviridae* family is divided into two subfamilies, *Coronavirinae* and *Torovirinae*. Only viruses of the *Coronavirinae* subfamily have been reported to infect birds (Cook 2008). Three genera, *Alphacoronavirus, Betacoronavirus,* and *Gammacoronavirus* have been used to replace the traditional group 1, 2, and 3 coronaviruses (González et al 2003). Alphacoronaviruses and betacoronaviruses are found in mammals, whereas gammacoronaviruses are detected primarily in birds (Muradrasoli et al 2010; Chu et al 2011).

Two *Gammacoronavirus* species are currently recognised; *Avian coronavirus* and *Beluga whale coronavirus SW1* (De Groot et al 2011). *Avian coronavirus* includes avian infectious bronchitis virus (IBV), turkey coronavirus (TCoV), pheasant coronavirus (PhCoV), and recently identified coronaviruses from several species of wild birds (Jonassen et al 2005; Woo et al 2009).

Coronaviruses similar to the group 3 coronaviruses have been found in a variety of avian species (Cavanagh 2005; Jackwood et al 2010) and a fourth genus, *Deltacoronavirus*, has recently been proposed (Chu et al 2011; Woo et al 2010).

8.1.2. OIE list

Avian infectious bronchitis is an OIE-listed disease.

8.1.3. New Zealand status

Serological testing confirms that IBV is widespread in New Zealand (Watts 2010) and 28 strains have been identified using PT-PCR (Ramneek et al 2005). Exotic strains of IBV are listed as unwanted exotic organisms (MAF 2011).

Coronaviruses have not been described in other avian species in New Zealand.

8.1.4. Epidemiology

Coronaviruses have a worldwide distribution, are highly infectious and extremely difficult to control. They can cause respiratory, enteric, and in some cases hepatic and neurological diseases in a wide variety of animals and in humans (Jackwood et al 2010).

Gammacoronaviruses are known to infect many species of birds, including chickens, turkeys, pheasants, ducks, and pigeons (Jones et al 2011). TCoV causes a highly contagious enteric infection, which can lead to mortality and growth retardation (Cavanagh 2005). PhCoV typically affects renal and respiratory tissues with consequent disease signs related to those body systems (Cavanagh 2005). IBV, recognised as the prototypic *gammacoronavirus*, primarily causes disease in chickens, characterised by upper respiratory tract signs, including nasal discharge, rales, watery eyes and lethargy (Cavanagh 2005).

There are no reports of natural or experimental infections of psittacine species with gammacoronaviruses.

The emergence of severe acute respiratory syndrome (SARS) in humans in 2003 has led to a marked increase in the number of coronaviruses discovered and coronavirus genomes being sequenced (Woo et al 2010; Mihindukulasuriya et al 2008). Since then, new members of the family *Coronaviridae* have been identified in birds (Jonassen et al 2005; Liu et al 2005), humans (van der Hoek et al 2004; Woo et al 2005), bats (Poon et al 2005), and other mammals (Dong et al 2007).

Coronaviruses have a high mutation and recombination rate (Muradrasoli et al 2010) which can result in the generation of new species or genotypes (Woo et al 2010) and may allow them to adapt to new hosts and ecological niches (Woo et al 2009).

Relatively little is known about the biology of avian coronaviruses in wildlife (Chu et al 2011). IBV-like coronaviruses have been detected in healthy galliform and nongalliform birds but not in psittacines (Hughes et al 2009; Jackwood et al 2010). Hughes et al (2009) sampled 46 species of wild birds in northern England and detected coronavirus RNA only in wildfowl (*Anseriformes*) and waders (*Charadriiformes*). This finding was corroborated by Chu et al (2011) and Muradrasoli et al (2010). Cardoso et al (2011) sampled captive exotic birds in Brazil and also failed to find coronaviruses in psittacine species.

Other studies have detected novel coronaviruses in wild birds (Jonassen et al 2005; Woo et al 2009). Gough et al (2006) isolated a coronavirus that is genetically distinct from alpha-, beta-, and gammacoronaviruses from a green checked Amazon parrot and provisionally named it *Parrot Coronavirus* (PaCoV). This is the only report of the isolation and characterisation of a coronavirus from psittacine birds and the aetiological role of this virus is unclear (Gough et al 2006). In a previous study, Hirai et al (1979) described the detection of a coronavirus-like agent in the livers of two Amazonia species of parrots. However, subsequent studies by the same authors proved this to be invalid (Hirai et al 1982). Additional novel coronaviruses that are genetically similar to the parrot coronavirus have been subsequently detected in mammals and birds (Dong et al 2007; Woo et al 2009). Viruses of this novel lineage have been proposed to form a new genus, provisionally named *Deltacoronavirus* (Chu et al 2011).

8.1.5. Hazard identification conclusion

There has been a single report of the isolation of a novel coronavirus from a green cheeked Amazon parrot (Gough et al 2006). The isolation was from a suspected case of proventricular dilation disease and there was no indication of disease caused by the coronavirus. Coronaviruses are frequently isolated from healthy wildlife and do not have a clear pathogenic role. The avian coronaviruses responsible for disease in chicken, turkeys, and pheasants have not been reported in psittacines. Therefore coronavirus species are not identified as a hazard in live psittacines.

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9. Influenza virus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Influenzavirus A is subtyped on the basis of serologic reactions to the haemagglutinin (subtypes H1 - 16) and neuraminidase (subtypes N1 - 9) surface glycoproteins (Spackman 2008). The distribution of virus subtypes varies by year, geographic location, and host species (Swayne and Halvorson 2008).

Strains of avian influenza (AI) are commonly separated into highly pathogenic strains (HPAI) and low pathogenic strains (LPAI). All HPAI virus isolates have been subtypes H5 or H7 but not all H5 or H7 isolates have been highly pathogenic. For regulatory purposes, the main basis for differentiation on HPAI and LPAI strains has been pathogenicity in susceptible chickens (OIE 2011).

9.1.2. OIE list

HPAI and LPAI in poultry are OIE listed diseases.

Article 10.4.1 of the current OIE *Code* (OIE 2011) states that, for the purposes of international trade, avian influenza in its notifiable form (NAI) is defined as an infection of poultry caused by any influenza A virus of the H5 or H7 subtypes or by any AI virus with an intravenous pathogenicity index (IVPI) greater than 1.2 (or as an alternative at least 75% mortality). NAI viruses can be divided into highly pathogenic notifiable avian influenza (HPNAI) and low pathogenicity notifiable avian influenza (LPNAI):

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

LPNAI are all influenza A viruses of H5 and H7 subtype that are not HPNAI viruses.

9.1.3. New Zealand status

Influenzavirus A in birds is listed as a notifiable organism (Tana et al 2011).

Avian influenza viruses of types H1N3, H4N6, H6N4, H11N3 and H5N2 have been isolated from healthy wild mallard ducks in New Zealand (Austin and Hinshaw 1984; Stanislawek 1990; Stanislawek 1992; Stanislawek et al 2002). The H5N2 virus was of low pathogenicity (Stanislawek et al 2002). Since 2004, MPI, in conjunction with the New Zealand Fish and Game Councils, the Department of Conservation and other stakeholders, has carried out surveillance for avian influenza on targeted migratory birds at Miranda, their main North Island arrival site (Stanislawek et al 2011). This surveillance has identified H1, H2, H4, H7, H10, and H11 subtypes and all the isolates have been determined to be low pathogenic strains (Tana et al 2007; Stanislawek et al 2011). In 2007, a further 34 AI isolates were recovered

from resident waterfowl, including two of the H5 subtype. These H5 isolates were determined to be low pathogenic H5N1 strains (Frazer et al 2008; MAF 2008).

A survey of domestic poultry found no evidence of antibodies to H5 or H7 AI subtypes (Tana et al 2007). Three free-range layer farms each had one positive reactor to the H5 subtype and follow-up investigations indicated historic exposure on one of these properties with no evidence of ongoing virus circulation (Tana et al 2007). Surveys of commercial duck, pheasant, quail, and turkey producers found no evidence of avian influenza viruses (Frazer et al 2008). A 2003 survey of domestic and feral pigeons found no evidence of AI infections (Black 2004). No AI viruses have been recovered from psittacines in New Zealand.

9.1.4. Epidemiology

AI viruses have a world-wide distribution and a broad host range. The overwhelming majority of isolates have come from waterfowl in the orders Anseriformes (ducks, swans, and geese) and Charadriiformes (shorebirds, gulls, and terns) (Swayne and Suarez 2000; Swayne and Halvorson 2008). Waterfowl are considered to be the biological and genetic reservoirs of all AI viruses and the primordial reservoir of all influenza viruses for avian and mammalian species (Webster et al 1992; Stallknecht 1998; Perdue et al 2000). Maintenance of viruses in these populations is aided by water becoming heavily contaminated during periods of congregation (Stallknecht and Shane 1988; Suarez 2000; Alexander et al 2003). Surveillance studies have found infection rates in passerine birds to be comparable with those in Charadriiformes (Capua et al 2005) and it has been suggested that passerines might also be reservoir hosts for some subtypes of AI (Ibrahim et al 1990; Papparella et al 1993; Panigrahy et al 2003). These natural hosts act as sources of infection for other species.

Wild psittacines are not considered to be natural hosts of AI viruses and most psittacine isolates have originated from captive birds (Imada et al 1980; Senne et al 1983; Stallknecht and Shane 1988; Alexander 2000; Panigrahy et al 2003; Hawkins et al 2006; Kaleta et al 2007; Pillai et al 2008). The HPAI H5N1 strain that originated in Southeast Asia has caused mortality in more than 60 wild bird species, including budgerigars (Perkins and Swayne 2003; Isoda et al 2006; EFSA 2008). Reports on HPAI viruses, aside from the Asian lineage H5N1 HPAI viruses, in wild birds have been limited to the isolation of H5N3 influenza virus from a common tern (*Sterna hirundo*) in South Africa in 1961, and H7N7 influenza virus from mallard ducks (*Anas platyrhynchos*) and mute swans (*Cygnus olor*) in close proximity to affected farms during the 2003 outbreak in the Netherlands (EFSA 2008).

Galliformes represent an abnormal host for influenza infection (Suarez and Schultz-Cherry 2000). AI is rare in commercial integrated poultry systems in developed countries but, when infection does occur, it can spread rapidly throughout the integrated system, resulting in epidemics of HPAI or LPAI (Swayne and Halvorson 2008).

Several domestic mammals, including pigs, cats, and dogs are susceptible to infection with H5N1 HPAI viruses under natural and experimental conditions (EFSA 2008). However, in natural infections, pigs and dogs appear to be dead end hosts and none of them have been implicated in the transmission of H5N1 to humans or to domestic birds. In most experimental studies in domestic mammals, H5N1 failed to spread between animals or from one animal species to another. In cats, however, it has been suggested that spread between cats and from cats to poultry may occur (FAO 2006; EFSA 2008). LPAI has also been associated with epidemics of respiratory disease in mink (Englund et al 1986), seals (Lang et al 1981; Webster et al 1981; Geraci et al 1982; Callan et al 1995), and whales (Lvov et al 1978;

Hinshaw et al 1986a). In a number of these cases, exposure to infected sea birds was suggested as the most likely source of virus.

Wild birds, particularly migratory waterfowl, may play a major role in the initial introduction of AI viruses into commercial poultry (Halvorson et al 1985; Hinshaw et al 1986b) but once established in commercial poultry, wild birds have very little or no role in secondary dissemination (Nettles et al 1985).

AI virus replicates in the respiratory, intestinal, renal, and reproductive organs and virus is excreted from the nares, mouth, conjunctiva, and cloaca of infected birds (Swayne and Halvorson 2008). The virus can be transmitted by the oral or oronasal routes and is spread by direct contact, aerosols, or fomites (EFSA 2008). However, air sampling during the 1983-84 HPAI outbreak in the northeastern United States did not recover virus from samples taken more than 45m downwind of an infected flock, suggesting airborne transmission is likely to be much less significant for transmission between farms than mechanical movement on fomites (Brugh and Johnson 1987). Long term carriers do not exist (Swayne and Halvorson 2008).

The incubation period of the disease is from 3 days in naturally-infected individual birds, up to 14 days for a flock (Swayne and Halvorson 2008). The *Code* gives the incubation period for the purposes of international trade as being 21 days (OIE 2011).

Most AI infections of psittacines have resulted in death before clinical signs were noticed. However, where disease signs were seen they included loss of condition, ruffled feathers, green diarrhoea, and nervous signs (Ritchie 1995; Alexander 1993).

LPAI infection of domestic poultry can result in mild to severe respiratory signs including coughing, sneezing, rales, rattles, and excessive lacrimation. Generalised clinical signs such as huddling, ruffled feathers, depression, lethargy, and, occasionally, diarrhoea have also been described. High morbidity and low mortality are normal for LPAI infections (Swayne and Halvorson 2008).

In contrast, most cases of HPAI infection of domestic poultry are associated with severe disease, with some birds being found dead before clinical signs are noticed. Clinical signs such as tremors, torticollis, and opisthotonus may be seen for 3-7 days before death. Precipitous drops in egg production are reported. Morbidity and mortality are usually very high (Swayne and Halvorson 2008).

Viral isolation in embryonated eggs is the preferred method for diagnosis due to its high sensitivity (EFSA 2008). Virus isolation can be performed by egg inoculation with oropharyngeal and cloacal swabs from live birds, followed by testing for haemagglutination activity (OIE 2009). The presence of influenza A virus is then confirmed using either an agar gel immunodiffusion test or an enzyme-linked immunosorbent assay (ELISA). Further subtyping of isolates can then be carried out using highly specific antisera or polyclonal antisera raised against a battery of intact influenza viruses. Alternatively, RT-PCR techniques are available to detect the presence of virus genome and the presence of NAI can be confirmed using specific primers (EFSA 2008).

The serological tests currently available are not all able to distinguish between sublineages or HP and LP strains (EFSA 2008). Agar gel immunodiffusion, haemagglutination, and haemagglutination inhibition tests and ELISAs have been developed (Swayne and Halvorson

2008; OIE 2009). Positive serological findings give no indication of current infection status as antibodies are unlikely to be detected until at least 7 days following infection (EFSA 2008). However, serology may be used as a component of a surveillance programme to demonstrate country, zone, or compartment freedom.

9.1.5. Hazard identification conclusion

HPAI and LPAI strains of avian influenza virus are distributed worldwide and psittacines are susceptible to infection. Given the wide range of LPAI viruses that have been described in New Zealand, LPAI is not identified as a potential hazard in imported live psittacines. New Zealand is free from all strains of HPAI. HPAI is identified as a potential hazard in live psittacines.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

AI virus has rarely been isolated from psittacine birds (Stallknecht and Shane 1988; Karesh et al 1997; Alexander 2000; Suarez 2000; Deem et al 2005; Stone et al 2005; Kaleta et al 2007; Swayne and Halvorson 2008) and psittacine birds are unlikely to act as reservoir hosts. The majority of infections in caged birds are in passerines (Fukushi et al 1982; Ibrahim et al 1990; Alexander 2000; Capua et al 2005; Kaleta et al 2005). The most likely sources of AI infection for psittacine birds are other birds, particularly passerines, in close proximity during international trade, quarantine, or in pet shops.

However, since the virus has been isolated from psittacines, and birds may be subclinically infected and thus remain undetected during routine clinical examination, the likelihood of entry of avian influenza virus with live psittacines is assessed to be low.

9.2.2. Exposure assessment

Imported psittacines may be integrated into aviaries or may contact other birds at shows or pet shops. The Department of Conservation exercises control over private holdings of native psittacine birds, which will reduce the likelihood of introduced exotic species mixing with captive native birds. However, native birds kept in captivity in contact with imported birds may be at risk. The likelihood of exposure of captive birds from infected imported psittacines is therefore assessed to be non-negligible.

Although imported psittacine birds are likely to be held in aviaries or cages, the enclosures in which they are kept may not preclude contact with wild birds. Escapes or illegal releases are not uncommon and would provide another exposure pathway. Additionally, aviary waste products may be disposed of at sites accessible to wild birds. The likelihood of exposure of wild birds to avian influenza virus from infected imported psittacines is therefore assessed to be non-negligible.

Wild birds have been implicated in the geographical expansion of HPAI virus outbreaks across Asia, the Middle East, Europe, and Africa (EFSA 2008). However, surveillance of wildlife during an H5N2 outbreak in poultry in the United States indicated there was limited transmission of virus from domestic poultry to wild birds and that wild birds had a very limited role in disease dissemination during the outbreak (Nettles et al 1985; Hinshaw et al 1986b).

Recommended minimum biosecurity standards for domestic producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures ensure that the likelihood of commercial poultry being exposed to free-living avian species are very low. However, the introduction of AI viruses to commercial poultry by migratory waterfowl has been documented (Halvorson et al 1985) so the likelihood of exposure of commercial poultry from free-living avian species is assessed as non-negligible.

9.2.3. Consequence assessment

Wild birds frequently carry AI viruses. However, mortalities are generally sporadic and outbreaks of disease are rare (Hansen 2006; EFSA 2008). Infection of wild birds with HPAI usually produces no mortality or morbidity (Swayne and Halvorson 2008) although recent H5N1 HPAI viruses have been associated with deaths in a number of wild bird species in Asia (Ellis et al 2004; Chen et al 2005; Sims et al 2005; Webster et al 2005). The impact on native bird species in New Zealand cannot, therefore, be predicted with any degree of confidence.

The introduction of HPAI in domestic poultry could result in widespread disease with high mortalities leading to disruption of the poultry industries and export trade in poultry products. The direct and indirect economic costs associated with H5N1 HPAI in Asia from late 2003 to mid 2005 have been estimated to exceed US$ 10 billion (Swayne and Halvorson 2008).

Sporadic cases of AI infection of humans have been documented and between 2003 and March 2012 the cumulative number of human infections was 596 with 350 deaths (World Health Organisation 2012). Human cases typically present with conjunctivitis, respiratory illness, or flu-like symptoms. Recent Asian H5N1 human cases have been closely associated with exposure to infected poultry in live poultry markets or villages (Swayne and Halvorson 2008). However, serological surveys of humans in four Thai villages (Dejpichai et al 2009) and a Cambodian village (Vong et al 2006) found no evidence of neutralising antibodies to H5N1 despite frequent direct contact with poultry likely to be infected with this virus, suggesting that the transmission potential from poultry to humans is likely to be low (Swayne and Halvorson 2008).

The introduction of HPAI in the commodity would be associated with non-negligible consequences to pet birds, the New Zealand poultry industries, wildlife and human health. The consequences are assessed to be high.

9.2.4. Risk estimation

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

9.3. RISK MANAGEMENT

9.3.1. Options

Article 10.4.6 of the current OIE *Code* (OIE 2011) recommends that for the importation of live birds other than poultry, regardless of the NAI status of the country of origin, veterinary authorities should require the presentation of an international Veterinary Certificate attesting that:

1. on the day of travel, the birds showed no clinical sign of infection with a virus which would be considered NAI in poultry;
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 travel and showed no clinical sign of infection with a virus which would be considered NAI in poultry during the isolation period;
3. a statistically valid sample of the birds, selected in accordance with the provisions of Article 10.4.29., was subjected to a diagnostic test within 14 days prior to travel 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;
5. if the birds have been vaccinated against NAI, it has been done in accordance with the provisions of the Terrestrial Manual and the nature of the vaccine used and the date of vaccination have been attached to the certificate.

Additionally, Article 10.4.9 of the current OIE *Code* (OIE 2011) recommends that for the importation of day-old live birds other than poultry; regardless of the NAI status of the country of origin, veterinary authorities should require the presentation of an international veterinary certificate attesting that:

1. on the day of travel, the birds showed no clinical sign of infection with a virus which would be considered NAI in poultry;
2. the birds were hatched and kept in isolation approved by the Veterinary Services;
3. the parent flock birds were subjected to a diagnostic test at the time of the collection of the eggs to demonstrate freedom from infection with NAIV;
4. the birds are transported in new or appropriately sanitized containers;
5. if the birds or parent flocks have been vaccinated against NAI, it has been done in accordance with the provisions of the Terrestrial Manual and the nature of the vaccine used and the date of vaccination have been attached to the certificate.

According to the *Code*, a country, zone, or compartment may be considered free from HPNAI when it has been shown that HPNAI infection has not been present for the past 12 months, although its LPNAI status may be unknown or, when, based on surveillance in accordance with Articles 10.4.27 to 10.4.33, it does not meet the criteria for freedom from NAI but any NAI virus detected has not been identified as HPNAI virus. If an outbreak of HPNAI occurs in a country previously recognised as free, under the OIE criteria HPNAI-free status can be regained 3 months after a stamping-out policy is applied, providing that surveillance in accordance with Articles 10.4.27 to 10.4.33 has been carried out during that three month period. However, as HPNAI freedom only considers the health status of poultry, restricting imports to countries free from HPNAI as described by the OIE *Code* would not effectively manage the risk associated with psittacines.

The OIE *Manual* (OIE 2009) describes both virus isolation and serological tests for the diagnosis of HPNAI.

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

Option 1

Birds to be imported could:

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

i. 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
ii. 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 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 tested 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. This would obviously be the most expensive option.

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10. Psittacid herpesvirus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Family: *Herpesviridae,* Subfamily: *Alphaherpesvirinae,* Genus: *Iltovirus.*

There are two recognised species in this genus: *Psittacid herpesvirus 1* (PsHV-1) (Pacheco's disease virus) and *Gallid herpesvirus 1* (infectious laryngotracheitis virus) (Davison 2010; Pellett et al 2012). *Gallid herpesvirus 1* is a disease of chickens and does not affect psittacine species.

Two novel psittacine herpesviruses have recently been described, one in African grey parrots and a blue and gold macaw (designated psittacid herpesvirus 2) (Styles et al 2005; Tomaszewski et al 2006) and another in a Bourke parakeet (designated Bourke herpesvirus) (Shivaprasad and Tomaszewski 2007).

Amazon tracheitis virus and parakeet herpesvirus are rare pathogens of Amazon parrots and parakeets respectively. Definitive information about these diseases is scarce and the viruses have not been classified by the International Committee on Taxonomy of Viruses (Pellett et al 2012).

10.1.2. OIE list

Not listed

10.1.3. New Zealand status

There remains some uncertainty surrounding New Zealand's status in regard to these viruses. Testing using PCR consensus primers and active surveillance of exotic parrots is needed before the absence of PsHV in New Zealand can be definitively established (Gartrell et al 2009).

A herpes virus resembling PsHV-1 was recovered from psittacine birds in the mid 1970s (Durham et al 1977). In 1997 Pacheco's disease was diagnosed in imported birds in New Zealand quarantine (Thornton and Stanislawek 2003). Subsequently a number of these birds were illegally moved into private collections. Some of these missing birds were traced, but most were never recovered and their disease status remains unknown. An extensive investigation did not reveal any evidence that the disease was introduced as a result of this incident (Loth 2003; Thornton and Stanislawek 2003).

In 2001 a serological survey was carried out for a number of diseases in a range of New Zealand birds, including 26 wild parrots and 70 captive parrots. Testing carried out in the USA resulted in one positive PsHV-1 virus neutralisation test out of 19 samples taken from one aviary. However, several months after the initial sampling, the bird that returned the positive serological test was resampled (blood and cloacal swab) and both serological and cultural tests were negative (Loth 2003). It has been speculated the Australian species of parrots may have co-evolved with a different PsHV which would not be detected by the methods used in these investigations (Gartrell et al 2009).

Pacheco's disease (PD) is associated with internal papillomatosis disease (IPD) which has been described in a sulphur-crested cockatoo in New Zealand (Gartrell et al 2009). The authors considered that this case of IPD could represent a neoplastic change independent of infectious agents or that it was caused by an undetected herpesvirus.

For the purposes of this risk analysis, Pacheco's disease virus is assessed to be exotic.

10.1.4. Epidemiology

Psittacid herpesvirus 1 (PsHV-1) is classified into four genotypes (genotypes 1, 2, 3, and 4) based on the UL16 gene sequence (Tomaszewski et al 2001, 2003; Orosz 2008). All four genotypes have the potential to cause PD but the susceptibility to each genotype varies between bird species (Tomaszewski et al 2003; Katoh et al 2011). For example, Amazon parrots are most commonly diagnosed with PD and have been identified with all 4 PsHV-1 genotypes, with genotypes 1, 2, and 3 considered highly pathogenic in these species; Pacific species such as cockatoos and cockatiels are relatively resistant to PD but can be infected with all four genotypes; macaws and conures are most commonly affected by genotype 4, rarely by genotype 3, and never by genotypes 1 and 2; and African grey parrots have been affected by genotypes 2, 3 and 4, but not genotype 1 (Tomaszewski et al 2006; Orosz 2008; Katoh et al 2011). Concurrent infection with more than one genotype is possible. There is a single confirmed case and several suspected cases of PD in non-psittacine species (Tomaszewski et al 2006).

Pacheco's disease has been described in many countries including the USA , Korea (Kwon et al 2003), Hungary (Bistyak et al 2004), Germany (Mutlu et al 1991), Greece (Iordanides et al 1994), Japan (Tsai et al 1993), Great Britain (Gough and Alexander 1993), Spain (Gomez-Villamandos et al 1991), Poland (Szeleszczuk et al 1990), and the Netherlands (Westerhof et al 1988). PsHV-1 has been isolated from green winged macaws in Australia (Gallagher and Sullivan 1997) although PD has never been reported (Raidal et al 1998; Ladds 2009).

Pacheco's disease is characterised by focal hepatic necrosis, with associated inflammatory lesions, and death. Mortality rates amongst birds showing clinical signs are usually high. Disease has often been associated with stress and occurs in susceptible populations crowded together in aviaries and quarantine facilities (Durham et al 1977; Gaskin et al 1978; Gough and Alexander 1993; Ritchie 1995; Thornton and Stanislawek 2003; Bistyak et al 2004).

PsHV-1 is transmitted by the faecal-oral route, generally by direct contact but also by aerosol or faecal contamination of food or water. Secretions from the oral cavity, regurgitated crop contents, and faeces are potentially infectious (Gravendyck et al 1998). Excreted virus may remain viable for long periods, and transmission via fomites is possible (Young 1995).

Limited experimental evidence suggests that the incubation period may be as short as a few days (Ramis et al 1996). Viraemia results in widespread virus dissemination to multiple tissues and organs including the integument, muscular, respiratory and circulatory system, bone marrow, the nervous system, thyroid and adrenal glands, spleen and liver, the urogenital tract, and the gastro-intestinal tract (Gravendyck et al 1998; Tomaszewski et al 2006). Apart from haematogenous dissemination, psittacine herpesviruses may spread along peripheral nerves to the brain (Gravendyck et al 1998).

Recovered birds may remain latently infected and shed PsHV-1 intermittently over many years (Ritchie 1995; Tomaszewski et al 2006). Despite this, the transmission rate to birds in

close contact with latently infected birds has been found to be low (Tomaszewski et al 2006). This suggests that virus shedding is infrequent or in low concentrations.

Affected birds show non-specific clinical signs, such as drowsiness, lethargy, anorexia, intermittent diarrhoea, polyuria, and polydypsia. Occasionally, affected birds may show conjunctivitis, sinusitis, haemorrhagic diarrhoea, and tremors. Gross and microscopic lesions include hepatitis, splenitis, and enteritis (Shivaprasad and Tomaszewski 2007).

The intensity and course of clinical disease vary according to species and susceptibility (Luppi et al 2011). Some birds survive the acute manifestations of the disease and become latently infected, whereas others become latently infected following an inapparent infection (Grund and Schlippenbach 2002 as cited in Tomaszewski et al 2006; Styles et al 2004).

Birds that that are persistently infected with PsHV-1 may go on to develop internal papillomatosis disease (IPD) (Styles et al 2004; Orosz 2008). IPD is characterised by progressive development of papillomas in the oral and cloacal mucosa as well as in the upper gastrointestinal tract (Johne et al 2002). The aetiology of IPD is not completely understood. All the evidence to date strongly suggests that PsHVs play an essential role in the development of mucosal papillomas and explain their apparent infectious nature (Goodwin and McGee 1993; Phalen et al 1997; Johne et al 2002; Styles et al 2004). Birds that survive acute systemic PsHV infections have been seen to develop mucosal papillomas and birds with mucosal papillomas have been shown to have circulating antibody to PsHVs (Phalen et al 1997). The prevalence of PsHV infection in parrots with mucosal papillomas is higher than that seen in parrots without them (Grund and Schlippenbach 2002 as cited in Styles et al 2004). PsHV DNA had been consistently demonstrated in mucosal papillomas (Johne et al 2002; Styles et al 2004; Legler et al 2008; Katoh et al 2011). There is no evidence to suggest that a papillomavirus is responsible for these lesions (Styles et al 2004, 2005).

Mucosal papillomas have been described in many species of captive neotropical psittacine birds, with the highest incidence occurring in macaws (*Ara* spp.), Amazon parrots (*Amazona* spp.), and conures (*Aratinga* spp.) (Johne et al 2002; Styles et al 2004). It has been speculated that some psittacine species may be resistant to infection (Legler et al 2008) resulting in the dissemination of mucosal papillomas without an ensuing outbreak of Pacheco's disease (Styles et al 2004).

Mucosal papillomas occur primarily in the oral cavity and cloaca and less frequently in the crop, oesophagus, proventriculus, ventriculus, and conjunctiva (Styles et al 2004). Depending on the location of the lesions, birds may show straining, bloody droppings, wheezing, vomiting, weight loss, dysphagia, or death (Katoh et al 2011). Some birds do not show any clinical signs initially but develop problems if stressed by other illness or environmental factors. Birds with papillomatosis tend to develop neoplasias of the pancreas or liver (Styles et al 2004; Katoh et al 2011). The disease has an extremely poor prognosis (Phalen et al 1997).

Outbreaks of disease are primarily observed when carrier birds are introduced in captivity or when birds are captured and transported (O'Toole et al 1992; Luppi et al 2011). It is therefore suggested that birds should be diagnosed while in quarantine (Luppi et al 2011).

The majority of parrots infected with PsHV-1 will have DNA virus in or on the membranes of their oral and cloacal mucosae (Tomaszewski et al 2006). Molecular detection of Pacheco's disease by PCR is highly sensitive and specific for the virus. Subclinically infected birds can

be detected with a nested PCR or real time PCR assay for PsHV-1 using swabs from the oral cavity and cloaca. PsHV-1 DNA can also be found in blood in some birds. However, oral and cloacal swabs are better sources of PsHV-1 DNA. Genotypes 1, 2, 3, and 4 are detected but not differentiated by PCR (Tomaszewski et al 2003). Styles et al (2004) report that the 23F primer set is highly sensitive but more recently Luppi et al (2011) demonstrated that the primers P-UL17/16 were more sensitive still. Virus excretion in latent carriers is intermittent and virus isolation techniques cannot be relied upon for diagnosis (Ritchie 1995). Likewise serological testing is insensitive for individual birds as the high antibody titres which may be found in acutely infected birds decline to low levels in latent carriers. Serology may, however, be useful for flock testing. Mucosal papillomatosis can be diagnosed by histology, PCR of biopsies, or scrapings.

Currently there is only one commercial PsHV-1 vaccine, derived from a single, unreported, serotype and is therefore of limited use (Tomaszewski et al 2006).

10.1.5. Hazard identification conclusion

Psittacine herpesvirus 1 causes a serious disease of psittacine birds and may be carried by asymptomatic birds. PsHV-1 is therefore identified as a potential hazard in live psittacines.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

PsHV-1 may be carried as a subclinical and latent infection in psittacine birds. These infections are unlikely to be detected during routine clinical examination. The likelihood of entry of PsHV-1 with live psittacines is therefore assessed to be non-negligible.

10.2.2. Exposure assessment

Virus shedding may occur in stressed infected birds. Excreted virus may remain viable for long periods, and transmission via fomites is possible. Imported psittacines may be integrated into aviaries or may contact other birds at shows or pet shops. Close contact with other psittacines may facilitate transmission via the faecal-oral route. The risk of exposure of wild native birds to PsHV-1 through imported psittacines is low, given the isolated nature of most native psittacine populations, and the close contact required for transmission of the virus. The Department of Conservation exercises control over private holdings of native psittacine birds, which will reduce the likelihood of introduced exotic species mixing with captive native birds. However, native birds kept in captivity in contact with imported birds may be at risk and escapes or illegal releases are not uncommon and would provide another exposure pathway. The likelihood of exposure of PsHV-1 is therefore assessed to be non-negligible.

10.2.3. Consequence assessment

PsHV-1 is highly host specific although the susceptibility of psittacines varies between species. Therefore there would be negligible consequences for species other than psittacines, including humans. PsHV-1 is not highly contagious and there has been no evidence of transmission or establishment of the 1977 or 1997 isolates of virus in feral or caged birds in New Zealand. Pacheco's disease is of significance in situations where birds are crowded and stressed in unnatural environments, therefore the likelihood of the virus establishing in populations of native birds in the wild is unknown but assessed to be low.

The introduction of the virus into established collections of non-native psittacines could result in disease and mortalities amongst these birds. Serious outbreaks have been reported in a variety of psittacine species. Therefore the consequences of entry and establishment of PsHV-1 are assessed to be non-negligible.

10.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result, the risk estimate for PsHV-1 is non-negligible and it is classified as a risk in live psittacines. Therefore risk management measures can be justified

10.3. RISK MANAGEMENT

10.3.1. Options

The following factors were considered when drafting options for the effective management of PsHV-1 in live psittacines:

• Clinical examination of birds is unlikely to detect latent carriers unless they present with mucosal tumours.

• Quarantining of birds may precipitate outbreaks of disease if latent carriers are included in the group. If birds die in quarantine the disease can be diagnosed by post mortem examination and testing of tissue samples. Therefore, quarantining of birds could be useful as a possible indicator of infection.

• The vast majority of parrots infected with PsHV-1 will have virus DNA in or on the mucous membranes of their oral and cloacal mucosae and these parrots can be detected by a PCR assay of scrapings or swabs of these surfaces. To consistently detect these parrots, however, it is necessary to submit samples obtained from both the oral and cloacal mucosae (Tomaszewski et al 2006).

• Importation from flocks that are regularly inspected and have never had confirmed cases of Pacheco's disease are likely to be safe sources from which to import.

One or a combination of the following options could be used for the effective management of PsHV-1 in live psittacines:

Option 1

Birds could be imported from countries that are free from psittacid herpesvirus, Pacheco's disease, and internal papillomatosis disease.

Option 2

Birds to be imported could be sourced from closed flocks in which psittacid herpesvirus, Pacheco's disease and internal papillomatosis disease have not previously been reported.

Option 3

Birds to be imported could be subjected to suitable PCR tests on cloacal and oral swabs within 7 days of travel, by a laboratory approved by the veterinary authority of the exporting country, with negative results.

Option 4

Birds to be imported could be quarantined for at least 28 days and be subjected to suitable PCR tests on cloacal and oral swabs after 3 weeks, with negative results. Birds that die while in quarantine could be submitted to a post mortem examination and suitable samples submitted to an approved laboratory for examination, by histology and PCR.

Option 5

Birds with clinical evidence of mucosal papillomas should not be imported.

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11. Psittacine poxvirus

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Species: Psittacinepox virus (PSPV) (Skinner et al 2012) is antigenically distinct from other avian pox viruses (Ritchie 1995; Lüschow et al 2004; Jarmin et al 2006).

11.1.2. OIE list

Not listed.

11.1.3. New Zealand status

Avipox virus infections are common in many avian species in New Zealand (Alley 2002). An outbreak of PSPV was reported in rosellas in Auckland in 2002. Although all at-risk birds were euthanased, the source of the infection could not be traced and MAF was not able to declare that the event was fully contained (King et al 2003). No further cases of PSPV have been reported and a recent study found no evidence of psittacine poxvirus in New Zealand birds (Ha et al 2011). For the purposes of this risk analysis, Psittacinepox virus is regarded as an exotic organism.

11.1.4. Epidemiology

Avian pox is a highly contagious disease of worldwide distribution that affects commercial poultry, domestic pets, and free-living birds of many species (Gonzalez-Hein et al 2008; Catroxo et al 2009). At least 232 avian species in 23 orders have been reported to have acquired a natural pox infection (Bolte et al 1999; Jarmin et al 2006). PSPV infections have been reported in several psittacine species although susceptibility appears to vary between species (Gonzalez-Hein et al 2008; Katoh et al 2010).

PSPV may be transmitted by direct contact or indirectly through infected objects or insect vectors (e.g. mosquitoes, midges, or flies), which are the primary sources of disease exposure (Gonzalez-Hein et al 2008). Poxviruses are extremely resilient in the environment (King et al 2003).

Poxviruses have immunosuppressive effects and disease can occur in three forms; cutaneous, diphtheritic, or septicaemic (Greenacre 2005; Wang et al 2006; Catroxo et al 2009). The expression of disease varies according to strain of virus, route of infection and the species, age and condition of the affected bird.

Cutaneous disease is characterised by the formation of discrete nodules on unfeathered skin, such as the cere, around the eyes, and the feet (Gonzalez-Hein et al 2008). The nodules progress from papules to vesicles and erosions. They are susceptible to secondary bacterial and fungal infections that delay healing and sometimes cause depigmentation at the site. Disease can resolve within a month or persist for more than a year. Mortality is generally low (Hitchner and Clubb 1980; King et al 2003; Greenacre 2005). In psittacines, clinical signs include ocular discharge, rhinitis, and conjunctivitis, followed by the appearance of ulcerations on the medial canthi of the eyes (McDonald et al 1981; Boosinger et al 1982; van Riper et al 2002; Smits et al 2005; Katoh et al 2010).

The diphtheritic form is usually fatal. It is characterised by fibrinonecrotic lesions on mucous membranes, particularly in the mouth, trachea and oesophagus. These bleed profusely if damaged or removed. The reported incubation period ranges from four days to five or more weeks, but most birds develop lesions within seven to 14 days. In large flocks, outbreaks may last for several months. Clinical signs include serous ocular discharge, blepharitis, rhinitis, and conjunctivitis followed by ulceration of the eyelid margins. Dry crusty lesions appear 12 to 18 days after infection (King et al 2003; Greenacre 2005).

Avian pox may become systemic when diphtheritic lesions cause defects in the mucosal barrier of the alimentary and respiratory tract. Lesions may develop in the throat, gastro-intestinal tract, lungs, and air sacs and result in severe upper respiratory tract disease, anorexia, diarrhoea, and high mortality (Graham 1978; McDonald et al 1981; Boosinger et al 1982; Tsai et al 1997; King et al 2003; Greenacre 2005). In some cases there may be no or few outward signs, other than general depression, illness and death. Histopathological lesions include necrosis of the heart and liver, as well as air sacculitis, pneumonia, peritonitis, and accumulation of necrotic debris on the surface of the alimentary tract (Katoh et al 2010). Characteristic intracytoplasmic inclusion bodies (Bollinger bodies) may be noted in lesions in the mucosa of the sinuses, trachea, crop, oesophagus, or throat (Goodpasture and Anderson 1962).

PSPV has been shown to vary in virulence in different hosts. Amazon parrots appear to be most susceptible to PSPV infections and may develop a severe upper respiratory tract disease (Katoh et al 2010). Pionus, macaws, conures, parakeets, and lovebirds are also considered highly susceptible to infection; whereas cockatoos and cockatiels appear to be more resistant (Emanuelson et al 1978; McDonald et al 1981; Boosinger et al 1982; Ritchie 1995; Katoh et al 2010). Attempts to infect chickens and canaries with PSPV have been unsuccessful (Hitchner and Clubb 1980) and there is no evidence that the virus can affect mammals.

Some birds may recover from disease and continue to shed virus intermittently from feathers, skin, or the gastrointestinal tract. Incubation periods of more than one month have been observed (Catroxo et al 2009) and it is possible that birds may be asymptomatic carriers for the disease (Alley et al 2010).

11.1.5. Hazard identification conclusion

Psittacine pox virus is widely distributed in the world and has been associated with significant disease and mortality in psittacine species. There has only been one report of PSPV in caged rosellas in New Zealand in 2002 and there is no evidence that the virus has established here. Poxviruses of psittacines are distinct from poxviruses affecting poultry and other avian species. PSPV is therefore identified as a potential hazard in the commodity.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

Psittacine species vary in their susceptibility to infection with PSPV and the virus is not always pathogenic. The virus has a long incubation period and recovered persistent carriers of infection have been described. Asymptomatic birds are extremely unlikely to be detected during routine clinical examination. The likelihood of entry of psittacinepox virus with live psittacines is therefore assessed to be non-negligible.

11.2.2. Exposure assessment

Poxviruses are extremely resilient in the environment and PSPV may be transmitted by direct contact or indirectly through contaminated objects or insect vectors. Other avipox viruses are known to occur in both caged and wild birds in New Zealand. Imported psittacines may be integrated into aviaries and may contact other birds at shows or pet shops. Although imported psittacine birds are likely to be held in aviaries or cages the enclosures in which they are kept may not preclude contact with wild birds or movements of insect vectors. Escapes or illegal realeases are not uncommon and would provide another exposure pathway. Additionally aviary waste products may be disposed of at sites accessible to wild birds. The likelihood of exposure of caged and wild birds to psittacinepox virus from infected imported psittacines is therefore assessed to be non-negligible.

11.2.3. Consequence assessment

Psittacinepox virus infection has been associated with high mortality and outbreaks of disease in a variety of psittacine species. The susceptibility of native parrot species is not known. The consequences of entry and establishment of psittacinepox virus are therefore assessed to be non-negligible.

Psittacinepox virus does not infect poultry, or other avian species, or mammals and there would be negligible consequences for human health.

11.2.4. Risk estimation

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

11.3. RISK MANAGEMENT

11.3.1. Options

PSPV infection may be confirmed by PCR techniques (Katoh et al 2010) although PCR may only be reliable when active lesions are present and may not detect subclinical carriers (Gartrell 2012). Virus neutralisation, ELISA, hemagglutination-inhibition tests, virus isolation, and electron microscopy may also be used for the diagnosis of avian poxvirus infection (Gonzalez-Hein et al 2008; Catroxo et al 2009; Katoh et al 2010). Poxvirus vaccines are available for use in several avian species. However, PSPV is serologically unrelated to other avipox viruses, thus effective prevention against PSPV requires a species-specific vaccine (Winterfield and Reed 1985).

Psittacinepox virus is not listed by the OIE and there are no international standards for this virus. One or a combination of the following risk management options could be considered in order to effectively manage the risk:

Option 1

Birds to be imported could be required to be healthy and not show any signs suggestive of psittacine pox infection before or during quarantine.

Option 2

Birds to be imported could:

i. be isolated in quarantine for 4 weeks; and
ii. be free from clinical signs of disease before and during quarantine.

Any suspicious lesions could be examined by virus isolation, histology and electron microscopy, with negative results. Birds that die while in quarantine could be subject to post mortem examination and suitable samples submitted to an approved laboratory for examination by histopathology, electron microscopy, and virus isolation.

Option 3

Birds to be imported could:

i. be isolated in quarantine for 4 weeks; and
ii. be free from clinical signs of disease before and during quarantine; and
iii. be subjected to suitable serological tests and PCR after 3 weeks, with negative results.

Any suspicious lesions detected during the quarantine period could be examined by virus isolation, histology and electron microscopy, with negative results. Birds that die while in quarantine could be subject to post mortem examination and suitable samples submitted to an approved laboratory for examination by histopathology, electron microscopy, and virus isolation.

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12. Avian orthoreovirus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Numerous avian orthoreoviruses have been isolated from commercial poultry flocks, including chickens, Muscovy ducks, turkeys and geese, and several different serotypes described. Sequence diversity is extensive (Attoui et al 2012).

Some reoviruses recovered from companion and aviary birds have been shown to be related to strains found in poultry, whilst others have been shown to be serologically distinct (Ritchie and Carter 1995; van den Brand et al 2007).

12.1.2. OIE list

Not listed.

12.1.3. New Zealand status

Avian reovirus was first identified in New Zealand poultry in 1976 (Green et al 1976; Bains and Tempest 1978; Saifuddin et al 1989) and vaccination against this virus in poultry is not uncommon (Howell 1992; Anonymous 1999; Poland 2004; Frazer 2008).

Avian reovirus infections in psittacines have not been described in New Zealand. While there has been speculation that the occurrence of reovirus-related disease in Australian king parrots (*Alisterus scapularis*), exported from New Zealand to Italy, might have been due to infection of those birds prior to export (Conzo et al 2001), it is more likely that the birds exported from New Zealand were infected by African grey parrots (*Psittacus erithacus erithacus*) from Zaire held in the same Italian quarantine facility.

12.1.4. Epidemiology

Avian orthoreoviruses have been recovered from multiple avian host species in many countries (Ritchie and Carter 1995; Doyle 1997; Jones 2000; van den Brand et al 2007). Reovirus and reovirus-like infections have been reported worldwide in a number of psittacine birds (Lüthgen et al 1980; Senne et al 1983; Ashton et al 1984; Wilson et al 1985; Graham 1987; Ritchie and Carter 1995; Conzo et al 2001; Sanchez-Cordon et al 2002). Most reports of psittacine reovirus disease relate to birds recently moved, birds in pet shops, or birds in (or recently released from) quarantine (Meulemans et al 1983; Senne et al 1983; Ashton et al 1984; Wilson et al 1985; Graham 1987; Conzo et al 2001; Sanchez-Cordon et al 2002; Pennycott 2004; van den Brand et al 2007; Perpinan et al 2010).

Much is still unclear regarding the epidemiology and pathogenicity of reovirus infections in psittacines.

Although reovirus infections are found in different psittacine species, infection in New World Psittaciformes is suggested to be rare and these species are thought to be more resistant than the Old World parrots (Conzo et al 2001; Manvell et al 2004). However in some reports, both New World and Old World species showed high mortality (van den Brand et al 2007). Avian reoviruses do not infect mammals (Attoui et al 2012).

The exact route of transmission of reovirus in Psittaciformes is still unclear but it is likely to be transmitted horizontally by direct or indirect contact with infected faeces, as in poultry (Jones and Guneratne 1984; van den Brand et al 2007). Egg transmission has been documented to occur occasionally in ducks, geese, turkeys, and chickens (Menendez et al 1975). This route of transmission has not been described in companion birds (Ritchie and Carter 1995).

The incubation period is not known, but may be as short as 2 days or as long as 4 weeks (van den Brand et al 2007). It has been suggested that recovered birds may be carriers (van den Brand et al 2007; Jones 2008) but this has not been proven. There is no indication of the time during which birds could remain carriers, but circumstantial evidence suggests that the virus may be carried by subclinically infected birds at least for several months. Reoviruses are relatively resistant in the environment and survive well outside the host (Jones 2000).

Reoviruses are not always pathogenic and have frequently been recovered from asymptomatic birds (Hollmén and Docherty 2007; van den Brand et al 2007). Senne et al (1983) described reovirus infection in asymptomatic psittacines in quarantine. A few strains have been associated with disease in several species of gallinaceous birds, psittacine birds, and waterfowl (Ritchie and Carter 1995).

Reoviruses are suggested to have an immunosuppressive activity (van den Brand et al 2007). This immunosupression can predispose the host to other pathogens, including salmonellosis, aspergillosis and paramyxoviruses, and accounts for the diversity of syndromes associated with reoviruses (Conzo et al 2001; van den Brand et al 2007; Perpinan et al 2010). Experimental infections in African grey parrots could not reproduce all the clinical signs and gross lesions seen in the birds from which the virus had been recovered (Graham 1987); this suggests that some lesions found in birds with natural reovirus infections are the result of concurrent infections and not the result of the reovirus itself (Perpinan et al 2010).

Morbidity and disease signs vary among different species and are also affected by the age of the host and virus strain, and by the presence of stress factors and secondary pathogens (Conzo et al 2001; Attoui et al 2012). Chronic respiratory signs have been described in Amazon parrots; gastrointestinal signs, nasal discharge, and emaciation are common in African grey parrots; and non-specific signs (incoordination, emaciation, and diarrhoea) have been reported in cockatoos (Wilson et al 1985; Conzo et al 2001). Outbreaks of high mortality, splenomegaly, and hepatomegaly have been reported in budgerigars (Manvell et al 2004; Pennycott 2004).

Common pathological findings include splenomegaly, hepatic congestion, and enteritis. Histopathology frequently shows hepatic coagulation necrosis and less prominent necrosis in spleen and bone marrow (van den Brand et al 2007).

The prognosis for Old World psittacines is usually poor, especially for African grey parrots and cockatoos, whereas New World psittacines usually respond to treatment (Ritchie and Carter 1995).

Orthoreovirus group-specific antigen may be demonstrated with the agar gel precipitin, fluorescent antibody, and complement fixation tests and ELISA. Orthoreoviruses can be further classified antigenically using serotype-specific antisera, and differences among strains have been compared using genome sequence information (Hollmén and Docherty 2007).

Diagnosis is best achieved by virus isolation and identification (Hollmén and Docherty 2007) of cloacal swabs.

Van den Brand et al (2007) suggest that diagnosis of reovirus infections in live psittacine birds may be possible by demonstrating precipitating or virus-neutralising antibodies (van den Brand et al 2007). However, serology for detection of reovirus antibodies is complicated due to antigenic cross-reactions among serotypes, the ubiquitous nature of reoviruses, and the possibility of subclinical and asymptomatic infections (Vasserman et al 2004; Hollmén and Docherty 2007).

12.1.5. Hazard identification conclusion

Reoviruses of psittacines are distinct from reoviruses affecting poultry species. Avian reovirus has been associated with significant disease and mortality in psittacine species and has not been reported in New Zealand. Reoviruses are therefore identified as a potential hazard in the commodity.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

Reoviruses are not always pathogenic and have been recovered from asymptomatic psittacine birds in quarantine stations. Furthermore, clinically affected birds that have recovered from the disease may remain carriers of the virus. These birds are unlikely to be detected during routine clinical examination. The likelihood of entry of exotic avian reoviruses with live psittacines is therefore assessed to be non-negligible.

12.2.2. Exposure assessment

The epidemiology of avian reovirus infection in psittacines is not completely understood although it is likely to be similar to that for avian reovirus infection in chickens. Transmission is probably horizontal by direct or indirect contact with infected faeces. Outbreaks of disease have frequently followed the introduction of new birds or the movement of birds in existing collections. Imported psittacines may be integrated into aviaries and may contact other birds at shows or pet shops. The risk of exposure of wild native birds to avian reoviruses through imported psittacines is low, given the isolated nature of most native psittacine populations, and the close contact required for transmission of the virus. The Department of Conservation exercises control over private holdings of native psittacine birds, which will reduce the likelihood of introduced exotic species mixing with captive native birds. However, native birds kept in captivity in contact with imported birds may be at risk. The likelihood of exposure of pet birds to avian reovirus is therefore assessed to be non-negligible.

Although imported psittacine birds are likely to be held in aviaries or cages the enclosures in which they are kept may not preclude contact with wild birds. Additionally, aviary waste products may be disposed of at sites accessible to wild birds. Escapes or illegal releases are not uncommon and would provide another exposure pathway. The likelihood of exposure of wild birds to avian reovirus from infected imported psittacines is therefore assessed to be non-negligible.

12.2.3. Consequence assessment

Avian reovirus infection may cause high mortality in naïve flocks and serious outbreaks of disease have been reported in a wide range of psittacine species. The introduction of avian reovirus into established psittacine collections or into free living populations of native or introduced psittacines could result in disease and mortalities amongst these birds. The consequences of entry and establishment of avian reoviruses are therefore assessed to be non-negligible.

Avian reoviruses do not infect mammals and there are negligible consequences for human health.

12.2.4. Risk estimation

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

12.3. RISK MANAGEMENT

12.3.1. Options

Avian reovirus is not listed by the OIE and therefore there are no international standards for it. Serological tests have not been validated for use in psittacines. However, the virus cross reacts with chicken isolates therefore tests used for avian reovirus in poultry could be used.

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

Option 1

Birds could be imported if they have resided in a country, zone or compartment free from avian reovirus (psittacine strains) since they were hatched or for at least the past 28 days.

Option 2

Birds to be imported could be subjected to suitable serological tests and virus isolation on cloacal swabs within 7 days of travel by a laboratory approved by the veterinary authority of the exporting country, with negative results.

Option 3

Birds to be imported could be quarantined for at least 28 days and be subjected to suitable serological tests and virus isolation on cloacal swabs after 3 weeks, with negative results. Birds that die while in quarantine could be submitted to a post mortem examination and suitable samples submitted to an approved laboratory for examination, by histopathology, immunohistopathology, and virus isolation.

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13. Cutaneous papillomas

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Psittacus erithacus timneh papillomavirus (PePV) is associated with cutaneous papillomas in psittacines. Only one other avian papillomavirus has been identified (*Fringilla coelebs papillomavirus*) from chaffinches (Tachezy et al 2002; Bernard et al 2012).

Internal papillomatosis disease (IPD) is characterised by papillomas in the oral and cloacal mucosa and in the upper gastrointestinal tract (Johne et al 2002). The evidence strongly suggests that the causative agent is psittacine herpesviruses (PsHV) and there is no credible evidence to suggest that a papillomavirus is responsible for these lesions (Sundberg et al 1986; Goodwin and McGee 1993; Phalen et al 1997; Johne et al 2002; Styles et al 2004, 2005). IPD is considered with the herpesviruses in this risk analysis.

13.1.2. OIE list

Not listed

13.1.3. New Zealand status

There are no reports of cutaneous papillomas in psittacines in New Zealand.

13.1.4. Epidemiology

Papillomaviruses are highly host species-specific and tissue restricted (Bernard et al 2012). PePV has been associated with benign cutaneous lesions (papillomas) in African grey parrots (O'Banion et al 1992; Tachezy et al 2002; Johne et al 2003; Styles et al 2004; Bernard et al 2012) although reports are extremely rare. Little is known about the transmission of the condition, which is benign in nature and does not require treatment (Ritchie 1995).

The presence of PePV can be confirmed by immunohistochemistry using papillomavirus-specific antibodies or detection of virions with electron microscopy. Primers capable of amplifying PePV DNA have been developed, and PCR could be used to confirm the presence of this virus in suspect lesions (Phalen 2007).

13.1.5. Hazard identification conclusion

There is no evidence of a transmissible viral aetiology for papillomas in psittacine birds other than African grey parrots. Additionally the condition is benign and does not require treatment. Therefore cutaneous papillomas are not identified as a hazard in the commodity.

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14. Arboviruses

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Arboviruses replicate in bloodsucking arthropods and are transmitted by bite to a vertebrate host. Over 100 arboviruses have been isolated from avian species or ornithophilic vectors (Guy and Malkinson 2008). However, natural and experimental infection of psittacines with arboviruses other than West Nile virus (WNV) has not been described[1]. Therefore this chapter will only consider the species *West Nile virus*.

14.1.2. OIE list

West Nile fever is an OIE listed disease.

14.1.3. New Zealand status

Whataroa virus is the only recognised New Zealand arbovirus of birds (Maquire et al 1967) and this has never been recorded in psittacines. WNV is a notifiable organism (Tana et al 2011).

14.1.4. Epidemiology

WNV is a zoonotic mosquito-transmitted arbovirus with a wide geographical range. Genetic analysis of WNV isolates separates strains into two clades. Lineage 1 isolates are found in northern and central Africa, Israel, Europe, India, Australia (Kunjin virus), North and Central America, and Columbia and Argentina in South America. Lineage 2 strains are endemic in central and southern Africa and Madagascar, with co-circulation of both virus lineages in central Africa (OIE 2008). Strains from each lineage have been implicated in human and animal disease (OIE 2008).

Infectious adult mosquitoes carry the virus in their salivary glands and infect susceptible hosts while feeding on blood. Birds which attain a viraemia and retain it for sufficient duration to infect biting mosquitoes are regarded as competent hosts. In contrast, mammals, reptiles, amphibians, and some species of birds are incidental or dead-end hosts. Thus WNV is maintained in a mosquito–bird–mosquito transmission cycle (OIE 2008). Mammal infection requires mosquitoes that bite both birds and mammals (bridge vectors) (Spurr and Sandlant 2004). At least 75 species of mosquitoes from 11 genera and 8 species of ticks have been found infected with WNV (Spurr and Sandlant 2004).

WNV has been identified in more than 326 bird species and corvid species are known to serve as amplifying hosts (CDC 2009). There is an increasing number of reports of WNV in psittacines (Komar 2003; D'Agostino and Isaza 2004; Ellis et al 2005; Carboni et al 2008; Stockman et al 2010; Palmieri et al 2011) and experimental studies have shown viraemia following WNV inoculation (Komar et al 2003). Of the reports in psittacines, only one bird was diagnosed ante mortem (D'Agostino and Isaza 2004).

[1] A single report of an *Orbivirus* (family Reoviridae) in psittacines exists. However, the virus was never fully characterised and no information regarding a vector was provided at the time of the original report, nor has any emerged since (Hirai et al 1979).

Different avian species have been shown to be viraemic for differing periods of time and with different levels of viraemia (Stockman et al 2010). Komar et al (2003) experimentally infected 178 birds of 25 species (including six budgerigars and six monk parakeets) by mosquito bite. Viraemia was detectable 1-2 days after infection in all species of birds, peaked after approximately 2-4 days, and persisted for 7 days or less in all but one bird. The budgerigars and monk parakeets did not develop levels of viraemia considered to be sufficient to infect mosquitoes.

Komar et al (2003) calculated viral competence indices which reflect the relative number of infectious mosquitoes that would be derived from feeding on a single host. Competence depends on the concentration of infectious virus particles in blood and the duration of an infectious level viraemia. Although this study consisted of a limited number of birds and has not been investigated further (Stockman et al 2010), they determined psittacines to be incompetent hosts due to a low titre and short-lived (0-4 days) viraemia (Komar et al 2003).

Observations of bird-to-bird transmission in the absence of arthropod vectors strongly suggest that alternative modes of transmission exist. This is supported, at least in some species, by laboratory findings of faecal-shed WNV and contact transmission, and by WNV-positive results from oral and cloacal swabs (Komar et al 2003; Dawson et al 2007). The mode of this "cage-mate" transmission is unknown (Komar et al 2003). A bird must have a viraemia of sufficient level and duration for the virus to be shed in mucosal secretions of the oropharynx and cloaca (Carboni et al 2008). Natural transmission *per os* has not been reported in birds. However, the viraemia profiles generated by oral inoculation are comparable to those derived from mosquito-borne infection (Komar et al 2003). WNV can also be detected in vascular feather pulp and nonvascular feathers from a broad spectrum of bird species (Nemeth et al 2009) and horizontal transmission of WNV in flocks may occur due to cannibalism and feather-picking (Banet-Noach et al 2003).

Clinical signs of WNV infection in humans, horses, and birds are highly variable, ranging from mild flu-like signs to severe encephalomyelitis (Palmieri et al 2011). Most humans and horses that become infected do not develop any clinical signs (D'Agostine and Isaza 2004) although at the height of the US epidemic in 2003, 9,862 cases were reported in humans resulting in 264 deaths (CDC 2003). The disease in horses is frequently characterised by mild to severe ataxia, and encephalitis occurs only rarely (OIE 2008). The mortality rate is approximately one in three clinically affected horses (OIE 2008). The most common signs of infection in birds with WNV include anorexia, weakness, lethargy, and neurological signs (D'Agostino and Isaza 2004; Stockman et al 2010). WNV infection in psittacines appears to be capable of causing a chronic disease, characterised by severe chronic inflammatory lesions associated with poor nutritional state (Stockman et al 2010).

WNV infection in psittacines poses a diagnostic challenge because clinical signs and lesions are nonspecific and there is no definitive ante mortem diagnostic test. Haemagglutination inhibition (HI) and plaque reduction neutralisation (PRN) methods are most commonly used for identifying WNV antibody in avian serum (OIE 2008). The PRN test is the most specific and is applicable to any species (OIE 2008). The Vec-test MNV Antigen Assay used commonly as a surveillance tool is insensitive for non-corvid species (Nemeth et al 2007). An RT-PCR assay for WNV RNA can also be performed on oral and cloacal swabs and serum samples, but rapid viral clearance means these tests often have negative results (Stockman et al 2010). There is no ELISA suitable for use in psittacines.

Apart from supportive therapies there is no effective treatment for WNV in either animals or humans. Vaccines are not available for psittacine species.

14.1.5. Hazard identification conclusion

Psittacine birds are not highly susceptible to West Nile virus but can be infected. Although the study of Komar et al (2003) suggests infection of psittacines is followed by a short-lived low titre viraemia and they may not be competent hosts for WNV, extrapolating the results from this study to an entire Family of birds may be unjustifiable (Gartrell 2012). WNV is therefore identified as a hazard in live psittacines.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

There are an increasing number of reports of psittacines infected with WNV. Psittacines are considered to have minimal viral competence associated with the lack of oral or cloacal shedding and the low level and duration of viraemia (Komar et al 2003; Palmieri et al 2011). However, the conclusion of low susceptibility is based on a study of a limited number of birds in an artificial environment (Stockman et al 2010) and detailed experimental and field studies are lacking (Palmieri et al 2011). Furthermore, infections are often subclinical or associated with non-specific signs and may not be detected during an ante-mortem examination. Therefore the likelihood of introduction of WNV with imported live psittacines is assessed to be very low.

14.2.2. Exposure assessment

Spurr and Sandlant (2004) completed a thorough literature review and assessed the risk of WNV establishing in New Zealand. They concluded that, "if introduced into New Zealand, WNV is likely to become established and persist in both mosquito (and tick) vectors and vertebrate hosts (such as birds and mammals)".

In order for WNV to establish in New Zealand, a sufficient number of vectors must feed on an infectious host to ensure that some survive long enough to feed again on a susceptible reservoir host. Additionally, there must be enough competent vectors and competent hosts to maintain the virus in the environment. Birds such as house sparrows and finches are highly competent hosts (Komar et al 2003), and may be capable of dispersing WNV in New Zealand.

One species of mosquito (*Culex quinquefasciatus*) present in New Zealand is known to carry and transmit WNV overseas (Akhter et al 1982; Hubálek and Halouzka 1999; Bernard and Kramer 2001; Goddard et al 2002; Spurr and Sandlant 2004). *C. quinquefasciatus* is a bridge vector as it bites both birds and mammals (Huhn et al 2003; Spurr and Sandlant 2004). A soft bird tick (*Ornithodoros capensis*), also present in New Zealand, has been shown experimentally to be capable of transmitting WNV (Hubálek and Halouzka 1999), although its vector competence in the wild is unknown (Spurr and Sandlant 2004).

Alternative modes of transmission for WNV in the absence of arthropod vectors include the oral-faecal route and direct contact between vertebrate hosts (Komar 2003).

The likelihood of exposure of WNV is assessed to non-negligible.

14.2.3. Consequence assessment

Introduction of WNV into competent New Zealand mosquitoes could result in transmission to animals, birds, and man. The distribution of the virus within New Zealand would likely be determined by vector distribution which is potentially limited by climate (Spurr and Sandlant 2004). Infection could result in clinical disease in people, horses, and some species of birds.

This disease would have a major economic impact on the equine industry as about one third of reported cases end fatally. Geese are the only known natural hosts of WNV among domestic avian species. Affected geese show various degrees of neurologic disease and mortality rates of 20-60% have been reported. WNV has been detected in >326 species of birds and may have an impact on New Zealand's indigenous birds, however as disease has only been reported in a few species the impact on native birds is unlikely to be high. Human infections generally occur as the result of mosquito bites. In most cases, the disease is mild or even asymptomatic, but some people will develop flu-like symptoms. In less than 1% of cases meningitis, encephalitis, or acute paralysis may occur, and in a proportion of these the disease may be fatal or result in permanent impairment.

The consequence of introduction and establishment of WNV in New Zealand is assessed to be non-negligible.

14.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result, the risk estimate for WNV is non-negligible and it is classified as a risk in live psittacines. Therefore risk management measures can be justified

14.3. RISK MANAGEMENT

14.3.1. Options

Chapter 8.16 of the OIE *Code* decribes international standards that could be applied to manage the risk of West Nile fever (WNF) associated with trade in birds other than poultry

Article 8.16.5. of the OIE *Code* makes the following recommendations for importation of birds other than poultry from WNF free countries or zones:

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

1. The animals were kept in a WNF free country or zone since birth or for at least 30 days prior to travel; or
2. The animals were kept in a WNF free country or zone for at least 15 days, were subjected, with negative results, to an agent identification test according to the *Terrestrial Manual* carried out on a sample collected at least 3 days after the commencement of the residence period and remained in the WNF free country or zone until travel; or
3. The animals:

    a. were vaccinated in accordance with the Terrestrial Manual 30 days before introduction into the free country or zone; and
    b. were identified as having been vaccinated; and
    c. were kept in a WNF free country or zone for at least 15 days; and

d. remained in the WNF free country or zone until travel;

AND

4. If the animals were exported from a WNF free zone, either:

    a. did not transit through an infected country or infected zone during transportation to the place of travel; or
    b. were protected from mosquito attacks at all times when transiting through an infected country or infected zone; or
    c. had been vaccinated in accordance with point 3 above.

Article 8.16.6. of the OIE *Code* makes the following recommendations for importation of birds other than poultry from WNF seasonally free countries or zones:

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the animals:

1. Were kept during the seasonally free period in a WNF seasonally free country or zone since birth or for at least 30 days prior to travel; or
2. Were kept during the WNF seasonally free period in a WNF seasonally free country or zone for at least 15 days prior to travel, and were subjected during the residence period in the country or zone to an agent identification test according to the *Terrestrial Manual*, with negative results, carried out on a sample collected at least 3 days after the commencement of the residence period and remained in the WNF seasonally free country or zone until travel; or
3. Were kept during the seasonally free period in a WNF seasonally free country or zone for at least 15 days prior to travel, and were vaccinated in accordance with the *Terrestrial Manual* 30 days before introduction into the free country or zone against WNF, were identified as having been vaccinated and remained in the WNF seasonally free country or zone until travel;

AND

4. If the animals were exported from a WNF seasonally free country or zone, either:

    a. did not transit through an infected country or infected zone during transportation to the place of travel; or
    b. were protected from mosquito attacks at all times when transiting through an infected country or infected zone; or
    c. were vaccinated in accordance with point 3 above.

Article 8.16.8. of the OIE *Code* makes the following recommendations for importation of birds other than poultry from WNF infected countries or zones:

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

1. The birds showed no clinical sign of WNF on the day of travel; and
2. The birds were kept in a quarantine station in a mosquito-free environment for 30 days prior to travel and a statistically valid sample was subjected, with negative results, to an

agent identification test according to the Terrestrial Manual at least 3 days after the commencement of the residence period.

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

Option 1

Birds could be imported without restriction from countries that are free from WNV[2].

Option 2

Birds could be imported without restriction from countries that are seasonally free from WNV during the season of freedom from the disease[3].

Option 3

Birds to be imported from all countries could be subjected to quarantine in an insect free premise for a period of at least 7 days.

Option 4

Birds could be imported from countries that are infected with WNV provided all the provisions in Chapter 8.16 of the *Code* are adhered to.

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[2] The *Code* provides definitions for West Nile disease free countries.
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15. Avian bornavirus

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Avian bornavirus is phylogenetically related to *Borna disease virus* (BDV), the sole species in the genus *Bornavirus* of the family *Bornaviridae* in the order *Mononegavirales* (Wünschmann et al 2011).

Avian bornavirus (ABV) is the causative agent of proventricular dilatation disease (PDD) (de Kloet et al 2011). At least seven distinct ABV genotypes have been identified (Villanueva et al 2010; Payne et al 2011).

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

A case that histologically resembled PDD in a conure was described in New Zealand in 1996 (MAF 2010). No further suspicious cases have been reported. For the purposes of this risk analysis PDD is regarded as an exotic disease.

15.1.4. Epidemiology

ABV causes progressive lethal neurologic disease in psittacine birds (Kistler et al 2008; Gray et al 2010). The most common form of this disease, PDD, has been recorded in over 80 species of psittacine birds (Gregory et al 1994; Payne et al 2011) and in some passerines (Weissenböck et al 2009b). Large parrots are the most frequently and most severely affected birds (Piepenbring et al 2012).

PPD was first described in the late 1970s, but its aetiology remained unknown until 2008 when the correlation between ABV and PDD was discovered. Numerous studies and trials have since confirmed this association (Honkavuori et al 2008; Kistler et al 2008; Lierz et al 2009; Rinder et al 2009; Weissenböck et al 2009a; Kistler et al 2010; Villanueva et al 2010; Piepenbring et al 2012).

ABV is distributed in captive psittacine collections throughout the world including Australia, Israel, the USA, Canada, Germany, Austria, Switzerland, Hungary, Spain, Italy, Denmark, and the UK (Heffels-Redmann et al 2011).

PDD is a severe, often fatal disorder of the central nervous and the digestive systems, which mainly affects psittacine birds (de Kloet et al 2011). PDD was initially reported as a unique disease entity in macaws in the late 1970s but has since been described in a variety of species including cockatoos, macaws, amazons, and African grey parrots (Gregory et al 1994; Staeheli et al 2010; de Kloet et al 2011). PDD has previously been known as macaw wasting syndrome, proventricular dilatation syndrome, neuropathic gastric dilatation of psittaciformes, and myenteric ganglioneuritis (Gregory et al 1994; Doneley et al 2007; Staeheli et al 2010).

The disease is characterized by lymphohistioplasmocytic infiltration of the ganglia of the central and peripheral nervous system leading to gastrointestinal dysfunction or neurological

signs (Staeheli et al 2010; Heffels-Redmann et al 2011).

The clinical presentations of PDD may be predominately neurological (weakness, ataxia, proprioceptive deficits, seizures, blindness), gastrointestinal (wasting, passage of undigested food, regurgitation, delayed crop emptying, impaction of the proventriculus, and diarrhoea), or a combination thereof (Gregory et al 1994; de Kloet and Dorrestein 2009; Staeheli et al 2010; Payne et al 2011).

Inflammation of the autonomous nervous system of the alimentary tract is pathognomonic. However, inflammatory lesions may be more widespread and involve the central nervous system, kidney, adrenal gland, heart, and lungs (Wünschmann et al 2011). The typical findings in post mortem examination are dilatation of the oesophagus, proventriculus, ventriculus, or small intestine, and muscle atrophy (Staeheli et al 2010). The presence of lymphoplasmacytic infiltrates in the enteric nerve plexuses of the proventriculus and ventriculus are characteristic and, less frequently, also in the oesophagus, crop, and duodenum (de Kloet and Dorrestein 2009; Staeheli et al 2010).

There is little published about the transmission of the virus and the epidemiology and pathogenesis of ABV remain unclear (Payne et al 2011). Several authors consider that ABV is not highly contagious (de Kloet and Dorrestein 2009) and note that cage mates of birds diagnosed with PDD are frequently unaffected (Doneley et al 2007). However, others have suggested more efficient transmission from infected birds (Kistler et al 2010; Gartrell 2012). The presence of ABV in faeces has prompted postulation of faecal-oral transmission, but other routes such as vertical transmission may be possible (Lierz et al 2011). PDD is often associated with poor hygienic conditions and is rare in well-managed collections (de Kloet and Dorrestein 2009).

ABV has been detected in psittacines without pathological lesions or clinical signs of disease (Lierz et al 2009; Heffels-Redmann et al 2011). Subclinically infected birds may excrete the virus for extended periods and the incubation period may vary from weeks to years (De Kloet and Dorrestein 2009; Villanueva et al 2010). Discrepancies in infection status and in the development of clinical signs in infected but clinically healthy birds could be a result of host factors (such as age and immune status), strain, virulence, and adaptation of the virus (Piepenbring et al 2012). Birds carrying ABV are not immune to superinfection with a different strain of the same genotype (Payne et al 2011).

Definitive diagnosis of PDD requires demonstration of lymphoplasmacytic ganglioneuritis in the gastrointestinal tract (Payne et al 2011). In live birds, ABV can be diagnosed directly by detection of RNA using reverse transcription polymerase chain reaction (RT-PCR) on nonvascular contour (chest) feather calami (de Kloet et al 2011), faeces, and crop and cloacal swabs (de Kloet and Dorrestein 2009, Lierz et al 2009). Avian bornaviral RNA in feathers remains stable for at least 4 weeks when stored at room temperature, whereas most other tissues require storage at temperatures below freezing (de Kloet et al 2011). Multiple feathers should be used as positive results are not consistently obtained from single feathers (de Kloet et al 2011). Diagnosis can also be made by serological detection of anti-P40 nucleoprotein (and other bornaviral protein) antibodies (de Kloet et al 2011). Serological methods include a western blot assay (de Kloet and Dorrestein 2009; Lierz et al 2009; Villanueva et al 2009), an enzyme-linked immunosorbent assay (ELISA) (de Kloet and Dorrestein 2009), and an indirect immunofluorescence assay (IFA) (Gray et al 2010).

Many birds may serve as healthy carriers of ABV for years in the absence of a detectable

serological response to the virus (Payne et al 2011). Additionally seroconversion may not necessarily coincide with the presence of viral RNA (Villanueva et al 2010; Heffels-Redmann et al 2011). Therefore testing of only one kind of sample may imply a high risk of false negative results and it is strongly recommended to combine molecular, biological, and serological testing of a variety of samples for the diagnosis of ABV infection (Heffels-Redmann et al 2011).

15.1.5. Hazard identification conclusion

Avian bornavirus is an exotic organism that may cause severe illness and death in psittacines and possibly other avian species. It is therefore identified as a potential hazard.

15.2. RISK ASSESSMENT

15.2.1. Entry assessment

The induction period of avian bornavirus may be long and birds may carry the infection subclinically. These birds are unlikely to be detected during ante mortem examination. Therefore the likelihood of entry is assessed to be non-negligible.

15.2.2. Exposure assessment

Avian bornavirus is not considered by some authors to be highly contagious. However, imported psittacines may be housed with other birds and may come into contact with wild native psittacines. The incubation period may be protracted and subclinically infected birds may shed the virus in their faeces. Therefore, the likelihood of exposure is assessed to be non-negligible.

15.2.3. Consequence assessment

Since large parrots have high economic value and the disease is invariably fatal the introduction of the disease agent is likely to have serious economic consequences for breeders and owners of psittacine birds. The effect of introduction of ABV on native psittacines is not known but it is likely that they would be as susceptible as other psittacine species. ABV is not infectious to non-avian species and is not a zoonotic disease. Therefore, the consequences of introduction of avian bornavirus are assessed to be moderate.

15.2.4. Risk estimation

Since the entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and avian bornavirus is classified as a risk in live psittacines. Therefore, risk management measures can be justified.

15.3. RISK MANAGEMENT

15.3.1. Options

Avian bornavirus is not listed by the OIE, thus there are no international standards for risk management. Infection does not always result in detectable antibody production. Therefore, a combination of molecular and serological diagnostic tests on a number of different tissues should be performed simultaneously.

Since the disease can have a long incubation period, quarantine is not a viable option to prevent the spread of the disease. The importation of animals could be restricted to countries where the disease has not been reported.

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

Option 1

Birds which have been resident since birth in countries where the virus or disease has never been reported could be imported without restrictions.

Option 2

Birds could be imported from flocks with a 4 year history of freedom from the disease (4 years is the maximum suggested incubation period).

Option 3

Birds to be imported could be tested by a RT-PCR on feather calami or a cloacal swab and faeces, as well as a serological test (ELISA) for P40 antibodies, with negative results.

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16. *Bordetella avium*

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

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

16.1.2. OIE list

Not listed.

16.1.3. New Zealand status

B. avium has not been identified in New Zealand.

16.1.4. Epidemiology

B. avium is the causative agent of bordetellosis in avian species, also referred to as turkey coryza and *Bordetella avium* rhinotracheitis (BART). Turkeys are the natural host of *B. avium* although the organism has also been recovered from other wild and domesticated avian species (Raffel et al 2002; Jackwood and Saif 2008). *B. avium* has been reported in a variety of psittacine species including parrot finches (*Erythrura psittacea*), cockatoos (*Kakatoe galleria* and *Cacatua moluccensis*) (Hinz and Glünder 1985; Clubb et al 1994), cockatiels (Clubb et al 1994; Fitzgerald et al 2001; Matsuda et al 2009), conures (*Aratinga* spp.), a moluccan cockatoo, and macaws (*Ara* spp.) (Clubb et al 1994; Raffel et al 2002).

In the majority of cases infection with *B. avium* is not associated with clinical signs and the organism is thought to be widely distributed in healthy birds (Raffel et al 2002). Reports of disease associated with *B. avium* in psittacines are limited to cases of "lockjaw" (temporomandibular rigidity) in juvenile cockatiels (Clubb et al 1994; Fitzgerald et al 2001; Matsuda et al 2009) and mild upper respiratory disease in other species (Clubb et al 1994).

In one study experimental infection of cockatiels with *B. avium* reproduced the disease but outbreaks of "lockjaw" syndrome have also been seen with other bacterial agents (Greenacre et al 1999; Fitzgerald et al 2001). It has been postulated that "lockjaw" in cockatiels is a consequence of non-specific respiratory infection rather than a specific disease caused by a single aetiological agent (Fitzgerald et al 2001).

B. avium is highly sensitive to a variety of antibiotics (Matsuda et al 2009) and birds with mild signs may respond to antibiotic therapy. However, treatment of cockatiels with "lockjaw" has been largely unsuccessful (Clubb et al 1994; Raffel et al 2002; Alison and Smith 2005).

In turkeys, disease is usually seen from 2 to 6 weeks (Hinz et al 1978; Panigrahy et al 1981; Boycott et al 1984). Outbreaks are usually associated with a high morbidity and low mortality (Saif et al 1980; Kelly et al 1986) although higher mortality rates and more severe clinical signs may be seen in the presence of concomitant infections (Saif et al 1980; Boycott et al 1984; Cook et al 1991). Gross lesions (nasal and tracheal exudates, distortion of tracheal cartilage, and hyperaemia of the nasal and tracheal mucosae) are confined to the upper respiratory tract (Arp and Cheville 1984).

B. avium infection of chickens results in respiratory disease with similar but less severe clinical signs and lower mortality compared to the disease described in turkeys. Simmons et al (1981) described disease in 2-to-6-week-old broilers which presented as severe conjunctivitis, rales and nasal exudation. At post mortem examination, affected chickens were found to have a small to moderate accumulation of clear mucus in the nasal turbinates and trachea with no other changes observed in uncomplicated cases. As has been described with turkeys, secondary bacterial infections were also seen and were associated with perihepatitis, pericarditis, and airsacculitis. Coryza-like disease in ducks has been produced experimentally using isolates of *B. avium* recovered from turkeys (Hinz et al 1983).

Little is known about transmission or prevalence of *B. avium* in birds other than turkeys or if the bacterium causes disease in wild birds (Raffel et al 2002). *B. avium* is highly contagious and has a predilection for ciliated epithelial cells of the upper respiratory tract (Sebaihia et al 2006; Jackwood and Saif 2008), and is considered an opportunistic pathogen (Greenacre et al 1999). Transmission of infection occurs through close contact or exposure to contaminated litter or water and aerosol transmission is considered unlikely (Simmons and Gray 1979). *B. avium* survives for long periods of time in water and dilute salt solutions, and can remain virulent in litter for up to 6 months (Raffel et al 2002). Subclinically infected carrier birds are a common source of infection (Clubb et al 1994).

Diagnosis can be made by serology (microagglutination or ELISA), culture of tracheal swabs, and PCR of blood or nasopharyngeal swabs (Raffel et al 2002; Tüerkyilmaz et al 2009; Zoologix 2010). Antibodies are detectable for a long periods after infection (Raffel et al 2002) and serological tests remain positive for a period after *B. avium* can no longer be cultured (Jackwood and Saif 2008). In experimentally infected poults, antibody is detectable by serology from 2 weeks following infection until at least 5-7 weeks (Jackwood and Saif 2008). ELISA is thought to be the most sensitive of the serological tests and has the added benefit of detecting maternal antibody (Jackwood and Saif 2008; Tüerkyilmaz et al 2009).

A vaccine is not available for use in cockatiels, though vaccines are available for use in turkeys (Alison and Smith 2005).

16.1.5. Hazard identification conclusion

B. avium is associated with the "lockjaw" syndrome of cockatiels and has been found in clinically and subclinically infected psittacines. *B. avium* is identified as a potential hazard in live psittacines.

16.2. RISK ASSESSMENT

16.2.1. Entry assessment

Psittacines may be subclinically infected with *B. avium* and carrier birds are likely to remain undetected following clinical examination. Therefore, the likelihood of introduction of *B. avium* with imported live psittacines is assessed to be non-negligible.

16.2.2. Exposure assessment

Introduced psittacine birds may travel throughout New Zealand and may mix with other birds in aviaries or at bird shows. Wild birds may have contact with aviaries containing introduced

psittacines and also with disposed aviary waste. Therefore, the likelihood of exposure of *B. avium* is assessed as non-negligible.

16.2.3. Consequence assessment

Introduction of *B. avium* could lead to disease and mortalities in cockatiels and possibly other psittacine species. *B. avium* can infect poultry and other avian species and introduction of the organism could result in the establishment of a significant disease of turkeys. The effect of infection in native birds is unknown but likely to be minor as disease is largely confined to turkeys and cockatiels. The organism is not zoonotic and there would be no consequences for human health. Therefore the consequences of introduction of *B. avium* are assessed as non-negligible.

16.2.4. Risk estimation

Since entry, exposure, and consequence assessments for *B. avium* are all non-negligible, the risk estimation is non-negligible and *B. avium* is classified as a risk in live psittacines. Therefore risk management measures can be justified.

16.3. RISK MANAGEMENT

As discussed above, diagnosis of *B. avium* can be made by serology (microagglutination or ELISA), culture of tracheal swabs, and PCR of blood or nasopharyngeal swabs (Raffel et al 2002; Tüerkyilmaz et al 2009; Zoologix 2010). Antibodies are detectable for a long periods after infection (Raffel et al 2002) and serological tests remain positive for a period after *B. avium* can no longer be cultured (Jackwood and Saif 2008). In experimentally infected poults, antibody is detectable by serology from 2 weeks following infection until at least 5-7 weeks (Jackwood and Saif 2008). ELISA is thought to be the most sensitive of the serological tests and has the added benefit of detecting maternal antibody (Jackwood and Saif 2008; Tüerkyilmaz et al 2009). Refelcting this, the use of a serum ELISA may be preferable to bacterial cultures or PCR tests to manage the risk in imported psittacines.

16.3.1. Options

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

Option 1

Live psittacines could be imported if they

i. show no clinical signs of respiratory disease on the day of travel *and*
ii. come from establishments which contain at least one cockatiel *and*
iii. come from establishments where “lockjaw” syndrome has not been recognised

Option 2

Live psittacines could be imported if they

i. show no clinical signs of respiratory disease on the day of travel *and*
ii. test negative to an ELISA within two weeks of travel

Option 3

Live psittacines could be imported if they

i. are quarantined for 4 weeks prior to the day of travel *and*
ii. test negative to an ELISA within two weeks of travel *and*
iii. show no clinical signs of respiratory disease on the day of travel

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17. *Borrelia burgdorferi*

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

Borrelia burgdorferi is a spirochete, first recovered from the mid-gut of the deer tisk Ixodes dammini, and identified as the cause of Lyme disease (Burgdorfer 1991)

17.1.2. OIE list

Not listed.

17.1.3. New Zealand status

A serological survey of dogs in New Zealand with clinical sigsn consistent with Lyme disease showed noe evidence of exposure. There is no evidence of borreliosis in New Zealand (Midwinter 1999).

17.1.4. Epidemiology

Lyme disease, caused by *B. burgdorferi*, affects dogs, horses, cattle, and humans. These species are incidental hosts to an organism that normally cycles between reservoir hosts (predominantly small mammals) and tick vectors (generally of the *Ixodes* genus). The maintenance hosts for adult ticks are larger mammals which are not reservoir hosts for *Borrelia*. In Europe, the main vector is *Ixodes ricinus*, in the eastern United States it is *I. scapularis*, in the western United States it is *I. pacificus,* and in Eurasia it is *I. persulcatus* (Nadelman and Wormser 1998). The distribution of *Ixodes* spp. ticks that are able to transmit the agent of Lyme disease spreads in a broad band across North America, Europe, and northern Asia (Anonymous 2003).

Borreliae cannot survive as free living organisms. Small mammals and birds are reservoir hosts and infection is transmitted by certain *Ixodes* species of tick. Haematophagous arthropods, including other tick species, fleas, flies and mosquitoes have been found to be infected in nature. These other arthropods are believed to have acquired infection from feeding on infected vertebrates but they have not been capable of transmitting infection to new hosts experimentally (Piesman 1997). Their role, if any, relative to the known tick vectors, is considered insignificant. Natural transmission of the organism by any means other than by tick inoculation has not been reported.

Borreliosis is the most frequent tick-transmitted zoonotic disease in the northern hemisphere affecting humans (up to 155 cases per 100,000 individuals) (Wilske 2005). The bacterium has specific tropism for skin, musculoskeletal tissue, joints and the central nervous system depending on the species involved.

Early symptoms of human borreliosis include a red, enlarging rash from the site of tick bite, and flu-like symptoms. Many complications may follow an untreated case, such as meningitis, Bell's palsy (paralysis of part of the face), heart block and painful joints, muscles and bones (AOCD 2004; Bratton 2005).

Avian species are recognised to develop subclinical infections with *B. burgdorferi* and serve as hosts for tick species capable of spreading this spirochaete to mammals (Ginsberg et al

2005; Comstedt et al 2006; Kipp et al 2006; Poupon et al 2006). A single report of *B. burgdorferi* infection of a psittacine species has been described, with the organism identified in the blood and liver of an African Grey parrot imported from Ghana to Switzerland (Ehrsam 1977).

17.1.5. Hazard identification conclusion

Because infection of psittacines has been described, *B. burgdorferi* is identified as a potential hazard.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

Psittacines imported from countries where *B. burgdorferi* is present may be subclinically infected and would therefore be unlikely to be detected by clinical examination. However, only one report of infection in a psittacine can be found, so the likelihood of introduction of *B. burgdorferi* with imported live psittacines is assessed to be very low.

17.2.2. Exposure assessment

Two *Ixodes* spp. ticks have been recovered from psittacine species in New Zealand; *I. auritulus* group and *I. kerguelenensis* (Heath 2010). *I. auritulus* has been recognised as a competent vector of *B. burgdorferi* (Scott et al 2010) and is known to have a widespread distribution in New Zealand (Cane 2009).

I. auritulus zealandicus (the proposed sub-species recognised in New Zealand) is usually associated with burrow-nesting sea birds although exceptions are recognised (Cane 2009). There is considerable morphological variation among specimens of *I. auritulus* although the vast majority of infectations are found on passeriformes (González-Acuña et al 2005). In New Zealand, specimens of *I. auritulus* are found associated with nests as well as on host birds (Cane 2009).

Although a competent host tisk for *B. burgdorferi* is recognised in New Zealand, it is only rarely associated with psittacines and it is highly unlikely that imported psittacines will be kept in environements in New Zealand where *I. auritulus* are likely to be found. Reflecting this, the likelihood of exposure is assessed to be negligible.

17.2.3. Risk estimation

Since the exposure assessment for *B. burgdorferi* is negligible, the risk estimate is negligible and risk management measures cannot be justified.

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18. *Coxiella burnetii* and *Coxiella*-like organisms

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

Coxiella burnetii is an obligate intracellular gram-negative bacterium. *C. burnetii* is currently the only species in the genus, although new *Coxiella*-like organisms have recently been detected in psittacines (Shivaprasad et al 2008), arthropods (Cutler et al 2007), and avian ticks (Reeves et al 2006). *Coxiella burnetii* occurs in two morphological forms, a small-cell variant and a large-cell variant (Coleman et al 2004).

18.1.2. OIE list

Q fever is listed in the OIE *Code* as a disease of multiple species.

18.1.3. New Zealand status

Coxiella burnetii is a notifiable organism (Tana et al 2011). A number of investigations have demonstrated that New Zealand is free from Q fever (Worthington 2001).

18.1.4. Epidemiology

Q fever is a highly contagious zoonotic disease caused by *C. burnetii*. *C. burnetii* is commonly present in all countries worldwide, except in New Zealand (Worthington 2001; EFSA 2010) and may be carried by many domesticated and wild animals including mammals, birds, reptiles, and arthropods (Cutler et al 2007).

Antibodies to *C. burnetii* have been demonstrated in more than 49 species of wild and domestic birds in North America, Europe, Japan, India, and other countries (Enright et al 1971; Riemann et al 1979; Yadav and Sethi 1980, as cited in Shivaprasad et al 2008; To et al 1998). It is not known if these antibodies were specific for *C. burnetii* or were a result of cross reaction to some novel *Coxiella*. Recently infections of psittacine birds with *Coxiella*-like organisms has been reported (Shivaprasad et al 2008; Woc-Colburn et al 2008).

Infection in birds is usually subclinical (Barnes 2003) and experimental infection of birds has produced neither clinical signs nor lesions (Sethi et al 1978). However, Shivaprasad et al (2008) describe disease associated with *Coxiella*-like bacteria in psittacines in which clinical signs included anorexia, lethargy, and weight loss and post mortem examination revealed emaciation, and enlarged livers and spleens. Similarly, fatal coxiellosis in lorikeets was reported by Woc-Colburn et al (2008).

Cattle, sheep, and goats are usually subclinical carriers but infection may result in abortions, infertility, neonatal deaths, and occasionally hepatitis and pneumonia (Krauss 1989; Shivaprasad et al 2008). Infected animals shed *C. burnetii* in birth products, faeces, milk, and urine (EFSA 2010). In humans, Q fever can be asymptomatic or present in an acute form (pneumonia, hepatitis, abortion) or a chronic form (endocarditis, osteomyleitis) (Stein and Raoult 1999; Cone et al 2006; EFSA 2010). Although the disease is usually treatable with antibiotics, in rare cases Q fever can be fatal (EFSA 2010).

C. burnetii is highly resistant in the environmental and can survive for long periods (EFSA 2010). Domestic ruminants are the main reservoirs and principal source of infection for humans and human outbreaks generally involve occupationally exposed people (farmers, veterinarians, slaughterhouse personnel) (EFSA 2010). Ticks may also play an important role in maintaining and spreading the organism. At least 40 species of ticks from 11 genera can be infected (Maurin and Raoult 1999). Humans are mainly infected via aerosols generated from excreta from infected animals, but transmission may also occur by the ingestion of unpasteurised milk, raw eggs, or other contaminated material (Stein and Raoult 1999; EFSA 2010). There are no reports of humans being infected by contact with psittacine birds although Stein and Raoult (1999) report a human outbreak of Q fever following exposure to pigeon faeces and pigeon ticks.

As infected animals generally remain subclinical, the incubation period and the interval to the development of antibodies are uncertain. In humans, the incubation period is 1-3 weeks and the development of detectable antibodies takes 2-3 weeks after the onset of symptoms (Maurin 1999). Therefore it is reasonable to conclude that antibodies may not be detectable for up to 6 weeks following infection. Organisms can be recovered from faeces of infected mammals for up to 40 days post-infection (Little 1983), but whether a similar period of excretion occurs in birds is not known.

Antibodies can be detected in birds using complement fixation, immunofluorescence, microagglutination tests or ELISA (Fournier et al 1998; Maurin and Raoult 1999; Porter et al 2011). Additionally the organism can be detected in clinical samples by PCR or can be isolated in embryonated eggs or cell cultures (Fournier et al 1998; Maurin and Raoult 1999; Auricu-Bovery et al 2003). PCR is useful for detection of early acute cases, or applied to tissue samples from focal manifestations. However, it is recommended that PCR be used in addition to serology (Cutler et al 2007).

It is assumed that serological tests for *C. burnetii* will detect antibodies to closely related *Coxiella*-like organisms.

18.1.5. Hazard identification conclusion

C. burnetii is an exotic, notifiable, and zoonotic organism. *Coxiella*-like organisms are exotic and zoonotic. Both are capable of infecting birds and therefore are identified as potential hazards in live psittacines.

18.2. RISK ASSESSMENT

18.2.1. Entry assessment

Acute and chronic infections of psittacines with *C. burnetii* and *Coxiella*-like organisms may occur in the absence of clinical disease and thus not detected during clinical examination. Therefore the likelihood of entry of *Coxiella burnetii* and *Coxiella*-like organisms is assessed to be non-negligible.

18.2.2. Exposure assessment

C. burnetii and presumably *Coxiella*-like organisms are highly infectious and could be spread to in-contact birds and other animals. It is not known whether the New Zealand cattle tick, *Haemaphysalis longicornis*, can become infected, but other species of this genus are

susceptible to infection. The likelihood of exposure of *Coxiella burnetii* and *Coxiella*-like organisms is assessed to be non-negligible.

18.2.3. Consequence assessment

Many psittacine species are known to be susceptible to infection with *C. burnetii* and *Coxiella*-like organisms but disease as a result of infection is very uncommon. Nevertheless, infection could spread amongst both exotic and native species of birds, livestock, and other animals and result in sporadic cases of serious disease in humans. The consequence assessment is therefore assessed to be non-negligible.

18.2.4. Risk estimation

Since the entry, exposure and consequence assessments are non-negligible, the risk estimation is non-negligible and *Coxiella burnetii* and *Coxiella*-like organisms are classified as risks in live psittacines. Therefore risk management measures can be justified.

18.3. RISK MANAGEMENT

18.3.1. Options

Birds have not been described as having a significant role in the epidemiology of *C. burnetii* and *Coxiella*-like organisms. Cattle, sheep, and goats are the principal source of infection for humans. Since domestic ruminants are considered the main reservoir, with birds only rarely reported to be shedders involved in human infections (Stein and Raoult 1999), it may be considered unnecessary to impose restrictions on their importation. Ticks may be important in the maintenance and transmission of the disease and birds should also be subjected to the measures proposed in the ectoparasites Chapter of this risk analysis. Infected psittacines are likely to be asymptomatic carriers of infection and quarantine may not prevent the entry of the organism. There are also no effective treatment regimes or vaccinations described for psittacines.

Option 1

Psittacine birds that are tick free could be imported without restrictions because the disease is rare in psittacines and they have not been described as being important in the epidemiology of the disease.

Option 2

Birds could be tested by a suitable serological test for Q fever within 3 weeks of travel, with negative results, and be subjected to the measures required to effectively manage the introduction of ticks.

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19. *Mycobacterium* spp.

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

Mycobacterium spp. are acid-fast, aerobic, non-spore-forming, non-motile, facultative intracellular, rod shaped bacilli (Mendenhall et al 2000; Quinn et al 2002).

Mycobacteria have been conventionally classified into four or five broad taxonomic groups based on their pathogenicity for humans and animals, rate of growth at optimum temperatures, and effect of visible light on pigment production (Inderlied et al 1993).

Avian mycobacteriosis is caused predominantly by three species of mycobacteria: *Mycobacterium avium, M. intracellulare* (together comprising the *M. avium* complex (Inderlied et al 1993)), and *M. genavense* (Witte et al 2008). Other species of *Mycobacterium*, including *M. bovis*, *M. fortuitum, M. gordonae, M. kansasii, M. nonchromogenicum,* and *M. tuberculosis*, have only rarely been identified in pet birds (Hoop et al 1996; Bargalló et al 2009; Grunkemeyer 2010).

19.1.2. OIE list

None of the avian *Mycobacterium* spp. are listed.

19.1.3. New Zealand status

Mycobacterium bovis is endemic in cattle and farmed deer in New Zealand and is the subject of an eradication campaign in a Pest Management Strategy under the Biosecurity Act 1993 (Montgomery 1999).

M. paratuberculosis is present in cows, deer, and sheep with low prevalence throughout New Zealand (Montgomery 1999).

Tuberculosis (TB) remains a significant human disease globally and in New Zealand it is notifiable to the Medical Officer of Health under the Tuberculosis Act 1948. In 2010 there were 661 cases of TB notified, comprised of 304 cases of TB disease and 357 cases of TB infection (Bissielo et al 2011). Contact with birds is not considered a risk factor in developing TB (Bissielo et al 2011).

M. avium and *M. intracellulare* are endemic in New Zealand birds (De Lisle 1987; Montgomery 1999) and have been detected in a range of species including fowl (Black 1997a), kiwi (Davis et al 1984), harrier hawk (Orr 1995), ostriches (Black 1997b), lovebirds (Anonymous 1999), and peacocks (Anonymous 2000).

The status of *M. genavense* in New Zealand is unknown. However, the organism has been isolated from a human patient in New Zealand and it was thought that the infection was acquired in New Zealand (Worthington 2010). No further cases have been reported.

19.1.4. Epidemiology

The *Mycobacterium avium* complex (MAC) is composed of opportunistic pathogens capable of causing disease in animals and humans. The MAC is a serological complex of 28 serovars of two species, *M. avium* and *M. intracellulare* (Inderlied et al 1993).

Mycobacteriosis is a common disease of both captive and free-ranging birds and has been diagnosed in nearly every avian order (Lennox 2007). Witte et al (2008) found mycobacteriosis in 1.2% of 13,976 birds studied and Lennox (2007) estimated a disease prevalence of 0.5% - 14% in captive psittacines.

In psittacines, mycobacteriosis is considered a disease of individual birds (Lennox 2007). Birds can present with weight loss, poor feathering, polyuria, diarrhoea, and abdominal distension or acute death (Lennox 2007).

M. genavense is recognised as the most frequent agent of avian mycobacteriosis in psittacines and other pet birds (Hoop et al 1996; Portaels et al 1996; Shitaye et al 2010) followed by the *Mycobacterium avium* complex (Hoop et al 1996; Witte et al 2008; Manarolla et al 2009). Other species of *Mycobacterium* have only rarely been identified in pet birds (Hoop et al 1996).

In both birds and humans, infection with *M. avium* or *M. genavense* results in similar clinical signs (Mendenhall et al 2000; Shitaye et al 2010).

M. avium causes a slow spreading, granulomatous infection seen predominantly in birds and in human AIDS patients (Mendenhall et al 2000). Organisms are ingested, then disseminate to the intestine and internal organs, resulting in chronic weight loss, diarrhoea, emaciation, and death (Mendenhall et al 2000). Tubercles developing at the site of infection in the intestinal wall allow for constant shedding into the environment (Mendenhall et al 2000). *M. avium* has been diagnosed as the cause of a number of cases of disease in psittacines (Reed and Johnson 1994; Stanz et al 1995).

M. genavense is a slowly growing, fastidious, ubiquitous saprophytic mycobacterium (Portaels et al 1996) and an opportunistic pathogen in humans particularly those infected with AIDS (Hoop et al 1996; de Lastours et al 2008). *M. genavense* has been reported as the cause of granulomatous lesions in psittacines (Hoop et al 1993; Kiehn et al 1996; Ferrer et al 1997; Manarolla et al 2009) and has been identified in other animals including a dog, a feline immunodeficiency virus (FIV) infected cat, and two ferrets (Mendenhall et al 2000; Gomez et al 2011).

The source, epidemiology, and mode of infection of *M. genavense* are unknown (Portaels et al 1996). Oral, aerosol, contaminated water, and haematogenous routes have all been suggested (Witte et al 2008; Gomez et al 2011) but as for *M. avium*, organisms are thought to be typically shed in the faeces (Lennox 2007).

M. avium and *M. genavense* are stable in the environment and persist for long periods (Lennox 2007). The organisms are unlikely to be a health risk to humans with normal immune systems (Lennox 2007). The extreme rarity, in non-HIV infected patients, of human diseases due to *M. genavense* suggests a low virulence for this organism (Tortoli et al 1998).

Mycobacteriosis is difficult to diagnose in live birds, and because of non-specific post mortem signs and difficulties in culturing, the causative agents are rarely identified (Mendenhall et al 2000; Hoop et al 1996). Culture may take up to four weeks and requires the use of special media (Mendenhall et al 2000). Several PCR techniques have been developed. The combination of PCR followed by restriction enzyme digestion is more efficient than PCR amplification followed by sequencing and allows the detection of and distinction between *M. avium* and *M. genavense* (Mendenhall et al 2000).

Mycobacterium tuberculosis causes TB in humans (Washko et al 1998; Schmidt et al 2008) and is a rare disease of parrots (Woerpel and Rosskopf 1984; Hoop 2002; Steinmetz et al 2006). The disease does not occur in wild parrots and it is considered that humans are the most likely source of infection in psittacines (Hoop 2002; Steinmetz et al 2006). A summary of reported cases of tuberculosis in parrots has been given by Schmidt (2008). Lesions occur most commonly on the unfeathered head region and include subcutaneous and cutaneous nodules, granulomas or sinuses on the membrana nictitans and retrobulbar tissues, in the infraorbital sinuses, nares, and oral cavity. Lung lesions and other lesions of generalised tuberculosis also occur (Washko et al 1998; Schmidt et al 2008). Birds with advanced disease show signs of wasting and general malaise.

19.1.5. Hazard identification conclusion

Mycobacterium avium, *M. intracellulare*, and *M. tuberculosis* occur commonly in New Zealand and are therefore not identified as potential hazards in live psittacines.

Mycobacterium genavense is a ubiquitous saprophyte and opportunistic pathogen. The status of *M. genavense* in New Zealand is unknown. However, it is recognised that the specific agent responsible for mycobacteriosis in birds is often undiagnosed. *M. avium* and *M. genavense* result in nearly identical clinical signs following infection (Mendenhall et al 2000; Shitaye et al 2010). As *M. genavense* is not more pathogenic or virulent than species of avian *Mycobacterium* present in New Zealand, it is not identified as a potential hazard in live psittacines.

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20. *Mycoplasma* spp.

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

The avian mollicutes are divided into two orders: *Mycoplasmatales* (containing the genera *Mycoplasma* and *Ureaplasma*) and *Acholeplasmatales* (containing the genus *Acholeplasma)* (Nicolet 1996; Stipkovits and Kempf 1996).

The only reports of *Mycoplasma* species which have been isolated from psittacine species are of *M. gallisepticum, M. iowae,* and an unidentified *Mycoplasma* species (Bozeman et al 1984).

20.1.2. OIE list

Mycoplasmoses due to *Mycoplasma gallisepticum* and *Mycoplasma synoviae* are OIE-listed diseases. There are no reports of *M. synoviae* in psittacine species.

20.1.3. New Zealand status

M. gallinaceum, M. gallinarum, M. gallisepticum, M. iners, M. synoviae and *A. laidlawii* are present in New Zealand (Black 1997; Christensen 1997; Bingham 2010; Bingham 2011). *M. gallisepticum* is endemic in New Zealand (Pohl 1966; McCausland 1972; Lohr 1975) although it has been suggested that more virulent strains may be present overseas (Christensen 2010). Positive serology has also been reported for *M. meleagridis* in turkeys (Pohl 1969; Anonymous 1994a - 1996d) and an unidentified mycoplasma (assumed to be *M. anatis*) has been isolated from a Peking duck (Hemsley 1996).

20.1.4. Epidemiology

In general *Mycoplasma* spp. have a limited host range and a preference for a single species. No reports of human infections with *Mycoplasma* species that infect birds have been located. There is little published information on mycoplasmosis in psittacine birds although it has been described as a common cause of upper respiratory disease in captive birds (Rosskopf and Woerpel 1996; Spira 1996; Kleven 2008).

Avian mycoplasma infections are rarely associated with marked clinical signs unless they are accompanied by concurrent infections or environmental stressors (Levisohn and Kleven 2000). Reports of natural infections of *Mycoplasma* spp. causing disease in psittacines are rare. One report describes the isolation of *M. gallisepticum*, *M. iowae*, and an unidentified *Mycoplasma* species from the respiratory tract of a single yellow-napped Amazon parrot from a flock in which 200 of 1,100 had died with upper respiratory disease. The authors did not consider that the *Mycoplasma* spp. were primary pathogens in the disease and a number of other pathogens were also isolated. Subsequent challenge studies in budgerigars and chickens with the isolated *Mycoplasma* organisms failed to induce lesions in chickens or establish persistent infections in budgerigars (Bozeman et al 1984). The latter finding is supported by Brown and Butcher (1991) who found that budgerigars, unlike chickens, are capable of rapidly eliminating *Mycoplasma*.

A more recent study by Gomes et al (2010) investigated the occurrence of *M. gallisepticum* in wild captive psittacines that had died during rehabilitation or translocation. *M. gallisepticum*

was diagnosed by PCR in over 80% of the birds, but was neither correlated with any observed clinical signs nor attributed as the cause or death. The high occurrence was thought be a consequence of the proximity or cohabitation with other bird species during confinement.

Lierz and Hafez (2009) investigated a small number of psittacine birds with respiratory disease and found PCR evidence of *Mycoplasma*. They suggested that *Mycoplasma* most likely contributes to the aetiology of chronic respiratory disease in psittacines rather than being the primary cause.

Other references to *Mycoplasma* infections in psittacines are vague and lacking details (Mall et al 1975; Oladele et al 1999). Several studies have failed to isolate *Mycoplasma* spp. from psittacine birds (Shimizu et al 1979; Poveda et al 1990). Reports of mycoplasmosis in psittacines usually refer to *M. gallisepticum*.

M. gallisepticum has a world-wide distribution (Levisohn and Kleven 2000) and occurs primarily in domestic and free-ranging gallinaceous birds (Ley 2008) but has also been recovered from a variety of other avian species (Buntz et al 1986; Reece et al 1986; Cookson and Shivaprasad 1994; El-Shater 1996; Murakami et al 2002; Benčina et al 2003). No zoonotic issues are associated with *M. gallisepticum* (Levisohn and Kleven 2000). In poultry, the incubation period varies from 6 to 21 days depending on strain virulence (Ley 2008). Transmission occurs vertically (Lin and Kleven 1982; Glisson and Kleven 1985; Ortiz et al 1995), and horizontally by direct or indirect contact via the upper respiratory tract or conjunctiva (Levisohn and Kleven 2000).

M iowae is almost worldwide in distribution, although due to eradication efforts it is now rarely encountered in commercial poultry (Bradbury and Morrow 2008). The natural host of *M. iowae* is the turkey (Bradbury and Kleven 2008) but it has also been found in a variety of other avian species (Yoder and Hofstad 1962; Benčina et al 1987a, 1987b; Lo et al 1994; Al-Ankari and Bradbury 1996). There are many different strains of *M. iowae*, and an unusually large degree of antigenic variation among strains (Al-Ankari and Bradbury 1996). Naturally-occurring disease appears to be restricted to turkeys with occasional reports of disease in chickens (Al-Ankari and Bradbury 1996; Trampel and Goll 1994). There is only one report of isolation of and no reports of disease caused by *M. iowae* in psittacines. Transmission of *M. iowae* is predominantly vertical although horizontal transmission may also occur (Bradbury and Kleven 2008). Unlike other avian mycoplasmas, *M. iowae* shows a resistance to bile salts and a predilection for the gastrointestinal tract (Bradbury and Kleven 2008).

The lack of a cell wall renders mollicutes fragile in the environment (Bradbury and Morrow 2008). They are readily killed by disinfectants and do not survive for prolonged periods outside the host (Bradbury and Morrow 2008; Ley 2008).

20.1.5. Hazard identification conclusion

Apart from isolated cases, reports of *Mycoplasma* infections in psittacines refer to *M. gallisepticum*. Infections other than *M. gallisepticum* are rare and there is no evidence that psittacines act as reservoir hosts for *Mycoplasma* spp.

Where mycoplasma infections have been associated with disease, they have been one of a number of organisms and have never been correlated with clinical signs in a mixed infection amongst which the mycoplasmas may have made a contribution (Bozeman et al 1984).

The highly virulent *M. gallisepticum* strains reported in poultry overseas (Christensen 2010) have not been identified in psittacines. As the less virulent strains of *M. gallisepticum* are endemic in New Zealand, they are not identified as a hazard in live psittacines.

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21. *Salmonella* spp.

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

There are two currently recognised species of *Salmonella*; *S. bongori* and *S. enterica*, and approximately 2500 serotypes. Most salmonellae of veterinary and public health relevance are in the sub-species *Salmonella enterica* subspecies *enterica*. According to the latest nomenclature, a *Salmonella* of serotype *typhimurium* is classified as *Salmonella enterica* subspecies *enterica* serotype Typhimurium. In this chapter the common abbreviated convention is used and the organism is referred to as *S.* Typhimurium. Some serotypes are further partitioned on the basis of phage type.

The *Salmonella* serotypes Pullorum and Gallinarum are specific to birds (Marietto-Gonçalves et al 2010). *Salmonella* Pullorum is the causal agent of pullorum disease and *Salmonella* Gallinarum is the causal agent of fowl typhoid. These two bacteria are currently placed in a single species, *Salmonella enterica* subsp. *enterica* serovar Gallinarum-Pullorum, hereafter referred to as *S.* Gallinarum-Pullorum (Shivaprasad and Barrow 2008).

Paratyphoid salmonellae include the motile non-host-adapted *Salmonella* serotypes including *S.* Typhimurium and *S.* Enteritidis (Gast 2008). Over 2500 serotypes of paratyphoid salmonellae are recognised (Gast 2008).

S. arizonae represent a diverse group of bacteria of over 300 serotypes and were previously classified in the genus Arizonae (Shivaprasad 2008).

Other *Salmonella* serotypes considered in this risk analysis include *S.* Abortusovis and *S.* Dublin.

21.1.2. OIE list

Salmonella Abortusovis, *Salmonella* Pullorum (pullorum disease) and *Salmonella* Gallinarum (fowl typhoid) are OIE listed diseases. Although paratyphoid salmonellae are not OIE listed, the *Code* contains sections concerned with their prevention, detection, and control (Chapter 6.5).

21.1.3. New Zealand status

Salmonella isolates recovered from human and non-human sources in New Zealand are submitted to the Enteric Reference Laboratory of the Institute of Environmental Science and Research Ltd (ESR) for serotyping.

In 2010, 1146 cases of salmonellosis were notified (26.2 per 100 000 population), and the number of cases notified each year has remained fairly stable since 2005 (ESR 2010a). Details of the serotypes identified are published regularly and show that a wide variety are present in this country. *S.* Typhimurium DT160 is the most common serotype confirmed (ESR 2010a). It has been estimated that officially recorded data probably represents less than 10% of the real incidence of foodborne disease occurring in the community in countries with developed surveillance systems (Clark et al 2000).

Exotic serovars and phage types of salmonellae are notifiable organisms (Tana et al 2011).

S. Pullorum and *S.* Gallinarum are exotic to New Zealand (Black 1997). Ongoing serological surveillance of commercial chicken breeder flocks has demonstrated freedom from *S.* Pullorum (Anonymous 2000, 2001; Poland 2002, 2004, 2005; Tana 2007; Frazer 2008). A small serological survey of Old English game fowl in 2005 found no evidence of exposure to *S.* Pullorum (Christensen 2006).

S. arizonae has never been reported in animals or birds in New Zealand.

S. Enteritidis DT4 and *S.* Typhimurium DT104 have been isolated from humans (but not animals) in New Zealand on several occasions including 2011 (ESR 2011a - 2011c). *S.* Typhimurium DT104 is of particular importance because it exhibits resistance to several commonly used antibiotics (Plagemann 1989; Hogue et al 1997; Jones et al 2002). Cattle are considered to be the primary host of *S.* Typhimurium DT104 (Davies 2001) and reports involving psittacines are rare (Hudson et al 2000).

Salmonella 4,[5],12:i:– has been confirmed in humans in New Zealand on a number of occasions but it has never been found in animals (ESR 2010b). *Salmonella* 4,[5],12:i:– are of increasing concern in Europe as a marked increase in resistance to ampicillin, streptomycin, sulphonamides, and tetracycline (R-type ASSuT) has been observed in isolates from clinical and meat samples (ESR 2010b, Hopkins et al 2010).

An epidemic of *S.* Typhimurium DT160, commencing in the winter of 2000, resulted in the death of a large number of sparrows (Alley et al 2002). Infections were also diagnosed in two sulphur-crested cockatoos and a captive kaka. This organism had not been identified in New Zealand prior to isolation from a human in 1998.

21.1.4. Epidemiology

There is little specific information about *Salmonella* infections in psittacines. In avian veterinary literature a distinction is usually made between infections caused by the two non-motile serotypes, *Salmonella* Pullorum (pullorum disease) and *Salmonella* Gallinarum (fowl typhoid), which are host-adapted serotypes of poultry, and salmonellae of the Arizonae and paratyphoid groups.

Mortalities of wild parrots due to salmonellosis have not been reported but *Salmonella* spp. are known to infect a variety of Psittaciformes in captivity and outbreaks of different intensity have been reported in aviaries (Butron and Brightsmith 2010). Prevalence of *Salmonella* in live captive psittacine birds has ranged from 0% to 32% in various studies (Allgayer et al 2008). Among psittacine species, *Salmonella* Typhimurium is the serotype most frequently isolated, but *S. arizonae*, *S.* Enteritidis, and *S.* Pullorum have also been reported (Smit 1965; Ward et al 2003; Marietto-Gonçalves et al 2010).

Clinical signs of salmonellosis in psittacine birds vary from mild enteritis to a severe illness characterised by anorexia, diarrhoea, lethargy, and dyspnoea (Orós et al 1998; Ward et al 2003). Death without previous signs, resulting from systemic disease with polyserositis, may also occur (Panigraphy and Gilmore 1983; Coleman 1993, as cited in Orós et al 1998). Outbreaks with high mortality have been observed, often exacerbated by concurrent disease and other stressors (Ward et al 2003). Gross findings include hepatomegaly, splenomegaly, enteritis, pyogranulomatous encephalitis, nephropathy, and pulmonary congestion (Panigrahy and Gilmore 1983; Coleman 1993, as cited in Orós et al 1998; Ward et al 2003).

The epidemiology of different *Salmonella* serotypes follows broadly similar patterns although host specificity varies between serotypes (Hattman et al 1976; Tsolis et al 1999; Rabsch et al 2002).

Spread is mainly via the faecal-oral route, both from direct contact and from ingestion of contaminated water or food (Ward et al 2003). Vertical transmission of *Salmonella* can either occur through dissemination of highly invasive strains into eggs before oviposition (Gast and Beard 1990a; Keller et al 1995) or through penetration into or through the shell and shell membranes (Henzler et al 1998; Williams et al 1998; Gast 2008).

*Salmonella*e may be introduced into a flock by feed (Cox et al 1983; Rose et al 1999), invertebrate vectors (Kopanic et al 1994; Olsen and Hammack 2000; Davies and Breslin 2003; Skov et al 2004), rodents (Henzler and Opitz 1992), wild birds (Refsum et al 2002), or even human sewage (Kinde et al 1996). Some birds can become subclinical carriers and intermittently shed bacteria for a prolonged period extending from weeks to months (Gast 2008; Butron and Brightsmith 2010; Marietto-Gonçalves et al 2010). *Salmonella* can survive for extended periods on fomites and reportedly can persist for 28 months in avian faeces (Ward et al 2003) and up to 200 days in soil (Butron and Brightsmith 2010).

Faecal shedding of salmonellae occurs for the first 2-3 weeks after infection of adult birds then steadily declines (Gast and Beard 1990a; Gast and Beard 1990b; Shivaprasad et al 1990). Intestinal colonisation of adult birds is usually followed by dissemination to a wide range of internal organs (Gast and Beard 1990b).

Studies of *Salmonella* infections in travellers returning to Sweden have shown that international travel is an effective means of moving salmonellae of different serotypes and phage-types between countries (Nygard et al 2004; Ekdahl et al 2005). The potential for environmental contamination and distribution of infection to humans and animals has also been highlighted (Sahlstrom et al 2004; Sahlstrom et al 2006).

Diagnosis can be made by identification of the organism, serology, and PCR (OIE 2008). There are several limitations of the diagnostic process. Isolation of the organisms in faeces may not detect carriers of infection because excretion can be intermittent and therefore repeated sampling and culture is often necessary (Butron and Brightsmith 2010). Furthermore standard culture-based methods are laborious, costly, and take at least 3 days to complete (Löfström et al 2010). Serological tests include rapid whole blood agglutination, rapid serum agglutination (RST), tube agglutination, and micro-agglutination. Serology is best applied on a flock basis and a series of two serological tests are required in individual birds (OIE 2008). Positive serological results do not necessarily imply ongoing *Salmonella* spp. infection because antibodies can still be detected long after complete recovery (Butron and Brightsmith 2010). Several PCR methods have been described that identify a wide range of *Salmonella* serotypes in various specimens (Schuurman et al 2007; Sareyyupoglu et al 2008; Löfström et al 2010). PCR tests have not been fully validated internationally (OIE 2008) however Allgayer et al (2008) used PCR to detect *Salmonella* species in captive psittacine birds and found a higher prevalence than that reported using other methods suggesting PCR is highly sensitive.

There are no suitable vaccines for use in psittacines and treatment can be difficult as resistance of salmonellae to antibiotics is widespread.

S. Gallinarum-Pullorum

Chickens are the natural host of *S.* Gallinarum-Pullorum and it is a common pathogen of chickens and turkeys (Shivaprasad and Barrow 2008). *S.* Gallinarum-Pullorum is thought to have low pathogenicity in other avian species and disease is rarely described (D'Aoust and Prescott 2007). Natural infections with *S.* Pullorum, of species other than chickens or turkeys, have usually resulted from exposure to infected chickens (Snoeyenbos 1991; Asterino 1996). This proposal is supported by the effective eradication of *S.* Pullorum from New Zealand through the implementation of a programme directed solely at commercial poultry.

S. Gallinarum-Pullorum is uncommonly reported in psittacines (Georgiades and Iordanidis 2002; Allgayer et al 2008). One report describes septicaemia caused by *S.* Pullorum in a cockatoo (Liow 1978), and another describes a serological response to *S.* Pullorum in wild and captive blue-fronted Amazon parrots (Deem et al 2005).

Paratyphoid Salmonellae

Paratyphoid salmonellae have a wide variety of pathological effects in poultry which vary between serotypes and strains (Barrow et al 1987; Okamura et al 2001; Roy et al 2001;). In other avian species, paratyphoid infections are almost always subclinical (Allgayer et al 2008).

Young chicks are highly susceptible to paratyphoid salmonellae and infection is associated with illness and high rates of mortality. Infection of adult birds with large doses of paratyphoid salmonellae may cause no signs of illness (Humphrey et al 1989).

Reports of paratyphoid infections in psittacines include outbreaks of *S.* Typhimurium in a collection of rainbow lorikeets and black winged lories (Shima and Osborne 1989) and in a zoological collection of lories and lorikeets (Ward et al 2003). There was also a report of a *S.* Typhimurium U286 infection in African grey parrots following importation into the United Kingdom (Anonymous 1981). The author proposed that poor hygiene, poor management, and stress from the time of capture to importation contributed to these cases.

Experimental infection of budgerigars and an African grey parrot with *S.* Typhimurium indicated a short incubation period (Schulze and Frede 1977). The incubation period in most outbreaks is more than 1 week (Ward et al 2003).

S. Typhimurium DT44 has been rarely isolated and there are no reports of the organism infecting avian species (Mackie et al 1996; Orós et al 1998; Sumner 2002).

S. arizonae

Avian arizonosis occurs throughout the world and has been associated with considerable losses in commercial turkey operations (Mayeda et al 1978; Crespo et al 2004). *S. arizonae* is most frequently seen in turkeys although infections of chickens and other avian species have been described (Edwards et al 1947; Silva et al 1980).

S. arizonae has been recovered from a variety of free-living avian species with no associated clinical disease (Windingstad et al 1977; Winsor et al 1981). *S. arizonae* has been described as the cause of a fatal hepatitis in a captive psittacine (Orós et al 1998) and has been reported from individual caged parrot, macaw, canaries, and sulphur-crested cockatoo (Edwards et al

1956; Edwards et al 1959; Panigrahy et al 1979; Orós et al 1998). The latter case followed shortly after the introduction of iguanas to the premises. These also became diseased and were found to be infected with the same organism. In addition, *S. arizonae* has been isolated from other reptiles (Sharma et al 1970; Cambre et al 1980) and a variety of mammals (Edwards et al 1956; Edwards et al 1959; Sharma et al 1970). *S. arizonae* is also recognised as an opportunistic human pathogen in immunocompromised individuals (Guckian et al 1967; Johnson et al 1976; Weiss et al 1986; Kelly et al 1995).

Naturally infected chickens show nervous signs including ataxia, torticollis, and opisthotonus (Youssef and Geissler 1979). Post mortem examination of infected individuals revealed septicaemia, enlargement of the gall bladder, engorgement of the uterus with urates, and unabsorbed yolk sac. Clinical signs in psittacines associated with *S. arizonae* are similar to those described in turkeys and broilers (Orós et al 1998). However, eye lesions similar to those described in broilers with natural and experimental *S. arizonae* infection have not been reported in psittacine birds (Orós et al 1998).

Other salmonellae

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

S. Dublin is host adapted to cattle with limited infections occurring in other species. There are a small number of reports of *S.* Dublin in poultry but reports of the organism in psittacine birds have not been located.

21.1.5. Hazard identification conclusion

S. Gallinarum-Pullorum and *S. arizonae* are uncommon in psittacines although birds may be carriers of the organism without developing clinical signs. Therefore *S.* Gallinarum-Pullorum and *S. arizonae* are identified as potential hazards in live psittacines.

S. Abortusovis and *S.* Typhimurium DT44 have never been reported in birds and are therefore not identified as potential hazards in live psittacines.

S. Dublin is host adapted to cattle and has never been reported in psittacines. It is therefore not identified as a potential hazard in live psittacines.

A limited pool of paratyphoid *Salmonella* spp. has been described in surveys overseas, the vast majority of which are recognised to be present in this country. There is no evidence to suggest that the exotic *Salmonella* serotypes found associated with psittacines overseas are any more pathogenic than the serotypes recognised as present in New Zealand. Paratyphoid salmonellae are identified not to be a potential hazard in the commodity.

21.2. RISK ASSESSMENT

21.2.1. Entry assessment

Psittacines may be subclinically infected with *S.* Gallinarum-Pullorum and *S. arizonae* and recovered birds may still be carrying the organisms. These birds would not show clinical signs that would be detected during clinical examination. Therefore, the likelihood of entry of *S.* Gallinarum-Pullorum or *S. arizonae* with imported live psittacines is assessed to be non-negligible.

21.2.2. Exposure assessment

S. Gallinarum-Pullorum and *S. arizonae* have been recovered from a variety of avian species, reptiles and mammals. It is reasonable to assume that free-living species in New Zealand could be exposed to the organisms from contact with an infected bird or contact with infected aviary waste. Furthermore, movement of birds could result in widespread transmission. Therefore, the likelihood of exposure of *S.* Gallinarum-Pullorum or *S. arizonae* is assessed to be non-negligible.

21.2.3. Consequence assessment

Wild and domestic birds and animals could become infected with *S.* Gallinarum-Pullorum and *S. arizonae* if they have contact with aviaries or waste products from aviaries or if infected birds escape from captivity. This could result in outbreaks of disease and production losses. Transmission to humans could occur following contact with psittacines and psittacine faeces, leading to sporadic cases of salmonellosis. *Salmonella* have been associated with a variety of disease syndromes in immunocompromised people, including gastroenteritis, septicaemia, and localised infections (Guckian et al 1967). Introduction of antibiotic resistant *Salmonella* strains could result in difficulties in treatment of human cases.

Therefore, the consequences of introduction of *S.* Gallinarum-Pullorum and *S. arizonae* are assessed to be non-negligible.

21.2.4. Risk estimation

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

21.3. RISK MANAGEMENT

21.3.1. Options

One or more of the following options could be considered in order to effectively manage the risks

Option 1

Psittacines could be imported without restrictions, provided they are healthy and show no clinical signs of infection.

Option 2

Psittacines could be required to have originated from premises where there have been no laboratory-confirmed cases of salmonellosis due to *S.* Gallinarum-Pullorum or *S. arizonae* for a period of 2-3 years.

Option 3

Psittacines for importation could be required to meet the standards set out in the OIE *Terrestrial Animal Health Code* for the importation of domestic birds. Article 10.7.2 of the current *Code* (OIE 2011a) recommends, for the importation of domestic birds, that:

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

i. showed no clinical sign of fowl typhoid and pullorum disease on the day of travel;
ii. come from establishments which are recognised as being free from fowl typhoid and pullorum disease; and/or
iii. have been subjected to a diagnostic test for fowl typhoid and pullorum disease with negative results; and/or
iv. were kept in a quarantine station for not less than 21 days prior to travel.

Option 3 could be extended to include *S. arizonae*.

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22. *Riemerella anatipestifer*

22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

Riemerella anatipestifer is a gram-negative, nonmotile, nonspore-forming rod, previously known as *Pfeifferella anatipestifer* (Hendrickson and Hilbert 1932), *Moraxella anatipestifer* (Bruner and Fabricant 1954) and *Pasteurella anatipestifer* (Segers et al 1993; Sandhu 2008).

22.1.2. OIE list

Not listed

22.1.3. New Zealand status

In New Zealand, an organism tentatively classified as *Pasteurella anatipestifer* was isolated from an incident of disease in ducks involving high mortality and pathology consistent with "new duck disease" in 1974 (Anonymous 1974). A further case, in which four of 16 ducks died in 1990, was consistent with *P. anatipestifer* infection although no isolates were definitively identified (Orr 1990). This history suggests that *R. anatipestifer* is likely to be present in New Zealand. However, given that no further isolates of this organism have been recorded since 1974, and that there are 21 different serotypes recognised globally (Sandhu 2008), it is reasonable to assume that only low virulent serotypes may be present in this country.

22.1.4. Epidemiology

R. anatipestifer is widely distributed around the world and is recognised, most commonly, when it causes disease in intensively reared ducks. Disease caused by *R. anatipestifer* is also known as new duck disease, duck septicaemia, anatipestifer syndrome, anatipestifer septicaemia, and infectious serositis (Sandhu 2008).

Infection with *R. anatipestifer* is considered to be primarily a disease of ducks and geese although disease outbreaks have been reported in turkeys (Zehr and Ostendorf 1970; Helfer and Helmboldt 1977; Frommer et al 1990). *R. anatipestifer* has also been uncommonly recovered from various other domestic and wild bird species (Rosenfeld 1973; Sandhu 2008). The only report of *R. anatipestifer* in a psittacine is that from a single budgerigar of unknown health status (Hinz et al 1998).

The severity of disease caused by *R. anatipestifer* varies widely depending on the strain of the organism, the infectious dose, the age of the host, and the route of exposure (Sarver et al 2005; Sandhu 2008). Twenty one serotypes have been described, with different serotypes predominating in different geographical locations (Sandhu and Leister 1991; Sandhu 2008). Divergence of these serotypes contributes to low cross-protection against different strains and variations in virulence factors, resulting in mixed infections of more than one serotype of *R. anatipestifer* in the same individual (Yu et al 2008).

Transmission is considered to occur via the respiratory route or through skin wounds, and wild birds and arthropods (*Culex* mosquitoes) have also been suggested (Eleazer et al 1973; Smith et al 1987; Cooper 1989). Infection is followed by an incubation period of 2-5 days before clinical signs are seen.

Clinical signs include listlessness, neurological signs, respiratory signs, ocular and nasal discharge, septicaemia, and death, with a mortality rate of between 5 to 75% (Zehr and Ostendorf 1970; Frommer et al 1990; Sandhu 2008). Post mortem findings are typically those of acute or chronic septicaemia, characterised by fibrinous pericarditis, perihepatitis, airsaccultitis, and meningitis (Zehr and Ostendorf 1970; Helfer and Helmboldt 1977; Smith et al 1987).

R. anatipestifer is reasonably resistant, surviving for 13 days in tap water and 39 days in poultry litter (Bendheim and Even-Shoshan 1975).

R. anatipestifer can be isolated from exudates and from lesions, and immunofluorescent procedures can be used to identify *R. anatipestifer* in tissue or exudates (Sandhu 2008). Both the agglutination test and ELISA can be used to detect serum antibodies, ELISA is the more sensitive of the two but is not serotype-specific (Sandhu 2008).

22.1.5. Hazard identification conclusion

There has only been one reported individual case of *R. anatipestifer* in psittacines. *R. anatipestifer* is a disease predominantly affecting ducks and psittacines could be regarded as accidental hosts. Exotic serotypes of *R. anatipestifer* are therefore not identified as a potential hazard in live psittacines.

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23. *Chlamydia psittaci*

23.1. HAZARD IDENTIFICATION

23.1.1. Aetiological agent

Nine Chlamydia species are currently recognised, namely *Chlamydia trachomatis*, *C. muridarum*, *C. pecorum*, *C. pneumoniae*, *C. psittaci*, *C. suis*, *C. abortus*, *C. caviae*, and *C. felis* (Kuo et al 2010).

Avian chlamydiosis is caused by *Chlamydia psittaci* (formerly *Chlamydophila psittaci*). Eight serovars of *Chlamydia psittaci* are currently recognised; six of these (A to F) occur in avian species and appear to be relatively host specific (Andersen and Vanrompay 2008). Avian *C. psittaci* strains are also classified into seven genotypes (A to F, and E/B) (Geens et al 2005b).

23.1.2. OIE list

Avian chlamydiosis is an OIE listed disease.

23.1.3. New Zealand status

Chlamydia psittaci is endemic in psittacine and pigeon populations in New Zealand (Cairney 1954; McCausland et al 1972; Bell and Schroeder 1986). Laboratory investigations between 1984 and 1985 identified *C. psittaci* isolates from budgerigars, parakeets, pigeons, rosellas, and cockatiels (Bell and Schroeder 1986). *C. psittaci* was also isolated from healthy New Zealand keas shortly after importation into the United Kingdom (Johnson et al 1984). Ornithosis in New Zealand feral pigeons is considered to have a prevalence rate of between 9.5% and 25% (Motha et al 1995).

It is not known which serovars of *C. psittaci* are present in New Zealand. However, the patterns of host preference of *C. psittaci* serotypes and the evidence of widespread infections of pigeons and psittacines in New Zealand is consistent with the presence of serotypes A and B in New Zealand. Other serovars (C, D, E, and F) are considered to be exotic.

23.1.4. Epidemiology

Chlamydia psittaci infection has been confirmed in more than 460 avian species in 30 orders (Kaleta and Taday 2003). Certain serovars or genotypes are usually associated with specific types of birds. Genotype A is usually associated with psittacines, genotype B with pigeons, genotype C with ducks and geese, and genotype D with turkeys (Andersen and Vanrompay 2008; Harkinezhad et al 2009). Genotype E has a more diverse host range and has been isolated from a variety of avian species, including turkeys, ducks, pigeons, ostriches, rheas (Harkinezhad et al 2009) and one Dinghy budgerigar (Duan et al 1999). Genotype F has been obtained from a single American parakeet (Andersen 1997) and a Belgian fattening turkey (Van Loock et al 2005). Genotype E/B is usually isolated from ducks but has also been demonstrated in pigeons, turkeys, and psittacines (Geens et al 2005a; Harkinezhad et al 2007; Harkinezhad et al 2009). Genotype E/B reacts with both the serovar E- and B-specific monoclonal antibodies, and can only be distinguished from genotypes E and B by *ompA* sequencing or by genotype-specific real-time PCR (Harkinezhad et al 2007).

In birds, *C. psittaci* produces a systemic infection, which varies according to the species and age of the bird, and the strain of *Chlamydia* (Sareyyupoglu et al 2007; Kuo et al 2010). Typical clinical signs in a susceptible host infected with a highly virulent strain include respiratory signs, mucopurulent nasal and conjunctival discharge, diarrhoea, polyuria, and dullness (Sareyyupoglu et al 2007). Strains of low virulence may produce no, or mild, clinical signs. Clinical signs in psittacines often include eye and nasal discharges or swelling, laboured breathing, diarrhoea, poor appetite, lethargy, 'fluffed up' appearance, weakness, and mortality (Harkinezhad et al 2007; OIE 2008). Subclinical infections can occur with strains of both low and high virulence (Sareyyupoglu et al 2007). Infection of birds with genotype E/B resulted in no clinical signs, except for turkeys which showed mild respiratory signs, suggesting this strain is less virulent (Harkinezhad et al 2007).

Many birds become chronically infected and show no clinical signs until stressed. These birds often shed *C. psittaci* intermittently and serve as a source of infection for humans and other birds (Harkinezhad et al 2007; Sareyyupoglu et al 2007; OIE 2008).

Psittacosis (in humans) is characterised by early flu-like symptoms which may be followed by severe systemic disease with interstitial pneumonia and encephalitis (Kuo et al 2010). Infected humans typically develop headache, chills, malaise and myalgia, with or without signs of respiratory involvement (OIE 2008). Psittacosis infection with genotype E/B was also unnoticed in the persons involved (Harkinezhad et al 2007).

Necropsy of affected birds will often reveal multifocal hepatic necrosis, spleen and liver enlargement, fibrinous airsacculitis, pericarditis, and peritonitis (OIE 2008). There may also be unusual feather coloration from altered metabolism in chronically infected birds (Rodolakis and Mohamad 2010).

Transmission of *C. psittaci* occurs through inhalation of contaminated material, with large numbers of chlamydiae found in the respiratory tract exudate, feather dust, and faeces of infected birds (Andersen 1996; Sareyyupoglu et al 2007; Schautteet and Vanrompay 2011). Transmission via arthropod vectors has also been suggested (Eddie et al 1962; Page et al 1975) and there is evidence for limited vertical transmission (Lublin et al 1996; Schautteet and Vanrompay 2011). The infection of eggs leads to embryonic death or to the hatching of live infected young birds (Rodolakis and Mohamad 2010).

In live birds, the preferred samples for diagnosis are pharyngeal and nasal swabs (Andersen 1996). Intestinal content, cloacal swabs, conjunctival scrapings, and peritoneal exudate can also be taken (OIE 2008). Proper handling of clinical samples is necessary to prevent loss of infectivity of chlamydiae during shipping and storage (OIE 2008).

C. psittaci serovars can be distinguished in specialised laboratories by a panel of serovar-specific monoclonal antibodies (Andersen 1991; Andersen 1997). Restriction fragment length polymorphism analysis and genotyping techniques are also available to distinguish serovars (Vanrompay et al 1997; Geens et al 2005a).

Diagnosis is by isolation and identification of the organism, demonstration of chlamydiae in tissues, complement fixation (CF) test, ELISA, and PCR (OIE 2008; Sachse et al 2008). PCR techniques are able to detect *C. psittaci* DNA in samples of tissues, faeces, and cloacal swabs, are sensitive and rapid, and allow molecular characterisation (Harkinezhad et al 2007; Sareyyupoglu et al 2007).

23.1.5. Hazard identification conclusion

C. psittaci serovars C and D have not been identified in Psittaciformes (Andersen 2005; Andersen and Vanrompay 2008) and are therefore not identified as potential hazards in the commodity.

C. psittaci genotypes A, B, E, F and E/B have been isolated from Psittaciformes. However, chlamydiae recovered from psittacine birds are almost always genotype A (Harkinezhad et al 2007; Andersen and Vanrompay 2008). *C. psittaci* serovars A and B are believed to be present in New Zealand and are therefore not identified as potential hazards in the commodity. *C. psittaci* genotype E/B causes no severe clinical symptoms in either birds or humans, has only been identified in psittacines in one study, and is serologically indistinguishable from serovar B (Harkinezhad et al 2007) which is present in New Zealand. Genotype E/B strain is therefore not identified as a potential hazard in the commodity.

As they are occasionally found in psittacines, *C. psittaci* serovars E and F are identified as a potential hazard in the commodity.

23.2. RISK ASSESSMENT

23.2.1. Entry assessment

The sources of infections of psittacines with chlamydial serovars other than A or B are unknown. However, the fact that they are only single records is consistent with these cases being adventitious infections from other reservoir hosts. Given the unique status of each report of *C. psittaci* serovars E and F from psittacine birds, the likelihood of their presence in psittacine birds is negligible and thus the likelihood of entry is assessed to be negligible.

23.2.2. Risk estimation

As the likelihood of entry is assessed to be negligible, the risk estimation is negligible and *Chlamydia psittaci* is not assessed to be a risk in live psittacines. Therefore risk management measures cannot be justified.

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24. Coccidia

24.1. HAZARD IDENTIFICATION

24.1.1. Aetiological agent

Apicomplexan protozoa in the genera *Eimeria*, *Isospora*, *Cryptosporidium*, and *Sarcocystis*.

Six species of *Eimeria* have been described in psittacines; *E. dunsingi*, *E. haematodi*, *E. psittacina*, *E. aratinga*, *E. amazonae*, and *E. ochrocephalae* (Greiner and Ritchie 1994; Gartrell et al 2000; Hofstatter and Kawazoe 2011).

Two species of *Isospora* have been described in psittacines; *I. melopsittaci,* and *I. psittaculae* (Chakravarty and Kar 1946; Bhatia et al 1973).

Three species of *Cryptosporidium* have been described in birds; *C. baileyi*, *C. meleagridis*, and *C. galli* (Muller 2010).

Sarcocystosis in psittacines is caused by *S. falcatula* (Levine 1986). *S. oliverioi* has also been recorded in a green-rumper parrotlet (*Forpus passerinus*). However, this is considered to be a synonym of *S. falcatula* (Odening 1998; Greiner 2008).

24.1.2. OIE list

Not listed

24.1.3. New Zealand status

At least 20 species of coccidia are known to occur in birds in New Zealand (McKenna 2010). The only coccidia recorded in psittacine species in New Zealand are *Cryptosporidium* sp. in a lovebird, *Eimeria* sp. in a kakapo, and *Toxoplasma gondii* in a kaka (McKenna 2010).

An unidentified sarcocyst has been observed on one occasion in New Zealand as an incidental finding in a native wood pigeon (Johnstone and Cork 1993). There are no records of sarcocysts in psittacines in this country.

24.1.4. Epidemiology

Eimeria spp.

It has been estimated that 45,000 different species of *Eimeria* exist, but compared with other coccidian parasites *Eimeria* spp. are highly host specific. Infection of psittacines is uncommon, and usually associated with only two species; *E. dunsingi* and *E. haematodi* (Greiner and Ritchie 1994; Gartrell et al 2000). *E. aratinga*, *E. amazonae*, and *E. ochrocephalae* are each limited to a single host species (orange-fronted conure, *Aratinga canicularis*, and yellow-crowned Amazons, *Amazona ochrocephala*, respectively) (Page and Haddad 1995; Gartrell et al 2000; Hofstatter and Kawazoe 2011).

Birds infected with *Eimeria* may show no clinical signs unless stressed (Greiner and Ritchie 1994; Gartrell et al 2000; Doneley 2009). Affected birds may be lethargic with weight loss and diarrhoea, and severely affected birds often die (Doneley 2009).

Isospora spp.

Isospora spp. are also highly host specific and only two species have been described in psittacines; *I. melopsittaci* and *I. psittaculae* (Chakravarty and Kar 1946; Bhatia et al 1973). *Isospora* spp. infection is usually inapparent and found in most parrot species around the world (Page and Haddad 1995; Doneley 2009). Severe infections are generally associated only with very young or immunocompromised birds (Page and Haddad 1995) and affected birds may develop melena, depression, weight loss, and diarrhoea (Greiner and Ritchie 1994; Doneley 2009).

Cryptosporidium spp.

Cryptosporidium spp. have been identified in at least six species of birds in New Zealand, including an outbreak in lovebirds (Belton and Powell 1987; McKenna 2010). Three main species are found in birds; *C. baileyi*, *C. meleagridis*, and *C. galli* (Muller 2010). Although cryptosporidiosis mainly infects the intestinal tract, renal and respiratory *Cryptosporidium* infections have also been diagnosed. The main clinical signs associated with enteric cryptosporidiosis are diarrhoea and enteritis (Muller 2010).

Cryptosporidium spp. can infect any epithelial surface, including the gastrointestinal, respiratory, and urinary tracts, the conjunctival sac, and the bursa (Doneley 2009). Infections are not host specific. However, movement from birds to mammals is not described and there is no zoonotic potential (Greiner and Ritchie 1994; Doneley 2009). *Cryptosporidium* is generally considered a secondary pathogen and in most situations it causes severe infections only in immunocompromised hosts (Greiner and Ritchie 1994; Doneley 2009). Clinical signs vary depending on the route of infection but may include depression, dehydration, anorexia, diarrhoea, coughing, sneezing, and nasal discharge (Doneley 2009).

Sarcocystis falcatula

Sarcocystis falcatula has an obligatory two-host life cycle. Sexual reproduction and sporogeny occurs in the intestines of the definitive host (opossum, *Didelphis virginiana*) followed by shedding of sporocysts in the faeces. The intermediate host (bird) ingests the sporocysts and asexual reproduction occurs resulting in sarcocyst development in striated muscle (Hillyer et al 1991; Greiner and Ritchie 1994; Doneley 2009). The cycle is completed when the definitive host eats the intermediate host and ingests the sarcocysts (Doneley 2009).

Sarcocystis falcatula has been associated with acute deaths in a variety of psittacine species although pathogenicity appears to depend on the species of bird and the infective dose of the parasite (Greiner and Ritchie 1994). In Old World Psittaciformes, asexual replication may cause fatal occlusion of blood vessels and many species (including budgerigars (*Melopsittacus undulatus*), cockatiels (*Nymphicus hollandicus*), African grey parrots (*Psittacus erithacus erithacus*), and various species of cockatoos (*Cocatua* spp.)) are highly susceptible to infection (Greiner and Ritchie 1994; Ladds 2009). In contrast, New World Psittaciformes are relatively resistant to *S. falcatula* (Ecco et al 2008).

Sarcocystosis may present in parrots in three different clinical forms; acute pulmonary, muscular, or neurologic disease (Hillyer et al 1991; Doneley 2009). Infections are usually peracute and present as sudden death (Ladds 2009). If clinical signs occur prior to death, they

are characterised by severe dyspnoea, yellow-pigmented urates, lethargy, anorexia, and neurologic signs (Greiner and Ritchie 1994; Ladds 2009).

Sarcocystis falcatula uses the North American opossum (*Didelphis virginianu*) as the definitive host (Hillyer et al 1991; Dubey et al 2000; Ecco et al 2008). However, recently the opossum *D. albiventris*, in Argentina, and *D. marsupialis*, in Brazil, have also been identified as definitive hosts of *S. falcatula* (Ecco et al 2008). *S. falcatula* appears to be restricted to North and South America where the definitive host is found (Dubey et al 1999; 2000).

24.1.5. Hazard identification conclusion

An *Eimeria* species has been described in a kakapo in New Zealand (McKenna 2010). *Eimeria* spp. are highly host specific and only a few have been recorded in psittacines. There is no evidence that more pathogenic species of *Eimeria* occur in other countries and *Eimeria* spp. are therefore not identified as a hazard in the commodity.

Isospora spp. are found in the intestinal mucosa of most parrot species around the world and are usually not associated with clinical signs of disease (Doneley 2009). There are very few published reports of *Isospora* sp. infection in psittacines and there is no evidence to suggest that *Isospora* spp. in other countries are more pathogenic than those already in New Zealand. *Isospora* spp. are therefore not identified as a hazard in the commodity.

A *Cryptosporidium* species has been already been described in psittacines in New Zealand (Belton and Powell 1987; McKenna 2010). *Cryptosporidium* spp. are not host specific and have been identified in multiple species of birds in New Zealand. There is no evidence that more pathogenic species of *Cryptosporidium* occur in other countries and *Cryptosporidium* spp. are therefore not identified as a hazard in the commodity.

Psittacines which survive infection with *Sarcocystis falcatula* may develop muscular sarcocysts containing infectious bradyzoites. *Sarcocystis falcatula* is therefore identified as a potential hazard in the commodity.

24.2. RISK ASSESSMENT

24.2.1. Entry assessment

New World Psittaciformes are relatively resistant to *Sarcocystis falcatula* and infection may go unnoticed despite the development of muscular sarcocysts. Therefore the likelihood of *Sarcocystis falcatula* entry with live psittacines is assessed to be low.

24.2.2. Exposure assessment

Sarcocystis falcatula undergoes an obligatory two-host life cycle. The definitive host of *Sarcocystis falcatula* is the opossum (*Didelphis* spp.) which is not present in New Zealand. Therefore the life cycle cannot be completed and the likelihood of exposure is assessed to be negligible.

24.2.3. Risk estimation

Since the likelihood of exposure is assessed to be negligible, the risk estimation is negligible and *Sarcocystis falcatula* is not classified as a risk in live psittacines. Therefore risk management measures cannot be justified.

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25. Haematozoa

25.1. HAZARD IDENTIFICATION

25.1.1. Aetiological agent

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

25.1.2. OIE list

Not listed

25.1.3. New Zealand status

Haemoproteus sp., *H. danilewsky*, *Leucocytozoon* sp.*, L. fringillinarum, L. tawaki*, *Plasmodium* sp. *P. cathermerium, P, elongatum,* and *P. relictum* have been recorded in New Zealand (McKenna 2010).

Of the above species, *Plasmodium relictum* and *Plasmodium* sp. were reported in psittacines.

As only 16 species of mosquitoes (Holder and Brown 1999) and no *Culicoides* spp. have been identified in New Zealand, the generally low prevalence of haematozoa may be due to a paucity of suitable vectors.

25.1.4. Epidemiology

A number of species of blood parasites belonging to the genera *Haemoproteus*, *Leucocytozoon*, *Plasmodium* and *Trypanosoma* have been described in birds world-wide.

Most haematozoa seem to live in balance with their avian hosts. Some may be opportunistic pathogens, particularly when they occur in combination with other infections, and association with disease does not necessarily indicate pathogenicity. The pathogenicity of the parasites has seldom been proven by experimental infection and it appears that most species are non-pathogenic or mildly pathogenic in the species in which they have evolved but may be highly pathogenic in naïve species. The best example of this is the introduction of the mosquito vector *Culex quinquefasciatus* and its associated parasite *Plasmodium relictum* into Hawaii which played a key role in the extinction of approximately half of the endemic bird species (van Riper et al 1986). Some species may successfully adapt to new parasites as demonstrated by the Hawaiian honeycreeper which developed a reproductive advantage following the malaria outbreak (Kilpatrick et al 2006).

Haemoproteus spp.

Haemoproteus spp. are the most frequently occurring of the avian blood parasites, although infection in psittacines is uncommon. The *Haemoproteus* genus contains nearly 200 species and varieties and has been described in over 1,700 species of birds in 110 families (Rae 1995). Transmission occurs via *Culicoides* midges and hippoboscid flies, which serve as both vectors and intermediate hosts (Rae 1995; Doneley 2009).

Haemoproteus spp. have been reported in several psittacine species including cockatoos and macaws. Infection in psittacines raised in a domestic environment has never been reported, leading to the suggestion of a lack of suitable insect vectors outside of the host's native habitat.

Haemoproteus is rarely pathogenic in psittacines (Rae 1995; Doneley 2009). Severe infections in stressed or immunocompromised birds may result in anaemia, anorexia, and depression, and once infected, a bird remains a carrier for life (Doneley 2009).

Leucocytozoon spp.

The *Leucocytozoon* genus contains over 100 species and is divided into two subgenera based on the vector species. *Leucocytozoon* is spread by blackflies (*Simulium* species) and *Akiba* is spread by *Culicoides* species of midges.

The only currently known member of the subgenus *Akiba* is *Leucocytozoon (Akiba) caulleryi* which has not been reported in psittacines. *Leucocytozoon* spp. infection has been reported occasionally in psittacines but more commonly in waterfowl, turkeys, raptors, and passerines. Psittacines tend to develop an aberrant infection, characterised by the presence of megaloschizonts in heart, gizzard, and skeletal muscles without circulating gametocytes (Rae 1995; Doneley 2009).

Various studies have confirmed that the majority of *Leucocytozoon* infections are subclinical (Doneley 2009; Ladds 2009). Clinical signs including anorexia, ataxia, depression, dehydration, and haemoglobinuria occasionally develop and the aberrant form of infection results in an acute illness of 24 to 48 hours duration, followed by death (Rae 1995). Fatal infections have been reported in budgerigars, lovebirds (*Agapornis* spp), quaker parrots (*Myiopsitta*), king parrots (*Alisterus*), Australian parakeets (*Neophema, Psephotus, Polytelis*), rosella parakeets (*Platycercus*), and kakariki (*Cyanoramphus*) (Rae 1995).

Plasmodium spp.

Plasmodium spp. are responsible for avian malaria and around 40 species have been described throughout the world (Atkinson and LaPointe 2009). Mosquitoes of the *Culex*, *Aedes*, and occasionally *Anopheles* genera act as both intermediate hosts and vectors for all *Plasmodium* spp.

Plasmodium has the widest host range of the haemoprotozoa, and malaria has been described in a number of companion and aviary birds. The parasites are most commonly found in passerines, which are the definitive (and reservoir) host (Atkinson and LaPointe 2009; Doneley 2009) although malaria in psittacines occurs rarely (Rae 1995). Infections in a green-wing macaw (*Ara chloroptera*) and a blue-headed parrot (*Pionus menstruus*) have been reported, and subclinical infections in cockatoos (*Cacatua* spp) have been attributed to non-pathogenic strains (Rae 1995).

The pathogenicity of various *Plasmodium* species ranges from non-pathogenic to virulent but various studies have confirmed that the majority of *Plasmodium* infections are subclinical (Atkinson and LaPointe 2009; Ladds 2009). Significant clinical findings, when they occur, are largely due to direct localisation of the protozoa in the endothelium or parenchymal cells of many organs (Ladds 2009). Infected birds may die suddenly without showing clinical signs or birds may exhibit depression, anorexia, vomiting, dyspnoea, and haemoglobinuria

(Doneley 2009). Pathologic findings include haemolytic anaemia, leukocytosis, and lymphocytosis. Splenomegaly, hepatomegaly, and haemorrhage have been reported at post mortem (Rae 1995). Many birds will become lifelong carriers (Doneley 2009).

At least three *Plasmodium* spp. including *Plasmodium relictum*, have been described in New Zealand, indicating that, for these species at least, suitable insect vectors are present. The New Zealand native mosquito *Culex pervigilans* is known to be a competent vector of *Plasmodium* spp. (Massey et al 2007), as is the introduced species *Culex quinquefasciatus* (Holder and Brown 1999). *Plasmodium relictum* is already widespread in New Zealand and has caused mortality in some species, including the New Zealand dotterel (*Charadrius obscurus*) and the threatened native mohoua (*Mohoua ochrocephala*) (Derraik 2006; Tomkins and Gleeson 2006).

Trypanosoma spp.

Several species of trypanosomes have been described in birds but they may all be referred to as *Trypanosoma avium* (Rae 1995; Ladds 2009). Although avian trypanosomes appear to have a worldwide distribution, the incidence of infection is usually low (Rae 1995). Canaries and finches appear to be the most commonly infected avian species but trypanosomes have also been reported in parakeets, macaws, and a conure (Peirce and Bevan 1977; Greiner and Ritchie 1994; Rae 1995).

Transmission is via blood-sucking arthropods. A range of insects have been implicated as intermediate hosts, and may also serve as active or passive vectors (Ladds 2009).

There is little evidence of pathogenicity, and infections are usually light (Rae 1995; Taylor et al 2007; Ladds 2009). Rare reports suggest that heavy infections may be associated with illness although clinical signs have not been reported in infected birds, and no gross or histological lesions have been described for avian trypanosomiasis (Rae 1995).

25.1.5. Hazard identification conclusion

At least two species of *Haemoproteus* are known to be present in New Zealand. *Haemoproteus* are rarely pathogenic in psittacines (Rae 2005; Doneley 2009) therefore it is unlikely that more pathogenic strains exist overseas that would be present in live psittacines. *Haemoproteus* is therefore not identified as a hazard in the commodity.

Trypanosoma avium is non-pathogenic (Taylor et al 2007) and therefore is not identified as a hazard in the commodity.

Since some species of *Leukocytozoon* and *Plasmodium* have not been described in New Zealand and indigenous birds may be highly susceptible to infection with these haematozoa, exotic species of the genera *Leukocytozoon* and *Plasmodium* are identified as a potential hazard in the commodity.

25.2. RISK ASSESSMENT

25.2.1. Entry assessment

Although there is usually a low prevalence (less than 3%) of haematozoa in psittacine species (Rooney et al 2001), a variety of haematozoa occur in many countries, and the likelihood that imported psittacines could be carrying exotic haematozoa is assessed to be non-negligible.

25.2.2. Exposure assessment

Escapes of psittacines and subsequent contact with other birds and parasite vectors cannot be excluded. Haematozoa are not contagious and require the presence of a suitable vector for transmission. Several species of haematozoa have been described in New Zealand so suitable vectors for at least some must be present. The likelihood of transmission of haematozoa to competent vectors and then to other birds is assessed to be non-negligible.

25.2.3. Consequence assessment

Budgerigars have been imported in the past with no adverse effects and the likelihood of importing parasites pathogenic to psittacines is low. However New Zealand's indigenous birds are potentially a naïve and highly susceptible population in relation to the introduction of exotic haematozoa and vector systems for at least some haematozoa are already present. The possible threat to New Zealand native birds has been demonstrated by an incident in which four keas were transferred from New Zealand to Malaysia and all died within 3 weeks from infection with *Plasmodium* (Bennett et al 1993). The consequences of introduction of exotic *Plasmodium* and *Leukocytozoon* species are assessed to be non-negligible.

25.2.4. Risk estimation

Since the entry, exposure, and consequence assessments are all assessed to be non-negligible, the risk estimation is non-negligible and exotic haematozoa of the genera *Plasmodium* and *Leukocytozoon* are classified as a risk in live psittacines. Therefore, risk management measures may be justified.

25.3. RISK MANAGEMENT

25.3.1. Options

Leucocytozoonosis may be diagnosed by detection of non-pigmented elongated gametocytes in peripheral blood (Ladds 2009) or by PCR (Cosgrove et al 2006).

The diagnosis of *Plasmodium* infection is by PCR, serology, or detection of schizonts and gametocytes in peripheral blood smears. The organism can be identified within erythrocytes (where it may displace the nucleus), thrombocytes, leucocytes, and endothelial cells (Ladds 2009). There is much debate as to the preferred technique and ideally several techniques would be used in conjunction (Cosgrove et al 2006). Control of infection is best accomplished by elimination of vector species (Rae 1995) and treatment with chloroquine and primaquine may be effective (Doneley 2009).

The following points were considered when drafting risk management options:

- Birds are long term carriers of many haematozoa and quarantine would not be a useful measure to prevent their introduction.
- The reliability of diagnostic techniques (serology, PCR, and microscopy) are widely debated.
- The *Code* does not contain any recommendations relating to haematozoa.

One or a combination of the following options could effectively manage the risk of haematozoa in live psittacines.

Option 1

Blood samples from birds to be imported (individually or pooled) could be examined by PCR using suitable primers for haematozoa. Birds (or flocks) which test positive could be further investigated to determine the species of haematozoa involved. All birds (or flocks) infected with exotic haematozoa 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 2

Blood smears from birds to be imported (individually or pooled) could be examined microscopically for species of *Plasmodium* and *Leukocytozoon*. All birds (or flocks) infected with *Plasmodium* and/or *Leukocytozoon* could be disqualified.

Option 3

As noted above, there is much debate as to the preferred diagnostic technique and ideally several techniques would be used in conjunction (Cosgrove et al 2006). Therefore, blood samples from birds to be imported could be subject to examination by PCR using suitable primers for haematozoa and examined microscopically for species of *Plasmodium* and *Leukocytozoon*. Birds determined to be infected using either of these tests could be disqualified.

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26. Microsporidiosis

26.1. HAZARD IDENTIFICATION

26.1.1. Aetiological agent

The phylum Microsporidia contains approximately 150 genera and 1200 species of obligate, intracellular, eukaryotic parasites closely related to fungi (Snowden and Phalen 2004; Sak et al 2010).

Encephalitozoon hellem is the most common microsporidian identified in psittacines and other birds (Gelis and Raidal 2006; Sak et al 2010). Less frequently identified are *Encephalitozoon cuniculi* and *Enterocytozoon bieneusi* (Sak et al 2010).

26.1.2. OIE list

Not listed

26.1.3. New Zealand status

There are no reports of *Encephalitozoon hellem* or *Enterocytozoon bieneusi* infection in New Zealand birds. *Encephalitozoon cuniculi* is present in rabbits in New Zealand (Orr 1997; Anonymous 2011) but has not been reported in any avian species.

26.1.4. Epidemiology

Microsporidia are nucleated, single-celled, obligate intracellular parasites that were originally classified with the protozoa but have recently been reclassified with the fungi (Didier 2005). They are found in a variety of vertebrate and invertebrate hosts of virtually all phyla, and are particularly prevalent in fish and insects (Snowden and Phalen 2004; Didier and Weiss 2006).

Most cases of microsporidial infections in wild birds are caused by *Encephalitozoon hellem* and birds are considered to be the primary hosts of this organism (Snowden and Phalen 2004; Sak et al 2010). Other microsporidial species are less commonly identified in birds, and are generally incidental findings (Greiner and Ritchie 1994; Sak et al 2010). Microsporidiosis in psittacine species is uncommon. *E. cuniculi* has been reported on one occasion in a cockatiel (*Nymphicus hollandicus*) (Kašičková et al 2007), and *E. hellem* has been reported in love-birds (Snowden et al 2000), parrots (Pulparampil et al 1998), a yellow-streaked lory (Suter et al 1998), and budgerigars (Black et al 1997).

Microsporidiosis is recognised as an opportunistic disease in immunocompromised people (Black et al 1997; Didier 2005), as well as in otherwise healthy immune-competent individuals, for example infection in travellers as a result of foodborne outbreaks (Didier and Weiss 2011). Fourteen species of microsporidia are currently known to infect humans, including *Encephalitozoon hellem*, *E. cuniculi,* and *Enterocytozoon bieneusi*, and these species have been identified in water sources as well as in wild, domestic, and food-producing farm animals (Didier 2005; Didier and Weiss 2006).

In birds, microsporidial infection is often associated with concurrent infection, inadequate husbandry, stress, or immaturity (Snowden and Phalen 2004; Doneley 2009). The majority of infections in birds are self limiting and inapparent, although develop depression, decreased appetite, and weight loss are the most commonly reported clinical signs (Snowden and Phalen

2004). Progressive anorexia, weight loss, respiratory disease and diarrhoea in an infected Amazon parrot have been reported over a one-month period (Greiner and Ritchie 1994). Reports in other avian species have described enteritis, hepatitis, nephritis, keratoconjunctivitis, sinusitis, and lower respiratory tract infections (Doneley 2009).

Encephalitozoon infections are believed to occur through ingestion or inhalation of spores, with primary infections developing in the epithelium of the small intestine or respiratory tracts respectively (Didier 2005). *E. hellum* is usually found in the liver, intestine, and kidney, but it has also been identified in the eyes, lungs, and spleens of infected birds (Gelis and Raidal 2006; Sak et al 2010). Post mortem findings include pale, swollen kidneys and an enlarged, mottled liver (Greiner and Ritchie 1994).

Microsporidial spores shed into the environment are highly resistant and may remain infective for many months (Black et al 1997). Diagnosis of infected birds is by PCR or histopathology; and treatment with albendazole (for *Encephalitozoon* infections) and fumagillin (for *Enterocytozoon* infections) may be of benefit (Franzen et al 1998; Didier 2005; Doneley 2009).

26.1.5. Hazard identification conclusion

Microsporidiosis in humans occurs worldwide, with prevalence rates as high as 50% depending on the geographic region, method of diagnosis, and population demographics (Didier and Weiss 2006). The source of most infections is unknown and the potential for waterborne and foodborne spread is being increasingly recognised (Didier and Weiss 2006).

Only sporadic cases of infection have been reported in birds and infections are usually sub-clinical. Furthermore, the species of microsporidia associated with psittacines are also found in humans and there are no mechanisms to prevent the introduction of these organisms by infected humans. Therefore, Microsporidia species are not identified as a potential hazard in the commodity.

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27. Flagellated protozoa

27.1. HAZARD IDENTIFICATION

27.1.1. Aetiological agent

Flagellated protozoa in the genera *Cochlosoma*, *Giardia*, and *Spironucleus* (formerly *Hexamita*).

Several species of *Giardia* have been reported in Psittaciformes including *G. psittacae*, and *G. duodenalis* (Filippich et al 1998).

27.1.2. OIE list

Not listed

27.1.3. New Zealand status

There are no reports of *Cochlosoma* spp. infection in New Zealand birds.

Giardia spp. have been found in at least 7 species of birds in New Zealand, including a kaka (McKenna 2010).

Spironucleus sp. is also known to occur in New Zealand, but has not been reported in psittacines (McKenna 2010).

27.1.4. Epidemiology

Giardia spp.

Giardia infection can affect all age groups, but young birds are more susceptible and high mortalities can occur (Filippich et al 1998). Adult budgerigars and cockatiels are largely subclinical carriers of *Giardia* spp. and frequently shed infectious cysts in the absence of clinical signs (Muller 2010). Infected birds may appear depressed and anorexic and display signs of malodorous mucoid diarrhoea, dehydration, malabsorption, weight loss, persistent feather picking, and pruritus (Filippich et al 1998; Greiner and Ritchie 1994; Doneley 2009).

In Australia, there have been reports of *G. duodenalis* infection in healthy wild-caught sulphur-crested cockatoos (*Cacatua galerita*) and *G. psittacae* in budgerigars (*Melopsittacus undulatus*) (Filippich et al 1998). Often *Giardia* infection is a coincidental finding.

Giardia psittaci is morphologically distinct from other species of *Giardia* and is not transmissible to mammals (Erlandsen and Bemrick 1987). It is mainly a pathogen of budgerigars and is considered to be endemic in Australia and probably New Zealand. *G. psittaci* is not considered zoonotic, but some avian *Giardia* isolates can infect mammals (Filippich et al 1998).

Cochlosoma spp.

Cochlosoma spp. are flagellated protozoa with a sucking disc similar to *Giardia*. In Australia *Cochlosoma* spp. are commonly found in the intestinal tract of cockatiels (Doneley 2009) and have also been reported in a healthy lorikeet (Ladds 2009). As in avian giardiasis, clinical

signs of *Cochlosoma* infection may be vague and lesions non-specific (Ladds 2009). Infection may cause enteritis with diarrhoea, weight loss, and death, and has been associated with pruritus and feather picking behaviour.

Spironucleus meleagridis

Spironucleus meleagridis (formerly *Hexamita* spp.) has been reported in healthy Australian king parrots, cockatiels, splendid grass parakeets, and lories (Greiner and Ritchie 1994; Doneley 2009). Demonstration of the parasite is common in bird faeces, and does not appear to cause a problem unless the birds are stressed (Greiner and Ritchie 1994). Infection can result in chronic, often intractable, diarrhoea, weight loss, and death (Doneley 2009).

Spironucleosis has been identified as an important contributing pathogen to a fatal syndrome of weight loss and diarrohoea in wild galahs in Queensland (Doneley 2012, cited by Gartrell 2012) although the role of other infectious organisms in this syndrome is unclear (Grillo et al 2012).

27.1.5. Hazard identification conclusion

Psittacines were imported into New Zealand for almost 150 years up to 1997 and several species of Australian psittacines have already established wild populations in New Zealand. *Giardia* spp. and *Cochlosoma* spp. are commonly found in Australian psittacines, and signs of infection are often absent, and when present are non-specific. It is therefore likely that *Giardia* spp. and *Cochlosoma* spp. that are associated with psittacines are already present in New Zealand and they are therefore not identified as a hazard in the commodity.

Spironucleus species are not host specific and a *Spironucleus* sp. has been reported in an ostrich in New Zealand (McKenna 2010). There is no evidence that more pathogenic species of *Spironucleus* occur in other countries and *Spironucleus meleagridis* is therefore not identified as a hazard in the commodity.

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28. Helminths

28.1. HAZARD IDENTIFICATION

28.1.1. Aetiological agent

The higher taxa containing helminths of veterinary importance are Nemathelminthes (roundworms), Platyhelminthes (flatworms), and Acanthocephala (thornyheaded worms) (Taylor et al 2007a).

The phylum Nemathelminthes has six classes but only one of these, the Nematoda, contains worms of parasitic significance (Taylor et al 2007a). The phylum Acanthocephala is closely related to the Nematoda (Taylor et al 2007a). The phylum Platyhelminthes contains the two classes of parasitic flatworms, the Trematoda (flukes) and the Cestoda (tapeworms) (Taylor et al 2007a).

28.1.2. OIE list

Not listed

28.1.3. New Zealand status

The nematode parasites *Ascaridia* spp., *A. platycerci*, *Capillaria* spp., *C. plagiaticia*, *Cyathastoma cacuatua*, *Microtetrameres nestoris*, *Oxyspirura* spp., *Procynea kea*, and *Syngamus trachea* have been recorded from psittacine birds in New Zealand (McKenna 2010)

The cestode parasites *Pulluterina nestoris* and *Stringopotaenia psittacea* have been recorded from psittacine birds in New Zealand (McKenna 2010).

28.1.4. Epidemiology

Psittacine species are susceptible to a large number of helminths although there are few reports of infestation causing disease (Huffman 2008). Several surveys of healthy birds have failed to demonstrate any helminths (Rooney et al 2001; Stone et al 2005; Masello et al 2006).

Nematodes (roundworms)

Ascarids are the most common nematode found in captive birds, particularly in budgerigars and cockatiels (Greiner and Ritchie 1994; Fedynich 2008). Several species are known to infect psittacines including *Ascaridia columbae*, *A. galli, A. platycerci*, *Baylisascaris procyonis*, and *Heterakis* spp. (Greiner and Ritchie 1994; Balicka-Ramisz et al 2007; Bartlett 2008). *Capillaria* spp. are also found in aviary birds, particularly in macaws, budgerigars, and gallinaceous birds (Greiner and Ritchie 1994; Yabsley 2008; Ladds 2009). Filarial nematodes, including *Pelecitus* spp., *Chandlerella* spp., *Cardiofilaria* spp., and *Eulimdana* spp. have also been reported in psittacine birds (Greiner and Ritchie 1994; Ladds 2009). Exotic respiratory tract nematodes have not been reported in Psittaciformes (Fernando and Barta 2008; Ladds 2009).

Nematodes may live in the small intestine, proventriculus and ventriculus, the surface and chambers of the eye, body cavity, subcutaneous connective tissues, heart, and air sacs of birds (Greiner and Ritchie 1994; Ladds 2009).

Most nematodes have a direct life cycle although some species require an intermediate host, usually an insect. Nematode eggs can remain infectious in the environment for several months in moist warm environments and are resistant to disinfectants (Greiner and Ritchie 1994).

Unlike in other bird species (particularly gallinaceous birds), the clinical effects of nematodes in psittacines are generally not severe (Greiner and Ritchie 1994). Clinical signs vary depending on the species and location of the parasite, but may include malabsorption, weight loss, vomiting, diarrhoea, intussusceptions, bowel occlusion, encephalitis, ataxia, torticollis, and death (Armstrong et al 1989; Greiner and Ritchie 1994; Wolf et al 2007; Churria et al 2011). Eye infections can result in eyelid spasms, conjunctivitis, and chemosis. Filarial nematodes are generally nonpathogenic (Bartlett 2008).

Some nematodes are host specific, but others are known to infect a wide range of avian host species with potential for transmission between domestic and wild birds (Fagerholm and Overstreet 2008; Fedynich 2008; Yabsley 2008).

Trematodes (flukes)

Avian trematodes have indirect life cycles involving both sexual and asexual phases of reproduction. Sexual reproduction occurs in a vertebrate definitive hosts and asexual reproduction occurs in one or two aquatic intermediate hosts (mollusc, amphibian, or fish) (Huffman 2008). The first, second, and definitive hosts must all be present in the environment in order to complete the cycle (Huffman 2008). Due to the aquatic life cycle, trematodes mainly infect waterbirds (McLaughlin 2008; Ladds 2009). Psittacines kept in captivity have minimal access to potential intermediate hosts and are unlikely to be infested with trematodes.

A wide range of trematodes occur in avian species and the majority are not associated with significant disease (Huffman 2008). Flukes are rarely found in psittacine birds (Greiner and Ritchie 1994; Lee et al 2005). There is a single case reported of a schistosome in a nanday conure that died after showing weight loss, anorexia, and blood-tinged diarrhoea (Greiner and Ritchie 1994). Trematodiasis has also been reported in cockatoos (Luthgen and Schutze 1981; Quesenberry et al 1986, as cited in Greiner and Ritchie 1994; Ladds 2009). Despite these isolated cases, trematodes are not recognised as a cause of disease in Psittaciformes (Huffman 2008).

Trematodes may reside in the liver or in the vasculature (Greiner and Ritchie 1994). Pathology may be due to a direct host-parasite interaction, mechanical insult resulting in tissue damage, or by the ingestion of host tissue. Host immune responses cause inflammation and immune-mediated pathology. The lesions are closely related to the anatomical location of the parasite (Huffman 2008). Clinical changes associated with liver fluke infections include hepatomegaly, depression, anorexia, mild anaemia, weight loss, diarrhoea, hepatic necrosis, elevated liver enzymes, and death (Greiner and Ritchie 1994).

Cestodes (tapeworms)

Cestodes are extremely common parasites of birds. Most are host specific for a single or few closely related birds (McDougald 2008; McLaughlin 2008). In psittacines, infections are most common in African grey parrots, cockatoos, and eclectus parrots (Greiner and Ritchie 1994).

Cestode life cycles are indirect and one or two intermediate hosts are required to complete the cycle (McLaughlin 2008). As for trematodes, transmission of cestodes with aquatic life cycles tends to be restricted to either marine or freshwater environments (McLaughlin 2008). Transmission of cestodes with terrestrial life cycles is uncommon in birds that do not have access to the ground (Greiner and Ritchie 1994).

Most cestode species infect the intestine, but some may be found in the caecae or gizzard (Greiner and Ritchie 1994; McLaughlin 2008).

Cestodes are not recognised as an important cause of disease in Psittaciformes (Ladds 2009). Infected birds seldom display clinical signs, and cestodes are not considered a problem unless present in massive numbers or in malnourished or debilitated hosts (Greiner and Ritchie 1994; McLaughlin 2008). Adult cestodes may cause damage to the gizzard lining, intestinal blockage, localised damage to the intestinal wall at the site of attachment, or irritation of the intestinal lining (McLaughlin 2008). Clinical signs are nonspecific and include emaciation, weakness, diarrhoea, and haemorrhage.

Acanthocephala

Acanthocephalans have an indirect life cycle and require vertebrates as definitive hosts and arthropods as intermediate hosts. Only eggs exist outside of a host, free in the environment and transmission from one definitive host to another requires that appropriate invertebrate intermediate hosts ingest eggs (Richardson and Nickol 2008). Acanthocephalans are relatively specific for intermediate hosts. Consequently, importation of exotic species seldom results in continuation of the life cycle through transmission to other animals (Richardson and Nickol 2008).

Acanthocephalans generally cause little overt pathology in avian hosts although serious adverse effects are possible under certain circumstances (Richardson and Nickol 2008). Species of acanthocephalans are harboured by birds of relatively few taxonomic orders, including Anseriformes, Charadriiformes, Passeriformes, Falconiformes, and Strigiformes (Richardson and Nickol 2008). A single case of Acanthocephalan infection has been reported in an Australian parrot in 1972 (Kelly and Finnie 1972). However, there are no subsequent reports and Acanthocephalans are not considered to be parasites of psittacine species.

Diagnosis of helminths is performed by examination of faeces using visual, floatation and sedimentation methods and larval culture (Taylor et al 2007c). A wide range of anthelmintics are available and effective for treatment (Greiner and Ritchie 1994; Taylor et al 2007b,).

28.1.5. Hazard identification conclusion

Internal parasites (nematodes, trematodes and cestodes) of psittacines are not well documented, however many species may be exotic to New Zealand.

Nematodes may be found in healthy psittacines and do not require an intermediate host. They are therefore identified as a potential hazard in live psittacines.

Trematodes are not recognised as an important cause of disease in Psittaciformes. However, there are a few reports of trematodiasis in psittacine species. Birds kept in captivity have minimal access to aquatic intermediate hosts and are unlikely to be infested with trematodes. Trematodes are therefore identified as a potential hazard in live psittacines.

Cestodes may be found in healthy psittacines. Cestode life cycles are indirect and infections are uncommon in captive birds that do not have access to the ground. Nevertheless they are identified as a potential hazard in live psittacines.

The literature contains only a single report of an acanthocephalan infection in a psittacine species. Furthermore acanthocephalans are host specific for their intermediate host. They are therefore not identified as a potential hazard in live psittacines.

28.2. RISK ASSESSMENT

28.2.1. Entry assessment

Psittacines may be infected with internal parasites (nematodes, trematodes, and/or cestodes) without displaying clinical signs of disease and thus remain undetected during routine clinical examination. The likelihood of entry of exotic helminths with live psittacines is therefore assessed to be non-negligible.

28.2.2. Exposure assessment

Imported psittacines will be confined indoors or in aviaries, with limited contact with other birds. Birds kept in captivity have minimal access to aquatic intermediate hosts and are unlikely to be infested with trematodes. However, faeces and waste materials from aviaries may be disposed of at sites accessible to wild birds and this material may be contaminated with helminth eggs. It is not known whether competent intermediate hosts exist for exotic trematodes and cestodes. Some helminth eggs are able to survive for extended periods in the environment. The likelihood of exposure to helminths from live psittacines is therefore assessed to be low.

28.2.3. Consequence assessment

Cestodes are generally non-pathogenic and trematodes are not recognised as an important cause of disease in Psittaciformes. However, internal parasitism of psittacine birds can result in signs of disease, reduced growth rates, and death. Some nematodes are relatively host specific, but others are known to infect a wide range of avian host species with potential for transmission between domestic and wild birds. The consequences of entry and exposure of helminths to avian species are therefore assessed to be non-negligible. Helminths of psittacine birds are not zoonotic therefore there are negligible consequences to human health.

28.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for helminths is non-negligible and they are classified as risks in live psittacines. Therefore risk management measures can be justified.

28.3. RISK MANAGEMENT

28.3.1. Options

The following factors were considered when drafting options for the effective management of the helminths in live psittacines:

- The number of parasites considered is large and many helminths of psittacines are yet to be described. Therefore specific options for each parasite are not practical and general measures designed to cover all helminths are necessary.
- Many helminth species are of minor importance as parasites of psittacines. However, deaths due to helminthiasis are reported to be common in captive birds and may be under-reported in veterinary literature (Gartrell 2012).
- All species considered can be treated effectively with anthelmintics. Treatment with praziquantel is effective for virtually all trematodes and cestodes, effective dosage regimens should be used.
- All the relevant parasites are diagnosed by examination of faeces. To cover all species, faeces should be examined by visual inspection, floatation, sedimentation methods, and larval identification techniques.

One or a combination of the following options could be used for the effective management of helminths in live psittacines:

Option 1

All birds to be imported could be treated as recommended by the manufacturer with an effective dose of anthelmintic that is efficacious against nematodes (ivermectin or other), trematodes (praziquantel), and cestodes (praziquantel) within 7 days of travel.

Option 2

Birds to be imported could be subjected to faecal tests within 14 days of travel by a laboratory approved by the Veterinary Authority of the exporting country and birds with positive results could be treated according to Option 1.

Option 3

In addition to Option 1, faecal samples could be examined by a laboratory approved by the Veterinary Authority of the exporting country 7-10 days following treatment, with negative results. If results are not negative, treatment could be repeated (using different anthelmintics if necessary) until a negative result is obtained. Travel could be within 7 days of a final negative faeces examination.

Option 4

As for Option 3 but with an additional anthelmintic treatment 3 days before travel.

Option 5

Birds to be imported could be quarantined for at least 3 weeks immediately before travel. 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. Birds could be subjected to anthelmintic treatment immediately before entry to quarantine, and while in quarantine the birds could be subjected to the treatment and testing procedures outlined in Options 1-4.

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29. Ectoparasites

29.1. HAZARD IDENTIFICATION

29.1.1. Aetiological agent

The parasites considered in this section are:

- **Fleas;** order Siphonaptera. There are over 2500 recognised flea species although very few have been described in psittacine species (Wall and Pitts 2005).
- **Lice;** order Phthiraptera. Lice species that parasitise birds belong to two of the chewing lice suborders: Amblycera and Ischnocera. There are at least 253 recognised genera of avian lice, the majority of which belong to the suborder Ischnocera, often called "feather lice" (Clayton et al 2008).
- **Mites**; order Acarina. Birds host a rich diversity of mite species, some dwelling on the surface of feathers but others inhabiting skin, nostrils, and respiratory passages (Proctor 2003). There are approximately 2000 named species of true feather mites, belonging to three families: Analgoidea, Freyanoidea, and Pterolichoidea. They are almost all obligatory symbiotes of birds, living in and on the skin (dermicoles), inside the quills (syringicoles), and on the surface of feathers (plumicoles) (Proctor 2003). Other feather mite species belong to the families Cheyletiellidae, Analgesidae, and Dermoglyphidae. In addition to feather mites, birds may be parasitised by mites from a wide range of families including Knemidokoptidae, Epidermoptidae, Thrombiculidae (chigger mites), Harpyrhynchidae, Syringophillidae (quill mites), Laminosioptidae (cyst mite), and Hypoderatidae (hypoderatid mites). Species of nasal mites belong to the three families Rhinonyssidae, Speleognathidae, and Turbinoptidae (Pence 2008).
- **Ticks**; order Acarina. There are hundreds of species of avian ticks which belong to two families: Argasidae (soft ticks) and Ixodidae (hard ticks) (Soulsby 1968).

29.1.2. OIE list

Not listed

29.1.3. New Zealand status

Five tick genera (*Amblyomma* spp., *Boophilus* spp., *Dermacentor* spp., exotic species of *Ixodes* spp., and *Rhipicephalus* spp.), and two screwworm genera (*Chrysomyia* spp. (Old World screwworm fly), and *Cochliomyia* spp. (New World screwworm fly)) are notifiable organisms in New Zealand (Tana et al 2011).

Records of avian ectoparasites in New Zealand are incomplete and are continuing to grow. To date, there are records of (Bishop and Heath 1998; Heath 2010):

- 86 species of feather mites (10 from psittacines)
- 9 species of nasal mites (none associated with psittacines)
- 19 species of other mites (3 from psittacines)
- 30 species of fleas (9 from psittacines)
- 15 species of ticks (3 from psittacines)

At least 12 species of lice are recognised in New Zealand, none of which were found on psittacines (Pilgrim 1976; Horning et al 1980; Palma 1991; Palma and Price 2000; Banks and Palma 2003; Palma and Price 2004; Palma and Price 2005).

29.1.4. Epidemiology

Species of fleas, chewing lice, mites, and ticks are found on captive and wild psittacines worldwide (Schwan et al 1983; Freitas et al 2002; Bochkov and O'Connor 2003; Stone et al 2005; Blank et al 2007; Hernandez et al 2007; Sychra et al 2007; Abo-Shnaf et al 2008; Dik 2010; Mastropaolo et al 2011). New species of ectoparasites are commonly reported on psittacines and other bird species (Dabert et al 2006; Dabert et al 2007; Hernandez et al 2007; Mironov and Dabert 2007) and it has been estimated that less than one fifth of the feather mites alone have been currently identified (Valim et al 2011).

Although many ectoparasite species are host specific, aberrant hosts housed together with birds of other species or families may be susceptible to infestation with new species (Sychra et al 2007). Psittacines may also be aberrant hosts of ectoparasites from other species (such as reptiles) with which they share their enclosure. Some of these may be vectors of disease for non-avian hosts (such as heartwater in cattle) (Scofield et al 2011).

Fleas

Fleas are ubiquitous and are the most common ectoparasite of companion animals. Fleas are host-preferential rather than host-specific and may be found on a range of hosts (Kelly et al 2005). Reports of flea infestation in psittacines are extremely scarce, but psittacines are susceptible to a range of avian flea species (Bishop and Heath 1998; Blank et al 2007; Heath 2010).

In other species, heavy flea infestations may result in anaemia, debilitation, and skin disease but no such reports exist for psittacine species. Some fleas are capable of transmitting disease causing agents (such as *Yersinia pestis* and *Francisella tularensis*) and may act as intermediate hosts for cestode and filarial infections (Wall and Pitts 2005).

Fleas must be associated with the host for survival. After feeding on the host's blood, mating occurs and the females lay eggs that drop off in the host's environment (Greene 2006). The eggs and larvae cannot withstand major variations in environmental conditions such as temperature and relative humidity.

Lice

The majority of avian lice species belong to the Ischnocera suborder, which are often called "feather lice" as they feed primarily on feathers and skin. The remainder of avian lice belong to the suborder Amblycera, which feed on feathers and blood (Clayton et al 2008).

There are several reports in the literature of psittacine birds infested with lice, either as primary hosts or as aberrant hosts (Freitas et al 2002; Stone et al 2005; Sychra 2005; Sychra et al 2007; Clayton et al 2008; Saxena et al 2009; Dik 2010). However, none of these have been associated with signs of disease.

Lice are generally not pathogenic and heavy infestations are usually indicative of other clinical problems (Davies 2007). Many avian lice are site- and host-specific and are found on

only one host genus or species. Others are less specific and occur on multiple host genera, family, or even orders (Clayton et al 2008).

Chewing lice are obligate, permanent parasites that complete their entire life cycle on the body of the host. Transmission among hosts often requires physical contact between birds, but some species are capable of "hitchhiking" on other insects such as hippoboscid flies (Clayton et al 2008).

Chewing lice are normally found in small, subclinical infections that are kept in check by regular host grooming. When present in large numbers, lice can cause extensive feather and skin damage, ill-thrift, decreased productivity, and mortality (Stone et al 2005). Anaemia is an uncommon finding (Clayton et al 2008). Chewing lice can also have indirect effects on the host by acting as vectors or intermediate hosts of other parasites although none have been specific to or associated with psittacines.

Mites

Hundreds of mite species from several families are recognised as parasitic to almost all bird species worldwide, however only a few are recognised as pathogens (Pence 2008). Mites are the most common ectoparasite found on psittacine species and prevalence rates (of feather mites, respiratory mites, and knemiodocoptes) range from 6.5% to 18% (Hernandez et al 2007; Bruno and Albuquerque 2008; Parsani and Momin 2009).

Mite infestations range in clinical severity from non-pathogenic to life-threatening, causing diseases of the skin, feathers, subcutaneous tissues, or respiratory tract. Clinical conditions resulting from acariasis in birds include anaemia, dermatitis, mange, feather damage or loss, and granulomatous inflammation in the subcutaneous tissues and in the respiratory tract (Pence 2008).

Host specificity varies dramatically across different species, genera, and families of mites. Cross transmission of many mite species is theoretically possible between wild and domestic bird hosts, although this is not known to occur commonly (Pence 2008).

Most mites complete their entire life cycle on the body of their avian host. Transmission between hosts may be by direct contact or at nest sites by actively motile larvae, nymphs, or adults (Pence 2008).

A few avian mite species have been associated with public health problems, including a transient dermatitis caused by dermanyssid mites and chigger dermatitis caused by larval trombiculids (Pence 2008). Chigger mites are also capable of transmitting the rickettsiosis, tsutsugamushi disease (scrub typhus) caused by *Orientia tsutsugamushi* (Pence 2008).

Clinical signs due to feather mites include irritation, ruffled plumage, and reduced feed intake (Baker 1996; Beck 2006). Knemidocoptic mange can result in cutaneous lesions of the face, legs and feet (Bruno and Albuquerque 2008; Grenz 2011). Respiratory mites may localise in paranasal sinus, trachea, and in the lungs (Hermandez et al 2007).

Ticks

Ticks are blood sucking parasites of mammals, birds, and reptiles. Some are important vectors of bacteria, rickettsiae, protozoa, and viruses, and others inject neurotoxins into their host causing paralysis and death (Loth 2005).

Ticks are uncommon on captive birds and usually attach around the head and eyelids (Forbes and Simpson 1993; Davies 2007).

Small numbers of ticks generally do not significantly affect the health of the bird (unless the ticks are vectors of disease), but heavy infestations can potentially cause severe clinical problems including anaemia, ill-thrift, poor growth, and mortality (Forbes and Simpson 1993; Stone et al 2005; Davies 2007).

Ticks may be classified as one-host ticks or multi-host ticks depending on whether they moult on or off the host.

29.1.5. Hazard identification conclusion

Mites

The common species of feather, respiratory and blood sucking mites, are already present in New Zealand (Bishop and Heath 1998; Davies 2007; Heath 2010). However there are thousands of avian mite species which are exotic, and some of these may induce more severe effects than the species already present.

Fleas

Fleas are not commonly found on psittacine birds, however psittacines are thought to be susceptible to a range of flea species, some of which have the potential to cause disease.

Lice

Lice are common in captive and wild birds although there are no records of lice causing disease in psittacine species. New species of ectoparasites are frequently reported on psittacines and the effects of these are not fully known.

Ticks

Ticks are an uncommon finding on captive birds, but can cause severe clinical problems when they do occur. Exotic tick species are notifiable organisms in New Zealand.

Although many ectoparasite species are host specific, aberrant hosts housed together with birds of other species or families may be susceptible to infestation with new species (Sychra et al 2007). Psittacines may also be aberrant hosts of ectoparasites from other species (such as reptiles) with which they share their enclosure. Some of these may be vectors of disease for non-avian hosts (Scofield et al 2011).

Fleas, mites, ticks and lice are therefore identified as a potential hazard in the commodity.

29.2. RISK ASSESSMENT

29.2.1. Entry assessment

Ectoparasites are widely distributed in the world and psittacine species may act as hosts (or aberrant hosts) to many species of fleas, mites, ticks, and lice. Overseas, the movement of captive birds has been implicated in the spread of ectoparasites into new areas (Nelson et al 1979; Schwan et al 1983). Most ectoparasites can be seen on visual examination and are likely to be detected during ante mortem examination. However, some can be small or well hidden (such as quill mites) and immature stages may not be obvious. Therefore the likelihood of entry of exotic species of fleas, mites, ticks and lice is assessed to be low.

29.2.2. Exposure assessment

Imported psittacines may be integrated into aviaries and may contact other birds at shows or pet shops. Although imported psittacine birds are likely to be held in aviaries or cages, the enclosures in which they are kept may not preclude contact with wild birds. The likelihood of exposure of wild native birds to external parasites through imported psittacines is low, given the isolated nature of most native psittacine populations, and the close contact required for transmission of the parasites. The Department of Conservation exercises control over private holdings of native psittacine birds, and it is unlikely that introduced exotic species would be mixed with captive native birds. However, native birds kept in captivity in contact with imported birds may be at risk and escapes and illegal releases are not uncommon. Additionally aviary waste products may be disposed of at sites accessible to wild birds.

The likelihood of exposure of caged pet birds and wild birds to exotic species of fleas, mites, ticks, and lice from infested imported psittacines is therefore assessed as non-negligible.

Recommended minimum biosecurity standards for domestic poultry producers (Poultry Industry Association of New Zealand 2007) include measures to minimise the biosecurity risk posed by wild birds. Such measures ensure that the likelihood of commercial poultry being exposed to free-living avian species is very low. It is therefore concluded that there is a negligible likelihood of commercial poultry being exposed to exotic species of fleas, mites, ticks and lice through infested wild birds.

29.2.3. Consequence assessment

Mites

Although some authors consider some feather mites to be harmless saprophytes, a more commonly expressed opinion is that feather mites cause distress and feather damage (Greve 1996) which can have a significant effect on wild, domestic, and commercial birds. Reports of psittacines infested with other species of mites are rare and the consequences of such infestations are not well known.

Fleas

Heavy flea infestations may result in anaemia, debilitation, and skin disease. Fleas may also transmit exotic disease causing agents and may act as intermediate hosts for species of cestode and filarial parasites. The effects of such organisms on the health of humans and animals could be severe.

Lice

Lice are generally not pathogenic and are common in both wild and captive birds in which they are usually host and site-specific. Heavy infestations are usually indicative of other clinical problems. There are no records in the literature of disease of psittacines caused by lice. However, lice can potentially cause feather damage which may impact pet and show birds. Lice are considered a minor problem in modern poultry operations because they are relatively easy to control. Avian lice are of little concern to humans because they cannot survive or reproduce off the body of the avian host and they do not transmit human pathogens.

Ticks

Heavy tick infestations may result in anaemia, debilitation, skin disease, and paralysis. Although ticks may harbour exotic diseases, no diseases of psittacines have been transmitted in this manner.

The impacts of mites, fleas, lice and ticks on the general health and well-being of show and pet birds are hard to measure but they are assessed as being non-negligible.

The consequences associated with the entry and establishment of exotic species of fleas, mites, ticks and lice are therefore assessed as non-negligible.

29.2.4. Risk estimation

Since the entry, exposure, and consequence assessments are non-negligible, the risk estimate for exotic species of fleas, mites, ticks and lice is non-negligible and they are classified as risks in the commodity. Therefore, risk management measures can be justified.

29.3. RISK MANAGEMENT

29.3.1. Options

Ectoparasites may be diagnosed by visual examination, deep skin scrapings, microscopic examination of feather shafts, and examination of brushing residues (Bruno and Albuquerque 2008; Pence 2008; Grenz 2011). Treatment of fleas, mites, lice and ticks is easy and effective and a wide range of insecticidal and acaricidal products are widely available (Sakar et al 2006; Davies 2007; Bruno and Albuquerque 2008; Beck 2006; Grenz 2011).

The following factors were considered when drafting options for the effective management of the ectoparasites in live psittacines:

- The number of ectoparasites considered is large and many species are yet to be described. Therefore specific options for individual species are not practical and general measures designed to cover all ectoparasites are necessary.
- Most ectoparasite species are of minor importance as parasites of psittacines.
- Ectoparasite species can generally be treated effectively with insecticides. In 2004, MAF Biosecurity New Zealand commissioned a review of the relative efficacy of acaracides. A series of studies were evaluated, looking closely at repellent effects, efficacy and duration of protection of acaricides belonging to seven chemical classes, either alone or as mixtures (Heath 2004).

- Ectoparasites may be diagnosed by visual examination and microscopic examination of the recovered debris following feather ruffling using a suitable insecticide dust or aerosol, or anaesthetic (Clayton and Walther 1997). Visual inspections are an important adjunct to insecticide treatment since treatment alone may not always be 100% effective.
- Ectoparasites of psittacines are not listed by the OIE and therefore there are no international standards for their control.

One or more of the following options could be used for the effective management of ectoparasites in live psittacines:

Option 1

All birds to be imported could be treated as recommended by the manufacturer with a suitable insecticide(s) that is efficacious against avian species of fleas, lice, mites, and ticks, within 7 days of travel. The treatment used could be integrated with the treatment for internal parasites.

Option 2

Birds to be imported could be thoroughly inspected immediately prior to export by visual inspection and microscopic examination of debris collected following a suitable feather ruffling technique and found to be free from fleas, lice, mites, and ticks. Birds could be further inspected upon arrival at the port of entry.

Option 3

Birds to be imported could be quarantined for at least 3 weeks immediately prior to travel. The pre-export quarantine premises could have smooth, painted walls, impermeable floors, and be regularly cleaned to remove all faeces and bedding materials. Floors, walls, and cages could be steam cleaned and disinfected with an insecticide(s) effective against all stages of fleas, lice, mites, and ticks prior to birds entering quarantine. Birds could be subjected to treatment with an effective insecticide(s) immediately prior to entering quarantine as described in option 1.

Option 4

The contents of the container in which the animal arrived could be inspected and determined free from fleas, lice, mites, and ticks. Birds found to be infested with fleas, lice, mites, or ticks could be transferred to a transitional facility and treated with a different insecticide(s) from that used previously. At the facility the container and its contents could be destroyed or thoroughly steam cleaned to remove any remaining parasites. All parasites found could be sent to a laboratory for identification. A biosecurity clearance could be given 48 hours after treatment when the inspector is satisfied that the animal and container are parasite-free.

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