

*Import Risk Analysis:*  
Budgerigars (*Melopsittacus undulatus*) from the United Kingdom

*DRAFT FOR PUBLIC CONSULTATION*



19 December 2008

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MAF Biosecurity New Zealand  
Pastoral House  
25 The Terrace  
PO Box 2526  
Wellington 6011  
New Zealand

Tel: 64 4 894 0100

Fax: 64 4 894 0731

Policy and Risk  
MAF Biosecurity New Zealand



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Approved for public consultation

A handwritten signature in black ink that reads 'Christine Reed'.

Christine Reed  
Manager, Risk Analysis  
MAF Biosecurity New Zealand

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# Contributors to this risk analysis

## 1. Authors

- N H Christensen      Avivet Ltd., Palmerston North, New Zealand
- Bob Worthington      Contractor, Risk Analysis, MAF Biosecurity New Zealand,  
Wellington

## 2. Internal Peer Review

- Stuart MacDiarmid      Principal International Adviser, Risk Analysis , MAF  
Biosecurity New Zealand, Wellington
- Howard Pharo      Team Manager, Risk Analysis (Animals), MAF Biosecurity  
New Zealand, Wellington
- Barbara Christensen      Adviser, Animal Imports, MAF Biosecurity New Zealand,  
Wellington
- Richard Norman      Incursion Manager, MAF Biosecurity New Zealand,  
Wellington
- Leone Basher      Senior Adviser, Animal Imports, MAF Biosecurity New  
Zealand, Wellington
- José Derraik      Senior Adviser, (Human Health), MAF Biosecurity New  
Zealand, Wellington
- Sandy Toy      Senior Adviser, (Indigenous Fauna), MAF Biosecurity New  
Zealand, Wellington
- Wlodek Stanislawek      Virologist, Investigation & Diagnostic Centre, MAF  
Biosecurity New Zealand, Wallaceville
- Rachel Somervell      Senior Adviser, Risk Analysis (Animals), MAF Biosecurity  
New Zealand, Wellington
- Lincoln Broad      Senior Adviser, Risk Analysis (Animals), MAF Biosecurity  
New Zealand, Wellington

## 3. External Scientific Review

- Simon M Shane      Adjunct Professor, Department of Poultry Science, North  
Carolina State University, USA
- Tom N Tulley      Professor of Zoological Medicine Louisiana State University,  
School of Veterinary Medicine, Baton Rouge, USA
- Jürgen E Lohr      Staatliches Tierärztliches Untersuchungsamt Aulendorf,

Löwenbreitestr, Germany

- Dennis Alexander Poultry Department, VLA Weybridge, New Haw, Addlestone,  
Surrey, United Kingdom
- G M Cross University Veterinary Centre, Diagnostic Services Laboratory,  
Faculty of Veterinary Science, The University of Sydney,  
Australia
- Stanley H Kleven Department of Avian Medicine, College of Veterinary Medicine,  
University of Georgia, Athens, USA
- Brett Gartrell Senior Lecturer in Avian and Wildlife Health Institute of  
Veterinary, Animal & Biomedical Sciences, Massey University,  
New Zealand
- E F Kaleta Institute für Geflügelkrankheiten, Justus-Leibig Universitas,  
Giessen, Germany
- Patricia McWhirter Highbury Veterinary Clinic, Burwood, Victoria, Australia

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## Executive Summary

This analysis examines the risks posed by infectious or parasitic agents when importing budgerigars from the United Kingdom.

Seventy nine organisms/diseases of concern of budgerigars are considered (Table 1). Of these 19 are classed as preliminary hazards and are subject to a risk assessment. As a result of this, a non-negligible risk is identified with the following hazards:

- Avian paramyxovirus 1 (low pathogenicity)
- Avian influenza (low pathogenicity)
- Pachecho's disease virus (Herpesvirus)
- Psittacine pox virus
- Rotavirus
- Exotic *Salmonella* spp.
- Coxiella burnetti* (Q fever)
- Protozoal blood parasites (Haematozoa)
- External parasites
- Internal parasites

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

# 1. Introduction

This analysis examines the risks associated with imports of live budgerigars from the United Kingdom.

There is currently no import health standard (IHS) for the importation of psittacine birds into New Zealand. It has been suggested that this situation may increase the risk of smuggling and the associated risk of introducing exotic diseases and that the development of an IHS may enable safe trade and reduce the motivation to smuggle such birds.

Budgerigars are native to Australia, and were first imported into Britain in 1840. The original imported birds have been transformed by selective line breeding into the modern exhibition budgerigar. This has involved the development of new colour and feather variations and an increase in size from an average 35 grams to over 60 grams. These birds are the internationally recognised show variety of the budgerigar and they may represent a distinct strain from the common pet budgerigar, but are also found as pets and in community aviaries.

The budgerigar is the most popular pet bird in New Zealand, where about 100,000 of these birds are bred each year. Despite their being freely imported from Australia for about 150 years up to 1997 and despite numerous reported escapes and deliberate releases, a feral population has not become established in this country. Besides New Zealand, budgerigars have been deliberately released without establishment in England, Hong Kong, Sicily, Japan, Colombia, Brazil and South Africa, but they did become established in Florida as a result of repeated deliberate releases in St Petersburg from 1951 onwards (Higgins 1999). The establishment of a breeding population in the wild in New Zealand resulting from sporadic escapes is therefore considered unlikely. This has important implications when considering the potential for disease transmission and effects on wildlife, which are likely to be much reduced compared with birds that are able to establish in the wild in New Zealand. In the case of highly bred exhibition budgerigars that are likely to be the imported, their ability to survive outside captivity is likely to be even more limited than pet budgerigars.

Budgerigars are native to Australia, and the export of wild caught birds has been prohibited by the Australian Wildlife Service for about 50 years. Various species of psittacine birds have been imported to New Zealand from Australia up to 1997. Imported birds have included Australian native birds kept as pets, such as sulphur crested cockatoos, as well as psittacine birds exotic to Australia that were imported into Australia prior to the imposition of quarantine restrictions in that country. Therefore, it is considered possible that some infectious and parasitic disease agents that occur in Australia may have been introduced into New Zealand through those bird imports. This appears to be the case with psittacine beak and feather disease which occurs commonly in rosellas and cockatoos that have established in New Zealand (Ha et al 2007). Whether other disease agents have been introduced in the same way is uncertain.

## 2. Scope

This analysis is limited to the infectious disease risks posed by the importation of live budgerigars from the United Kingdom. Genetic diseases are not considered.

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

The risk analysis is qualitative.

## 3. Commodity Definition

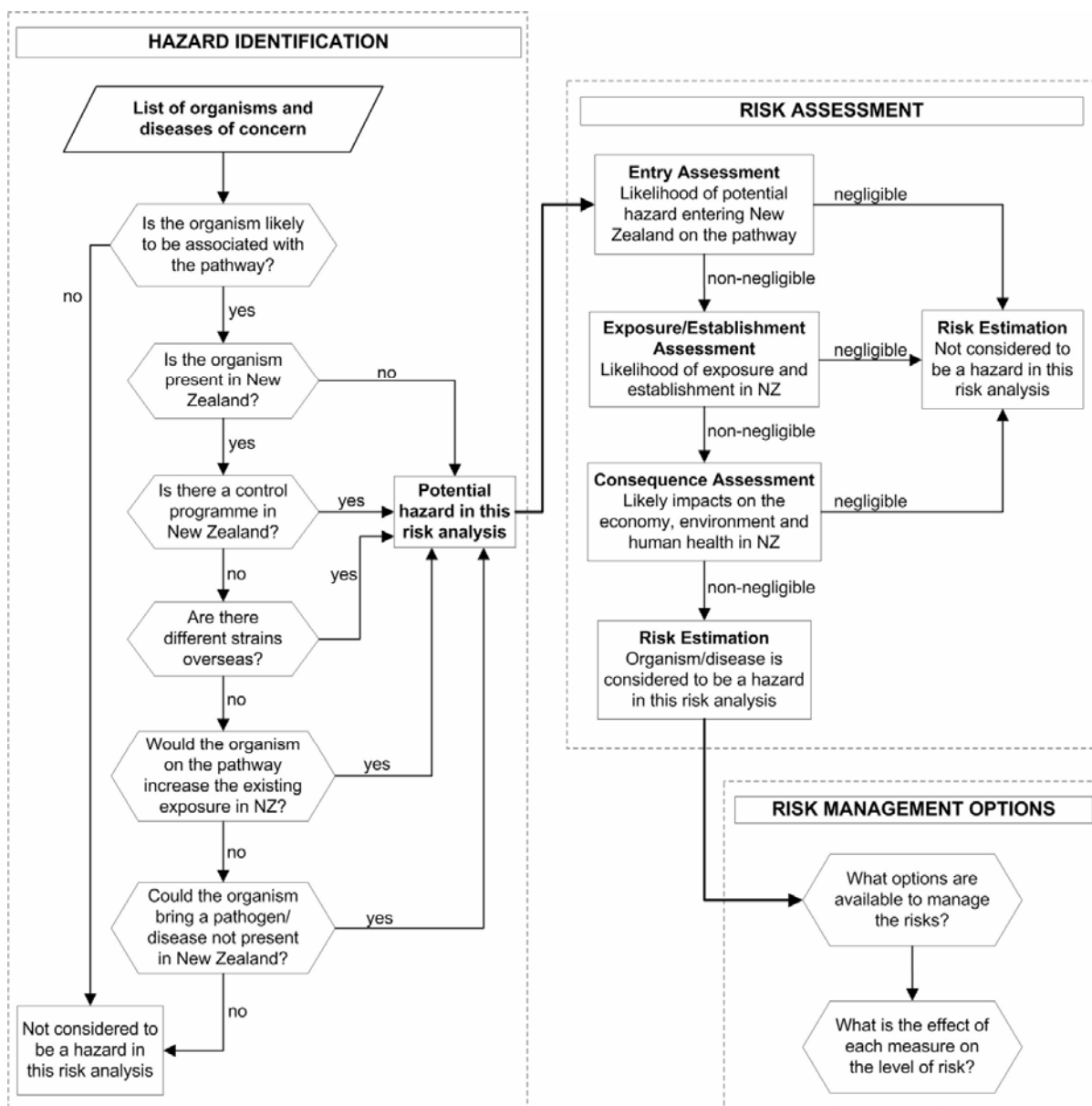
The commodity is defined as domestic budgerigars (*Melopsittacus undulatus*) from single closed donor flocks, maintained indoors in the United Kingdom. Birds will be sourced only from flocks that are breeders of exhibition type budgerigars and are inspected regularly by the United Kingdom Department for Environment, Food, and Rural Affairs (DEFRA). The flocks will be maintained as closed flocks with minimum introduction of birds of certified health status that have been strictly quarantined. Birds from such flocks will not have contact with other birds by being taken to shows and exhibitions. The premises of the exporting flock will have suitable quarantine facilities which are inspected and certified by DEFRA as suitable for quarantine of birds to be exported to New Zealand.

## 4. Risk Analysis Methodology

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

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

Figure 1. The risk analysis process.



#### 4.1. PRELIMINARY HAZARD LIST

The hazard identification process begins with the collation of a list of organisms likely to be associated with the commodity. Table 1 shows these organisms, together with some of the key information considered. This list was compiled from the diseases listed in the risk analysis for passerine hatching eggs (Simpson 2006), all the avian diseases mentioned in Overseas Market Access Requirements (OMARs) of all New Zealand's trading partners and diseases reported in the following text books:

11<sup>th</sup> Edition of *Diseases of Poultry* edited by Saif YM, Barnes HA, Glisson JR, Fadly AM, McDougald LR, and Swayne DE. Iowa State Press, Iowa, published 2003.

5<sup>th</sup> Edition of *Poultry Diseases* edited by Jordan FTW, Pattison MWB, Alexander DJ, and Faragher. T. Saunders Co. Ltd., London, published 2001.

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

*Avian Viruses. Function and Control* edited by Ritchie BW. Wingers publishing Incorporated, Lake Worth, Florida, published 1995.

Polyomavirus infection was added to the list as it was considered in the chicken meat import risk analysis (Ministry of Agriculture and Forestry 1999) at the request of the Department of Conservation (DoC). Polyomavirus was included in the DoC assessment of the risks faced by native parrots from exotic diseases and pests (Jackson et al 2000).

Table 1. Preliminary hazard list.

Pathogen	Occurs in NZ	Occurs in the UK	Recorded in budgerigars?	Strain variation NZ vs UK	Comments	Requires further consideration?
<b>Orthomyxoviruses</b>						
Highly pathogenic avian influenza virus (HPAI) H5/H7 strains	No***	Yes (OIE 2007)	Yes			Yes
Low pathogenic avian influenza viruses	Yes (Stanislawek et al 2002)	Yes	Yes (Alexander 1988; Imada et al 1980)	probably		Yes
<b>Paramyxoviruses</b>						
Newcastle disease virus, avian paramyxovirus 1 (APMV-1)	No***	No (OIE 2007)	Yes (Kaleta and Baldauf 1988)			No
Low virulence Newcastle disease virus (APMV-1)	Yes (Pharo et al 2000)	Yes	Yes (Kaleta and Baldauf 1988)	probably		Yes
Avian paramyxoviruses 2, 3, and 5	Some	Yes	Yes (Alexander & Gough 2003)			Yes
Avian paramyxoviruses 4, 6, 7, 8, and 9.	Some (Stanislawek et al 2001)	Yes	No			No
Pneumovirus (turkey rhinotracheitis, swollen head)	No*	Yes (Gough 2003)	No**			No
<b>Herpesviruses</b>						
Duck enteritis virus	No***	Yes (OIE 2007)	No**			No
Infectious laryngotracheitis virus	Yes****	Yes (OIE 2007)	No**			No
Marek's disease virus	Yes****	Yes (Purchase 1985)	No**			No
Psittacine herpes viruses Pacheco's disease, budgerigar herpes virus Amazon tracheitis virus	No (Alexander 1988)	Yes	Yes (Baker 1996a)	Yes		Yes

Pathogen	Occurs in NZ	Occurs in the UK	Recorded in budgerigars?	Strain variation NZ vs UK	Comments	Requires further consideration?
<b>Coronaviruses</b>						
Coronavirus enteritis virus	No	Yes	No			No
Infectious bronchitis virus	Yes****	Yes (OIE 2007)	No**			No
Psittacine corona virus	No	Yes (Gough et al 2006)	No (Gough et al 2006)			No
<b>Adenoviruses</b>						
Group I adenoviruses	Yes****	Yes	No**			No
Group II adenoviruses	No	Yes	No**			No
Group III Egg drop syndrome virus	Yes****	Yes	No**			No
<b>Avian pox viruses</b>						
Psittacine pox virus	Uncertain (King et al 2003)	Yes	No			Yes
<b>Circoviruses</b>						
Psittacine beak and feather disease virus	Yes (Anonymous 1994; Fraser et al 1999)	Yes	Yes(Albertyn et al 2004; Baker 1996b)			No
Chicken infectious anaemia virus	Yes****	Yes	No (Woods and Latimer 2003)			No
Pigeon circovirus	No*	Yes(Baker 1996a)	No(Shivaprasad et al 1994; Woods and Latimer 2003)			No
<b>Birnaviruses</b>						
Birnavirus (infectious bursal disease)	No (Bingham et al 2006)	Yes (OIE 2007)	No (Luckert and Saif 2003)			No
<b>Papovaviruses</b>						
Polyoma virus	Yes (Jacob-Hoff 2003)	Yes	Yes			No
Papilloma virus	No	Yes	No**			Yes
<b>Parvoviruses</b>						
Derzsy's disease virus	No***	Yes (Gough et al 1981)	No**			No
<b>Flaviviruses</b>						
West Nile virus	No (Spurr and Sandilant 2004)	Yes	Yes			Yes
Louping ill virus	No***	Yes	No**			No
Sindbis virus	No****	Yes (Buckley et al 2006; Buckley et	No**			No

Pathogen	Occurs in NZ	Occurs in the UK	Recorded in budgerigars?	Strain variation NZ vs UK	Comments	Requires further consideration?
		al 2003)				
<b>Reoviruses</b>						
Psittacine reovirus	Unknown	Yes (Manvell et al 2004; Pennycott 2004)	Yes (Manvell et al 2004; Pennycott 2004)			Yes
Rotavirus	Yes	Yes	Yes			No
Orbivirus	No (Duignan 2001)	Yes	Yes (Hirai et al 1979)			Yes
Other reoviruses	Yes****	Yes	No**			No
<b>Bunyaviruses</b>						
Nairovirus (Crimean-Congo haemorrhagic fever)	No***	No (Swanepoel and Burt 2004)	No**			No
<b>Borna virus group</b>						
Borna disease virus	No***	Yes (Reeves et al 1998)	No**		Cats in UK	No
<b>Picornavirus group</b>						
Avian encephalomyelitis virus	Yes****	Yes	No**			No
Avian nephritis virus	Yes (Howell 1992)	Yes	No (Imada and Kawamura 2003)			No
Duck hepatitis 1&3 (DVH 1 & 3) virus	No***	Yes	No (Woolcock 2003)		See also astrovirus	No
<b>Astroviruses</b>						
Astrovirus (DVH 2, turkey astrovirus)	No*	Yes	No **			No
<b>Hepatitis B virus</b>						
Hepadnavirus (duck virus hepatitis)	No	Yes	No**		Non-pathogen	No
<b>Retrovirus Group</b>						
Avian leucosis virus	Yes (Stanislawek 2001)	Yes	No**			No
Lymphoproliferative disease virus (LPDV)	No**	Yes	No**		Turkey (Payne 2002)	No
Reticuloendotheliosis virus	Yes (Howell 1992)	Yes	No**			No
<b>Unknown aetiology</b>						
Macaw wasting disease/proventriculitis	No***	Yes	Yes		Large psittacines	Yes
Transmissible	No***	Yes	No**			No

Pathogen	Occurs in NZ	Occurs in the UK	Recorded in budgerigars?	Strain variation NZ vs UK	Comments	Requires further consideration?
spongiform encephalopathy						
<b>Bacteria</b>						
<i>Chlamydomphila psittaci</i> (ornithosis)	Yes (Motha et al 1995)	Yes (OIE 2007)	Yes	No		No
<i>Salmonella Gallinarum</i>	No***	No (OIE 2007)	No**			No
<i>Salmonella Pullorum</i>	No***	Yes (OIE 2007)	No**			No
<i>Salmonella Arizonae</i>	No***	Yes (Hall and Rowe 1992)	No**			No
Exotic <i>Salmonella</i> spp. (numerous types and subtypes)	No	Yes	Yes	Yes numerous	Many exotic strains	Yes
<i>Salmonella</i> Enteritidis	Yes but not in poultry	Yes	Yes	Yes		Yes
<i>Escherichia coli</i> avian pathogenic <i>E coli</i> vero toxigenic <i>E Coli</i>	Yes ++	Yes	Yes	Yes	Universal	No
<i>Campylobacter</i> spp. (e.g. <i>C jejuni</i> )	Yes ++	Yes	Yes	No		No
Other enteric bacteria (Enterobacteriaceae)	Yes ++	Yes	Yes	Yes	Universal	No
<i>Pasteurella multocida</i>	Yes (Varney 2004)	Yes	Yes	No		No
<i>Riemerella anatipestifer</i>	Yes (Anonymous 1974)	Yes	No**			No
<i>Ornithobacterium rhinotracheale</i>	No***	Yes	No**			No
<i>Bordetella avium</i>	No***	Yes	No**			No
<i>Haemophilus paragallinarum</i>	No*	Yes	No (Blackall and Matsumoto 2003)			No
<i>Mycoplasma gallisepticum</i>	Yes****	Yes	Yes	No		No
<i>Mycoplasma iowae</i>	No***	Yes (Bradbury and Kleven 2003)	No (Bradbury and Kleven 2003),			No
Other <i>Mycoplasma</i> sp.	No*	No@	No**			No
<i>Mycobacterium tuberculosis</i>	Yes	Yes	No**		Antibiotic resistant strains	No
<i>Mycobacterium avium/intracellulare</i>	Yes	Yes	Yes	No		No
<i>Francisella tularensis</i>	No***	No (Tarnvik et al 2004)	No*			No
<i>Macrorhabdus ornithogaster</i>	Yes (Johnstone and Cork 1993)	Yes	Yes	No		No
Streptococci	Yes++	Yes	Yes	Yes	Universal	No

Pathogen	Occurs in NZ	Occurs in the UK	Recorded in budgerigars?	Strain variation NZ vs UK	Comments	Requires further consideration?
<b>Staphylococci</b>						
<i>Yersinia pseudotuberculosis</i>	Yes (Cork et al 1995; Gill 1996)	Yes				No
<i>Borrelia anserina</i> (avian spirochaetosis)	No***	No (Trees 1996)	Yes			No
<i>Borrelia burgdorferi</i> (Lyme disease)	No***	Yes (Kurtenbach et al 1998)	No**			No
Other spirochetes (intestinal)	Yes	Yes	Yes	No		No
<i>Coxiella burnetii</i>	No***	Yes (OIE 2007)	Not ** recorded		Notifiable	Yes
<i>Cowdria ruminatum</i>	No***	No (OIE 2007)	No**			No
<i>Aegyptianella pullorum</i>	No***	No	Yes		Vector absent in NZ	No
<b>Protozoal parasites</b>						
Blood parasites: <i>Haemoproteus</i> spp. <i>Leucocytozoon</i> spp. <i>Plasmodium</i> spp.	Some spp.	Some spp.	Some spp.	unknown	Numerous species	Yes
<i>Toxoplasma gondii</i> (Wilkins and O'Connell 1992)	Yes	Yes	Yes			No
<i>Encephalitozoon hellem</i>	Unknown	Unknown	Yes (Black et al 1997; Phalen 2005)	No		Yes
Other protozoal parasites	Multiple spp.	Multiple spp.	Some spp.			Yes
<b>Fungi and yeasts</b>						
Examples <i>Histoplasma</i> sp., <i>Cryptococcus</i> sp.	Some species	Yes	Yes	Numerous, some exotic		Yes
Internal parasites	Yes (Bishop and Heath 1998; McKenna 1998)	Yes	Yes	Variety of species		Yes
External parasites	Yes	Yes	Yes	Variety of species		Yes

- \* Extensive review of the New Zealand literature revealed no reports of the agent occurring in New Zealand.
- \*\* Extensive review of the literature revealed no reports of the agent occurring in budgerigars.
- \*\*\* Listed as exotic, unwanted or notifiable in the MAFBNZ register of unwanted organisms  
<http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>.
- \*\*\*\* Commonly reported by poultry industry in data provided to MAFBNZ and regularly published in the MAFBNZ magazine *Surveillance*.
- ++ Common isolates in New Zealand
- @ Review of the literature revealed no evidence of the occurrence of the organism occurring in UK.

## 4.2. HAZARD IDENTIFICATION

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

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

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

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

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

## 4.3. RISK ASSESSMENT

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

### Risk Assessment

- |                             |   |
|-----------------------------|---|
| a) Entry assessment -       | the likelihood of the organism being imported in the commodity.   |
| b) Exposure assessment -    | the likelihood of animals or humans in New Zealand being exposed to the potential hazard.   |
| c) Consequence assessment - | the consequences of entry, establishment or spread of the organism.   |
| d) Risk estimation -        | a conclusion on the risk posed by the organism based on the release, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is classified as a hazard. |

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

#### 4.4. RISK MANAGEMENT

For each organism classified as a hazard in the commodity the risk management section identifies the options available for managing the risk. Where the *Code* lists recommendations for the management of a hazard, these are described alongside options of similar, lesser, or greater stringency where appropriate. In addition to the options presented, unrestricted entry or prohibition may also be considered for all hazards. Final 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 is drafted.

#### 4.5. RISK COMMUNICATION

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

Following this period of public consultation on this draft document, a review of submissions will be produced and a MAF decision-making committee will determine whether any changes need to be made to this draft document in order for it to become the final risk analysis.

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

#### 4.6. SPECIAL CONSIDERATIONS

The incubation period and the time for which an animal may remain infectious are critical parameters for determining quarantine periods. An animal could have been infected with a disease on the day it goes into quarantine. After the incubation period for the disease, it could then be infectious for a period that differs for each disease. In many acute diseases the infectious period may correspond with the period for which the animal remains viraemic or bacteraemic. However, in cases of chronic diseases and diseases where animals remain chronic carriers of the organism, animals may be infectious for much longer periods. Animals should be quarantined for the maximum known incubation period plus the maximum period for which they remain infectious. Ideally the maximum period would be the mean period plus three standard deviations. This would cover 99.7% of cases. However, the true distribution of

incubation period and infectious period is usually not known because data are not available from a sufficiently large number of cases, or because of technical difficulties in obtaining accurate data. Data quoted may be unreliable because of the small numbers of animals used in experiments or because analysis was done at discrete intervals and therefore exact end-points were not determined. The measurements are also dependent on the accuracy and sensitivity of the method used to detect the infectious agent. For these reasons a conservative margin of error should be added to the best available estimates when determining quarantine periods. The margin of error added cannot be scientifically determined but relies on judgement, taking into account such things as amount and perceived accuracy of the available data, type of disease and the analytical methods used. In some infectious diseases recovered animals remain carriers of the infectious agent for long periods or even for life, and in these cases quarantine is not useful. In this risk analysis recommended quarantine periods are generally adjusted to whole weeks or months. When Import Health Standards are written by MAFBNZ based on this risk analysis these recommended periods may be modified.

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## 5. Avian Paramyxovirus-1

### 5.1. HAZARD IDENTIFICATION

#### 5.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Avulavirus*, *Newcastle disease virus* (APMV-1) (Lamb et al 2005). Strains vary from apathogenic to extreme virulence.

#### 5.1.2. OIE list

Listed.

#### 5.1.3. New Zealand status

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

#### 5.1.4. Epidemiology

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

The incubation period for the ND is given as 21 days in the *Code*, and recovered birds do not remain long term carriers of the virus.

Newcastle disease virus is probably capable of infecting all species of birds (Alexander and Gough 2003). Wild captive birds and pet birds frequently carry the virus. Importation of wild birds including psittacines, has been responsible for introducing the virus to the USA, or in other cases virus has been isolated from quarantined birds before they were released (Bunning-Fann et al 1992; Senne et al 1983).

APMV-1 of low virulence (apathogenic strains with ICPIs of 0-0.16) occur in New Zealand (Pharo et al 2000), chiefly in broiler breeders. Haemagglutination inhibition titres of between 1:16 and 1:1024 were detected in poultry, caged and wild birds (Stanislawek et al 2001).

The disease is rare in the UK, which is generally considered to be free from virulent Newcastle disease. The most recent outbreak of Newcastle disease in the UK occurred in farmed partridges in Scotland in 2006. It was caused by the pigeon variant of the virus (Anonymous 2006). The disease was promptly eradicated by slaughter of all infected birds. However, Newcastle disease strains of low virulence are universally distributed and the strains that occur in the UK could differ from those that occur in New Zealand. Virulent strains may be generated by mutation from avirulent strains. The events that occurred in

Australia where strains of low virulence mutated to virulence suggest that some low virulence strains are more likely to mutate to virulence than others (East et al 2006; Westbury 2001).

Diagnosis of infection can be made by virus isolation from tissues (Alexander 2004) or by PCR methods (Pham et al 2005; Wise et al 2004). Serological methods such as the haemagglutination inhibition (HI) test and ELISA are available for the detection of antibodies (Alexander 2004)

#### 5.1.5. Hazard identification conclusion

Since virulent Newcastle disease virus does not occur in the UK the likelihood of introducing the virus is negligible. However, the likelihood of introducing low virulence precursors of virulent strains is non-negligible. For practical purposes all APMV-1 strains are considered to be potential hazards in the commodity.

## 5.2. RISK ASSESSMENT

### 5.2.1. Entry assessment

Low virulence strains of APMV-1 occur in the UK but their prevalence in budgerigars is unknown. While it is unlikely that budgerigars would be carriers of these viruses, it is considered possible that the virus could circulate amongst a group of budgerigars. Therefore the likelihood of entry is considered to be non-negligible.

### 5.2.2. Exposure assessment

Imported budgerigars are likely to be mixed with New Zealand birds in aviaries and at shows and the disease could be transmitted to budgerigars, other birds and ultimately to poultry flocks. In addition, although imported exhibition budgerigars are unlikely to be allowed to readily escape from captivity, and are unlikely to survive for any significant length of time if they do, escape always remains a possibility. During the time escaped birds survive in the “wild” they could transmit virus to wild and feral birds. Therefore the likelihood of exposure of New Zealand birds to the virus is considered to be non-negligible.

### 5.2.3. Consequence assessment

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

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

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

#### 5.2.4. Risk estimation

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

### 5.3. RISK MANAGEMENT

#### 5.3.1. Options

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

- Freedom from disease signs would not be a useful measure since infections are likely to be sub-clinical.
- Serological testing of the flock of origin could be used to demonstrate flock-freedom from infection. However, positive serological tests may indicate previous infection and does not imply that the birds are presently infected.
- Quarantine alone would not be useful but quarantine and testing birds after they have been in quarantine for 3 weeks (one incubation period) could be used to demonstrate freedom of individual birds from infection.
- Since some operators find bleeding of budgerigars difficult, birds could be tested by virus isolation or PCR instead of serological tests (Alexander 2004; Pham et al 2005; Wise et al 2004) rather than serological methods. While serological tests require blood samples, PCR and virus isolation are carried out on cloacal swabs
- Vaccination is undesirable because vaccinated birds could harbour low virulence vaccine strains of the virus which do not occur in New Zealand.

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

The relevant *Code* recommendations are:

#### Article 10.13.5.

##### **Recommendations for the importation of live birds other than poultry**

Regardless of the ND status of the country, [zone](#) or [compartment](#) of origin, [Veterinary Authorities](#) should require the presentation of an [international veterinary certificate](#) attesting that:

1. the birds showed no clinical sign suggestive of ND on the day of shipment;
2. the birds were kept in isolation approved by the [Veterinary Services](#) since they were hatched or for at least the 21 days prior to shipment and showed no clinical sign of [infection](#) during the isolation period;
3. the birds were subjected to a diagnostic test within 14 days prior to shipment to demonstrate freedom from [infection](#) with NDV;
4. the birds are transported in new or appropriately sanitized [containers](#).

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

The following options, given in order of ascending stringency (and increasing cost), could be considered to effectively manage the risk:

### **Option 1.**

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 APMV-1, on samples taken at least 14 after entry into quarantine, with negative results; and:
- iii. have not been vaccinated against Newcastle disease;

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

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

### **Option 2.**

Birds to be imported could:

- 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 APMV-1, on samples taken at least 21 days after entry into quarantine, with negative results; and:
- iii. have not been vaccinated against Newcastle disease.

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

### **Option 3.**

A further option is to use sentinel birds in pre-export quarantine. The number of these would have to be determined according to the size of the shipment, but they would have to be 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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## 6. Avian Paramyxovirus-2

### 6.1. HAZARD IDENTIFICATION

#### 6.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Avulavirus*, *Avian paramyxovirus 2* (APMV-2) (Lamb et al 2005).

#### 6.1.2. OIE list

Not listed.

#### 6.1.3. New Zealand status

No isolations of APMV-2 have been made in New Zealand, although serological surveys have detected titres of 1:8 to 1:32 in a variety of cage and wild birds (Stanislawek et al 2001).

#### 6.1.4. Epidemiology

APMV-2 viruses were first isolated from chickens and turkeys in America and Europe, but are most commonly isolated from passerine birds (Alexander and Gough 2003), in which most infections are subclinical (Ritchie 1995). APMV-2 isolations are rarely reported from psittacine birds (Kaleta and Baldauf 1988). African grey parrots are regarded as most susceptible to the development of clinical disease, and although infection has been reported in budgerigars, no clinical signs have been reported (Kaleta and Baldauf 1988). The large numbers of isolations of APMV-2 from passerine birds compared to psittacine birds in UK quarantine facilities is considered to point to psittacine infections being related to close contact in transit or quarantine with passerine birds (Alexander 1996). Similar conclusions were drawn from a study of birds imported into the USA (Senne et al 1983).

#### 6.1.5. Hazard identification conclusion

Since APMV-2 causes occasional outbreaks of disease in poultry in the UK, and infections of budgerigars are likely to be subclinical, APMV-2 is a potential hazard in the commodity.

### 6.2. RISK ASSESSMENT

#### 6.2.1. Entry assessment

Since budgerigars are unlikely to become infected with APMV-2 unless held in close proximity with passerine birds or infected turkeys, the entry assessment for budgerigars sourced from a single closed budgerigar donor flock is considered to be negligible.

#### 6.2.2. Risk estimation

The likelihood of entry is assessed to be negligible. As a result the risk estimate for APVM-2 is negligible and it is not classified as a hazard in the commodity.

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## 7. Avian Paramyxovirus- 3

### 7.1. HAZARD IDENTIFICATION

#### 7.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Avulavirus*, *Avian paramyxovirus 3*(APMV-3) (Lamb et al 2005).

#### 7.1.2. OIE list

Not listed.

#### 7.1.3. New Zealand status

Isolation of the virus has not been reported. The virus is not listed as an unwanted or notifiable organism. Very low titres of 1:4 and 1:8 were recorded in a small number of birds during a survey (Stanislawek et al 2001).

#### 7.1.4. Epidemiology

APVM-3 has been detected in captive birds in quarantine (Alexander 1996; Shihmanter et al 1998). It may cause a mild respiratory disease in some birds, especially psittacine birds and turkeys, and field outbreaks amongst turkeys have been documented in the UK, Europe and North America (Alexander 1996). Neurological signs have been described in parakeets (Shihmanter et al 1998).

Using structural polypeptide analysis, and monoclonal antibody serotyping two sub-groups of APMV-3 are recognised: Group 1 isolates have come mainly from turkeys and Group 2 from psittacines (Anderson et al 1987).

APMV-3 antibodies show a degree of cross reactivity with APMV-1(Alexander and Gough 2003). For this reason there is uncertainty about whether the low prevalence of low antibody titres found in New Zealand birds indicates endemic infection.

World-wide there have been no reports of isolation of APMV-3 from wild birds, only from caged birds and predominantly from birds in quarantine (Alexander and Gough 2003) where infections are likely to be associated with stress.

#### 7.1.5. Hazard identification conclusion

Since there is uncertainty about the occurrence of the virus in New Zealand and it has been documented as occurring in the UK it is considered to be a potential hazard in the commodity.

### 7.2. RISK ASSESSMENT

#### 7.2.1. Entry assessment

The virus has been reported in turkeys in the UK. However the turkey and psittacine isolates appear to be antigenically different (Anderson et al 1987). Isolations of the virus from cage

birds have been almost exclusively confined to isolations from birds in quarantine. Therefore, the likelihood of entry of APMV-3 while importing birds from closed budgerigar flocks in the UK is extremely low but non-negligible.

### 7.2.2. Exposure assessment

New Zealand captive birds are likely to be exposed to imported birds in aviaries or at shows. Escaped exhibition budgerigars will not survive for long outside temperature controlled indoor aviaries. Therefore wild birds could be exposed to escaped budgerigars only during the time they survive in the wild and the likelihood of exposure of New Zealand wild birds is low.

### 7.2.3. Consequence assessment

APMV-3 is generally considered to be of minor importance. 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 considered 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 are no reports of APMV-3 infection of humans.

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

### 7.2.4. Risk estimation

The consequences of importation are considered to be negligible. As a result the risk estimate for APVM-3 is negligible and it is not classified as a hazard in the commodity. Therefore risk management measures are not justified.

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## 8. Avian Paramyxovirus- 5

### 8.1. HAZARD IDENTIFICATION

#### 8.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Avulavirus*, *Avian paramyxovirus 5* (APMV-5) (Lamb et al 2005).

#### 8.1.2. OIE list

Not listed.

#### 8.1.3. New Zealand status

Neither isolation of the virus nor presence of antibody to APMV-5 has been reported.

#### 8.1.4. Epidemiology

APVM-5 causes a rare disease of budgerigars that is characterised by very high mortality. The virus caused severe disease in bugerigars in Japan from 1974-6 (Alexander 1993; Nerome et al 1978). One outbreak of the disease has been recorded in the UK in 1993 (Gough et al 1993). Mustaffa-Babjee and Spreadborrow (1973, 1974) reported infection of budgerigars and wild rainbow lorikeets with a virus that may have been APMV-5 (Ritchie 1995).

The disease described in Japan was characterised by a high mortality rate of 95-100% (Alexander 1993; Nerome et al 1978).

APMV-5 causing death in budgerigars has been recorded only from one aviary in 1993 in the UK (Gough et al 1993).

#### 8.1.5. Hazard identification conclusion

Since APMV-5 has been recorded only once in budgerigars in the UK in 1993, it is considered that the virus is not endemic in the UK. It is also extremely rare in other countries. It is therefore considered that APMV-5 is unlikely to be associated with the commodity and the organism is not classified as a potential hazard.

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## 9. Avian Influenza Virus

### 9.1. HAZARD IDENTIFICATION

#### 9.1.1. Aetiological agent

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

#### 9.1.2. OIE list

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

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

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

#### 9.1.3. New Zealand status

Influenza A viruses (exotic avian strains) are listed as unwanted, notifiable organisms (MAF 2007). No influenza viruses have been recovered from budgerigars in New Zealand and there have been no reported outbreaks of HPNAI in any birds. Viruses of types H1N3, H4N6, H6N4, H11N3 and H5N2 have been isolated from healthy wild mallard ducks (Austin and Hinshaw 1984; Stanislawek 1990; Stanislawek 1992; Stanislawek et al 2002). In addition the following incompletely classified virus types have been isolated: H2N?, H7N?, H10N? (Stanislawek 2008) The H5N2 virus was of low pathogenicity (Stanislawek et al 2002). In 2008 a H5N1 virus was isolated from mallards. However, this isolate is a low pathogenicity strain, unlike the high pathogenic strain responsible for the world-wide pandemic of avian influenza (MAF 2008). A recent serological survey of poultry in New Zealand failed to show evidence of notifiable AI in broilers and layers (Tana et al 2007).

#### 9.1.4. Epidemiology

The family *Orthomyxoviridae* contains four genera: *Influenzavirus A*, *Influenzavirus B*, *Influenzavirus C*, and *Thogovirus*. AI is caused only by influenza A viruses. Type A viruses are divided into subtypes according to the antigenic nature of their surface glycoprotein haemagglutinins (H) and neuraminidases (N). There are currently 16 H types and 9 N types recognised and all combinations of H and N antigens are possible (Swayne and Halvorson 2003). All known HPAI isolates have either the H5 or H7 haemagglutinin but H5 and H7 isolates of low virulence are also known (Swayne and Halvorson 2003). Strain virulence can be determined based on the amino acid sequences at the proteolytic cleavage site on the H protein (Alexander 2004). However, identification of pathogenicity is still primarily determined by measuring an intravenous pathogenicity

index in chickens and growth in cell culture, causing a cytopathic effect in the absence of trypsin (Alexander 2004; Swayne and Suarez 2000). According to the most recent OIE definition notifiable avian influenza includes both HPAI strains and LPAI strains of the H5 and H7 subtypes (see Section 11.1.2). For the purposes of this risk analysis all Influenza A strains found in birds presented for export to New Zealand will be considered to be of concern.

The virus has a world-wide distribution, with a broad host range, with the overwhelming majority of isolates having come from waterfowl which are the natural hosts of the virus (Swayne and Halvorson 2003; Swayne and Suarez 2000). The virus can be transmitted by the oral or oronasal routes particularly in birds that are in close contact with each other. Long term carriers do not exist but the virus may be excreted for up to 30 days in ducks, 36 days in chickens and 72 days in turkeys (Swayne and Halvorson 2003). Circulation of the virus in a group of birds may result in long-term infection of the group. 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 2003). The *Code* gives the incubation period for the purposes of international trade as being 21 days.

Isolations of AI viruses from psittacine birds are uncommon. Most psittacine isolates have originated from captive birds that had died suddenly (Alexander 2000). Where disease signs were noticed prior to death these were described as loss of condition, ruffled feathers, green diarrhoea and nervous signs (Alexander 1993).

Gerlach reported that 7 out of 12 experimentally infected budgerigars birds showed ruffled feathers and diarrhoea and two birds died (Ritchie 1995). An H4N6 isolate was recovered from a budgerigar in Japan in 1977 (Imada et al 1980). The H5N1 virus currently affecting poultry in Asia was found to be pathogenic for budgerigars (Isoda et al 2006; Perkins and Swayne 2003).

There can be no certainty about what types of LPAI are present in any country, because strains present may change as migratory birds come and go, and mutations and reassortments of gene segments occur. Birds infected with LPAI strains may show no clinical signs. In the UK an outbreak of LPAI (H7N3) was recorded in Norfolk in 2006 (DEFRA 2006).

Since the emergence of the HPAI H5N1 virus a pandemic of avian influenza has spread through Asia, the Middle East, eastern and central Europe (Sabirovic et al 2006), and the first case occurred in England (Suffolk) in February 2007 (DEFRA 2007b). At the time of writing (November 2007) an outbreak of H5N1 has occurred in turkeys in the Norfolk/Suffolk area (DEFRA 2007a). The British authorities are slaughtering the infected flock and enforcing a 3km Protection Zone, a 10km Surveillance Zone and a wider Restricted Zone covering the whole of Suffolk and most of Norfolk around the infected premises. For the purposes of this risk analysis it will be assumed that the disease is eradicated. However, the risk analysis will be modified as necessary depending on the outcome of the control measures.

Several options are available for diagnosis. Since the nucleocapsid antigen of all Influenza A viruses is similar, the agar gel immunodiffusion test can be used as a group test for all Influenza A subtypes (Alexander 2004). For virus detection, an antigen detection ELISA based on a monoclonal antibody to the group specific nucleoprotein that can detect all Influenza A viruses is available as a commercial kit (Perkins and Swayne 2003). A matrix real time RT/PCR TaqMan is available at the Investigation and Diagnostic Centre for the detection of influenza A viruses and is preferred to antigen detection ELISA because of

greater sensitivity Stanislawek (2008).

#### 9.1.5. Hazard identification conclusion

Since HPAI has recently occurred in the UK, it is reasonable to consider that it has the potential to be associated with all bird species. LPAI strains occurring in UK are likely to differ from New Zealand strains and therefore these may be considered to potentially be associated with the commodity. Therefore HPAI and LPAI strains are considered to be potential hazards in this risk analysis.

## 9.2. RISK ASSESSMENT

### 9.2.1. Entry assessment

HPAI and LPAI viruses occur in the UK and the number and type of strains occurring there are likely to be constantly changing. Budgerigars in a closed flock are unlikely to be exposed to AI infection, and may be relatively resistant to the development of clinical signs. Therefore the likelihood of introducing the viruses is low but non-negligible.

### 9.2.2. Exposure assessment

Imported budgerigars will be introduced into collections where they will be in close contact with other birds. Imported birds may be further disseminated by sale and movement of birds to shows. Since budgerigars are not known to be carriers of AI viruses and since escaped budgerigars are unlikely to establish in the wild, the likelihood of transmission by escaped birds to wild birds is low. There is a low likelihood of transmission to raptors that kill and eat escaped budgerigars. Therefore it is considered that there is a low likelihood of exposure to New Zealand birds for viruses introduced into this country, and as such the likelihood of exposure is considered to be non-negligible.

### 9.2.3. Consequence assessment

Since influenza viruses are constantly mutating and recombining, the introduction of new viruses will increase the pool of viral genetic material available. This may increase the likelihood of emergence of a virulent strain of the virus through the importation of a low pathogenic H5 or H7 virus, or through the recombination of an imported neuraminidase moiety with a New Zealand H5 or H7. Emergence of a virulent strain of virus would have serious consequences for the poultry industry due to massive mortalities, destruction of flocks to prevent the spread of the disease, and through disruption of trade.

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

Infections of humans with AI strains occurred sporadically before 2003. As a consequence of the present pandemic in birds caused by the HPAI H5N1 strain, the World Health Organisation has reported that the cumulative number of cases in humans between 2003 and 12 November 2007 was 335 with 206 deaths (World-Health-Organisation 2007).

It is concluded that the likelihood of serious consequences caused by introducing avian influenza virus are non-negligible for poultry, native birds and human health.

#### 9.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for LPAI and HPAI viruses is non-negligible and they are classified as hazards in the commodity. Therefore risk management measures can be justified.

### 9.3. RISK MANAGEMENT

#### 9.3.1. Options

When considering options for effectively managing the risks the following points should be considered:

- Freedom from disease signs would not be useful since infections may be sub-clinical.
- Quarantine alone would not be useful in preventing export of sub-clinically infected birds, but quarantine and testing birds after they have been in quarantine for 3 weeks could be used to demonstrate that a group of birds to be imported is not infected with NAI.
- Serological testing could be useful but since some operators find bleeding budgerigars difficult virus detection methods may be preferred.
- Virus could be detected in swabs from birds in quarantine using an antigen detection ELISA or a PCR test that is group specific for influenza A viruses.
- The incubation period stated in the *Code* for international trade is 21 days.

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

#### **Recommendations for the importation of live birds other than poultry**

Regardless of the NAI status of the country, [zone](#) or [compartment](#) of origin, [Veterinary Authorities](#) should require the presentation of an [international veterinary certificate](#) attesting that:

1. the birds showed no clinical sign of [infection](#) with a virus which would be considered NAI in poultry on the day of shipment;
2. the birds were kept in isolation approved by the [Veterinary Services](#) since they were hatched or for at least the 21 days prior to shipment and showed no clinical sign of [infection](#) with a virus which would be considered NAI in poultry during the isolation period;
3. the birds were subjected to a diagnostic test within 14 days prior to shipment to demonstrate freedom from [infection](#) with a virus which would be considered NAI in poultry;
4. the birds are transported in new or appropriately sanitized [containers](#).

If the birds have been vaccinated, the nature of the vaccine used and the date of vaccination should be attached to the [certificate](#).

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

#### **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. Herpesvirus Infections of Budgerigars

### 10.1. HAZARD IDENTIFICATION

#### 10.1.1. Aetiological agent

Viruses of the family *Herpesviridae* are double stranded DNA viruses. Those isolated from birds are from the subfamily *Alphaherpesvirinae*. The international committee for virus taxonomy recognises *Psittacid herpesvirus 1* (PsHV-1) as the sole species of psittacine alphaviruses (Davison et al 2005), but this species has not yet been assigned to a particular genus. Another herpesvirus has been described in African Grey parrots and it has been proposed that it be recognised as psittacid herpesvirus 2 (Styles et al 2005). However, a large number of serotypes have been reported. According to one classification scheme there are 19 serotypes of avian herpesviruses (Kaletta 1998). An alternative scheme that recognizes 12 serotypes of avian herpesviruses plus some unclassified viruses (Ritchie 1995b) will be followed in this risk analysis. Avian serotypes 4, 5 and 6, are most commonly recovered from psittacine birds, including budgerigars. Cross neutralisation tests show that the psittacine herpesviruses fall into three main serogroups that are distinct from one another, but unrelated to the herpesviruses of other birds such as Marek's disease virus, infectious laryngotracheitis virus and duck viral enteritis virus (Ritchie 1995b). In addition to serotyping four genotypes of psittacid herpesviruses have been described (Tomaszewski et al 2003).

Pacheco's disease virus is the most notable member of serotype 4, and psittacid herpesviruses that make up serotypes 5 and 6 cause Pacheco's-like disease (Ritchie 1995b). Budgerigar herpes virus is grouped in serotype 7 and is related to pigeon herpesvirus (Ritchie 1995b).

The viruses associated with internal papillomatosis (mucosal papillomatois) are discussed in the section on papillomaviruses.

#### 10.1.2. OIE list

Not listed.

#### 10.1.3. New Zealand status

A herpes virus resembling Pacheco's virus was recovered from psittacine birds in the mid 1970's (Durham et al 1977). However, following a protracted court case in 1997 it was determined that the 1977 report did not constitute proof that Pacheco's disease had occurred in New Zealand. In 1997 Pacheco's disease was diagnosed in imported birds in New Zealand quarantine (Thornton and Stanislawek 2003). Subsequently some of the birds in the batch were smuggled out of quarantine and introduced into New Zealand private collections (Thornton and Stanislawek 2003). An extensive investigation did not reveal any evidence that the disease was introduced due to this incident (Loth 2003; Thornton and Stanislawek 2003). As a result, New Zealand is currently considered to be free from Pacheco's disease virus (Loth 2003).

#### 10.1.4. Epidemiology (with particular reference to budgerigars)

##### 10.1.4.1. Pacheco's disease

According to Ritchie (Ritchie 1995b), Pacheco's disease is caused by a serotype 4

herpesvirus while serotype 5 and 6 herpesviruses cause a Pacheco's-like disease.

Pacheco's disease has often been associated with stress and occurs in susceptible populations crowded together in aviaries and quarantine facilities where it is characterised by sudden death and liver pathology (Bistyak et al 2004; Durham et al 1977; Gaskin et al 1978; Gough and Alexander 1993; Ritchie 1995b; Thornton and Stanislawek 2003).

Pacheco's disease virus (PsHV-1) is recognised as causing severe mortality in confined groups of psittacine birds, but psittacid herpesviruses of all 3 serotypes have been isolated from subclinical carrier birds as well as from cases showing severe disease (Kaletta 1998; Ritchie 1995b). In budgerigars clinical disease is usually of lower severity than in larger parrots (Kaletta 1998; Ritchie 1995b). Budgerigars have been shown to develop disease following experimental exposure by natural (Ramis et al 1996) and experimental (intramuscular injection) routes (Cho and McDonald 1980). Budgerigars have been listed amongst the species resistant to infection with Pacheco's virus, in comparison with highly susceptible species such as Amazon parrots and cockatoos (Snowdon 1995). Only 3 cases of Pacheco's disease were found in a survey of the causes of mortality in 1525 dead and euthanased exhibition budgerigars examined in the UK from 1984 to 1995 (Baker 1996). However, this survey was based on gross pathology and histopathology and did not use any form of virological testing. Specific surveillance for Pacheco's disease in budgies in the UK has not been done.

Limited experimental evidence suggests that the incubation period may be as short as a few days in budgerigars (Ramis et al 1996). Recovered infected birds remain latently infected, subclinical long term carriers of virus and they shed the virus intermittently over many years at times of stress (Ritchie 1995b; Tomaszewski et al 2006).

PCR testing has been more sensitive than tissue culture for the detection of herpes virus in parrots (Tomaszewski et al 2001). High antibody titres may be found in recently infected birds but the antibody declines to low or negative levels in latent carriers. Virus excretion in latent carriers is intermittent and virus isolation techniques cannot be relied upon for diagnosis (Ritchie 1995b).

#### 10.1.4.2. Amazon tracheitis

Amazon tracheitis is characterised by necrotic tracheitis with typical herpesvirus intra-nuclear inclusion bodies in cells of the tracheal epithelium (Ritchie 1995a). The herpesvirus involved is closely related to infectious laryngotracheitis virus of chickens (Ritchie 1995b). The disease has been described on rare occasions from Amazon parrots in Germany and the USA, and is considered to be of negligible significance in birds other than Amazon parrots (Snowdon 1995). No reports could be found of its occurrence in budgerigars, or in the UK. Current opinion on this disease is that it does not constitute a separate group of psittacine herpesvirus, and need not be considered separately (Ritchie 1995a).

#### 10.1.4.3. Budgerigar herpesvirus (Psittacid herpesvirus serotype 7)

Serotype 7 herpesviruses include pigeon, falcon and owl herpesviruses and a budgerigar herpesvirus that is serologically related to pigeon herpesvirus (Ritchie 1995b). The fact that budgerigars can be infected with true pigeon herpesvirus add weight to the idea that budgerigar herpesvirus and pigeon herpesvirus can be considered to be the same virus (Ritchie 1995b).

Herpesviruses have been found in pigeons in New Zealand (Thompson et al 1977) and are common in Australian pigeons. Large numbers of pigeons have been imported from Australia over the past 30 years. Similar viruses are isolated from psittacine birds, especially budgerigars (Vindevoegel and Pastoret 1993). There is no reason to believe that budgerigar herpesvirus serogroup 7 and columbid herpesvirus are different viruses. Therefore the virus is considered to be already present in New Zealand.

#### 10.1.5. Hazard identification conclusion

Pacheco's virus and related viruses that cause Pacheco's-like disease are potential hazards in the importation of budgerigars from the UK. Amazon tracheitis is not a disease of budgerigars and columbid herpesviruses are endemic, so these are not considered to be potential hazards in the commodity.

## 10.2. RISK ASSESSMENT

### 10.2.1. Entry assessment

Since Pacheco's virus has been occasionally recorded in budgerigars in the UK, the entry assessment is low but non-negligible.

### 10.2.2. Exposure assessment

The risk of exposure of native birds to Pacheco's virus through imported budgerigars is low, given the isolated nature of most native psittacine populations, the close contact required for transmission of psittacine herpesviruses, and the inability of budgerigars, especially exhibition budgerigars to survive outside captivity. The exposure risk for non-native psittacine birds is mainly limited to the movement of imported birds from the importing aviary to shows, and for sale. Whilst this risk is low, it is non-negligible.

### 10.2.3. Consequence assessment

Snowdon (1995) considered that the effect of the introduction of Pacheco's disease virus on native Australian psittacine birds was largely a matter of speculation, and the position is probably similar in New Zealand. The only native New Zealand psittacines held in moderate numbers as pets and in mixed collections overseas are kakariki. Snowdon (1995) listed a single reported outbreak of Pacheco's disease in a red-fronted kakariki (*Cyanoramphus novaezelandiae*) in the USA (Gaskin et al 1978). There has been no evidence of transmission or establishment of the 1977 or 1997 isolates of virus in native or caged birds. The Department of Conservation exercises control over private holdings of native psittacine birds, and it is unlikely that native psittacines are held in high density conditions in this country where Pacheco's disease virus could initiate clinical disease. Since the disease is of significance only in situations where birds are crowded and stressed in unnatural environments, the likelihood for the virus to establish in populations of indigenous birds in the wild is considered to be extremely low.

Nevertheless, the introduction of the virus into established collections of non-native psittacines could result in transmission and mortalities amongst these birds. Therefore the consequences are assessed as non-negligible. Avian herpes viruses are not zoonotic agents and there is no threat to human health.

#### 10.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for Pacheco's disease virus is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified

### 10.3. RISK MANAGEMENT

#### 10.3.1. Options

The following key facts are relevant to the drafting of options to prevent the introduction of herpesviruses in the commodity:

- Clinical examination of birds is unlikely to be helpful since latent carriers occur and acutely infected birds often die suddenly without showing previous clinical signs.
- Quarantining of birds cannot be guaranteed to prevent entry of the virus since life-long latent carriers of the virus are known to occur. However, quarantining of birds is likely to precipitate outbreaks of disease if latent carriers of infection 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.
- There is no reliable way to detect latent Pacheco's virus infections in live birds (Phalen 2000).
- Virus isolation or identification procedures such as PCR are of limited value since latent carriers only excrete the virus sporadically. However, more recent evidence suggests that PCR on cloacal and oral swabs are a promising method for detecting carriers of the virus, and that the primer sets used detected all strains of psittacine herpesviruses (Tomaszewski et al 2001). A group of birds could be tested after they have been in quarantine for a period of 3 weeks. In these circumstances shedding by latent carriers could infect naïve birds in the group resulting in acute infections that could be readily detected by PCR.
- Serological testing is insensitive for individual birds but could be used as a flock test. However, there are considerable problems in obtaining sufficient serum for serological testing of budgerigars.
- Although no measures are available that guarantee that latent carriers will be identified the methods used in combination could provide a high level of assurance that the disease will not be introduced.
- Importation from flocks that are regularly inspected and have never had confirmed cases of Pacheco's disease are most likely to be safe sources from which to import birds.

There is no *Code* chapter on herpesviruses of budgerigars.

The available options in ascending order of stringency are:

#### **Option 1.**

Birds to be imported could be certified as coming from flocks in which the disease has not previously been reported.

#### **Option 2.**

Birds to be imported could:

- i. be sourced from closed flocks in which there is no previous history of herpesvirus infections; and
- ii. be quarantined for at least 28 days.

After 3 weeks in quarantine, cloacal swabs from all birds for export could be tested by a PCR test. Positive tests on samples from any birds while in quarantine could result in disqualification of the whole group.

Budgerigars that died while in quarantine could be submitted to a post mortem examination and suitable samples submitted to an approved laboratory for examination.

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# 11. Psittacine Pox Virus

## 11.1. HAZARD IDENTIFICATION

### 11.1.1. Aetiological agent

Family: *Poxviridae*. Genus: *Avipox*, *Psittacine pox virus* (Buller et al 2005)

*Psittacinepox virus* is antigenically distinct from *Fowlpox*, *Pigeonpox*, and *Quailpox* viruses (Ritchie 1995). Psittacine pox viruses form a clade distinct from other avipox viruses (Jarmin et al 2006; Luschow et al 2004).

### 11.1.2. OIE list

Not listed.

### 11.1.3. New Zealand status

Not listed as an unwanted or notifiable organism. One outbreak of the disease has occurred and all birds on the premises were destroyed (King et al 2003). The source of the infection could not be traced. Therefore, for this risk analysis it is regarded as an exotic organism.

### 11.1.4. Epidemiology

The virus is endemic in Europe and America (Ritchie 1995). Cutaneous, diphtheritic or systemic forms of the disease occur. The cutaneous form which presents as typical nodules on unfeathered skin can resolve in about a month or persist for up to a year, and mortality rates are low. The diphtheritic form, characterised by fibrinonecrotic lesions on mucous membranes is usually fatal. High mortality rates have occurred in parrots held in quarantine (Ritchie 1995). The incubation period is from 4- 30 days but following natural infection it is generally 7-14 days (Ritchie 1995). Birds may be latently infected with pox viruses and persistent infection for up to 13 months has been reported in chickens that shed the virus in feathers, skin or faeces for up to 13 months (Tripathy, 1975 quoted by (Ritchie 1995)). The histopathological lesions are diagnostic for the disease when typical inclusion bodies are demonstrated. The virus can be cultured in embryonated eggs or demonstrated by electron microscopy in papules, vesicles or scabs (Ritchie 1995). Transmission can occur by contact with virus through abraded skin but it is generally transmitted by mosquitoes or lice (Ritchie 1995). Antibody develops 1-2 weeks after infection and can be detected by agar gel diffusion, virus neutralisation ELISA or haemagglutination (Ritchie 1995).

### 11.1.5. Hazard identification conclusion

*Psittacine pox virus* is exotic and occurs in Europe. Since it can lead to mortalities in psittacine birds, it is considered to be a potential hazard.

## 11.2. RISK ASSESSMENT

### 11.2.1. Entry assessment

Psittacine pox occurs in Europe, the virus may be found in clinically infected birds or persistent carriers of infection. Therefore, the likelihood that infected budgerigars could be introduced from the UK is non-negligible.

### 11.2.2. Exposure assessment

Since imported budgerigars would be introduced into New Zealand aviaries and traded and taken to shows the virus could be transmitted to New Zealand caged birds. Other avipox viruses occur in New Zealand demonstrating that competent vectors are present and therefore the virus could be transmitted to caged or wild birds. There is also a remote risk that escapes of budgerigars could transmit the virus to indigenous psittacine birds. Therefore the likelihood of exposure of caged and wild birds is non-negligible.

### 11.2.3. Consequence assessment

Contact between introduced birds and caged birds could result in clinical disease and deaths in psittacine birds. Due to mixing of birds at shows the virus could become endemic. The consequences for native psittacines is unknown but are likely to be similar to those for other psittacines. The virus is not zoonotic. Therefore the consequences are considered to be non-negligible.

### 11.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for psittacine pox virus is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified.

## 11.3. RISK MANAGEMENT

### 11.3.1. Options

The following points were considered when drafting options to manage the risks associated with the introduction of psittacine pox virus in the commodity:

- Psittacine pox virus may be transmitted by clinically infected birds or persistent carriers of the virus.
- The virus can be demonstrated by virus isolation, electron microscopy or histopathology from suitable lesion material.
- Antibody to the virus can be demonstrated by several serological tests. However, it should be noted that it is noted that bleeding of budgerigars for serological testing may be difficult and hazardous to the budgerigars

There is no Code chapter relating to the disease.

Options for the management of the introduction of the virus in the commodity, in ascending order of stringency, include:

### Option 1.

Budgerigars for importation could be healthy and not show any signs suggestive of psittacine pox infection before or during quarantine.

### Option 2.

Budgies to be exported could:

- i. be isolated in quarantine for 3 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

### Option 3.

Budgies to be exported could:

- i. be isolated in quarantine for 3 weeks; and
- ii. be free from clinical signs of disease before and during quarantine; and
- iii. be tested serologically after at least 2 weeks in quarantine, with negative results.

Any suspicious lesions detected during the quarantine period could be examined by virus isolation, histology and electron microscopy, with negative results

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## 12. Psittacine Reovirus Infection

### 12.1. HAZARD IDENTIFICATION

#### 12.1.1. Aetiological agent

Family: *Reoviridae*, Genus: *Orthoreovirus*.

The virus has not been formally classified by the International Committee on Taxonomy of Viruses. The virus reacts with reovirus polyclonal antiserum but not with monoclonal antibodies for specific chicken reovirus isolates. Therefore, it is considered to be a psittacine reovirus (Van den Brand et al 2007).

#### 12.1.2. OIE list

Not listed.

#### 12.1.3. New Zealand status

Unknown in New Zealand

#### 12.1.4. Epidemiology

A disease syndrome characterised by high mortality and liver and spleen necrosis has been described in budgerigars and other psittacine birds (Manvell et al 2004; Pennycott 2004; Van den Brand et al 2007). The disease has occurred in the UK and in the Netherlands and may be present in other countries (Van den Brand et al 2007). Early descriptions of a reovirus infections in psittacines (Ashton et al 1984; Senne et al 1983) were followed by sporadic descriptions of disease associated with reovirus infections that have been summarised by Van den Brandt et al (2007). Disease was initially thought to be associated with parrots, with budgerigars being more resistant to infection. However, recently disease and high mortalities have been described in budgerigars (Pennycott 2004).

Reported disease outbreaks were often associated with introductions of new birds into a flock (Ashton et al 1984; Pennycott 2004; Van den Brand et al 2007). Mortalities commenced within 2-5 days of the introduction of new birds in some cases and after 3- 4 weeks in others, 70-100% of birds died (Van den Brand et al 2007). From this confusing evidence it appears that the incubation period could vary from 2 days up to about 4 weeks. Since the evidence is not specific, it is assumed in this risk analysis that it could be as long as 4 weeks. In the cases described in budgerigars in Scotland, mortality was about 50% in adult birds and almost all young birds died. After initial mortalities ceased and breeding recommenced there were further mortalities in young birds (Pennycott 2004). This seems to indicate that recovered adult birds carried the infection for at least a few months, and it has been suggested that recovered birds may carry the virus (Van den Brand et al 2007). The route of infection has not been clearly defined but it is likely to be transmitted by direct or indirect contact with infected faeces, as with reovirus infection in poultry (Van den Brand et al 2007).

Reoviruses have been demonstrated by immunochemistry or electron microscopy and virus isolation. The virus isolates from the UK and the Netherlands are similar. Diagnostic tests that would be useful in live birds have not been described.

The epidemiology of the infection is still poorly defined.

#### 12.1.5. Hazard identification conclusion

Since the disease causes significant mortality and it has not been described in New Zealand, it is considered to be a potential hazard in the commodity.

## 12.2. RISK ASSESSMENT

### 12.2.1. Entry assessment

The disease is associated with budgerigars and other psittacines and occurs in the UK. Because outbreaks of disease often follow introduction of new birds it must be assumed that subclinically infected birds may carry the infection. It is not known whether these birds are long-term or short term carriers of infection. Therefore, the likelihood of introducing the virus when importing budgerigars is considered to be non-negligible.

### 12.2.2. Exposure assessment

Since outbreaks of disease have followed the introduction of birds into flocks, the virus could be transmitted by carrier birds to New Zealand flocks into which imported birds are introduced. Therefore, the likelihood of exposure is considered to be non-negligible.

### 12.2.3. Consequence assessment

Since the disease is known to cause high mortality when introduced into naïve flocks the consequences for budgerigars and budgerigar fanciers and breeders could be significant. Since the virus typically causes disease in psittacines, it should be assumed that native psittacine birds and free living introduced psittacines such as rosellas, lorikeets and cockatoos would be susceptible. Therefore, the consequences of introducing the virus are considered to be non-negligible.

There is no indication that the virus is zoonotic or that it will infect mammals.

### 12.2.4. Risk estimation

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

## 12.3. RISK MANAGEMENT

When drafting options for the management of psittacine reovirus in the commodity the following points should be considered:

- Reovirus infection and the associated disease has occurred sporadically and has not been intensively investigated.
- The virus is associated with outbreaks of mortality in budgerigars and other psittacine birds, especially after the introduction of new birds into a flock.
- There are no suitable diagnostic tests presently available for live birds.
- It has been suggested that recovered birds may carry the infection but this has not been proved. There is no indication of the time during which birds could remain carriers.

There are no *Code* recommendations relating to the disease.

Options for the effective management of the virus in the commodity, given in ascending order of stringency include the following:

### **Option 1.**

Birds to be imported could be clinically healthy and come from closed flocks with no history of outbreaks of mortality during the previous 2 years.

Note that 2 years is a somewhat arbitrarily selected time since there is no definite evidence on which to base a time more precisely. However, if no disease has occurred and newly fledged budgerigars do not become infected it seems safe to assume that the virus is not present in the flock. The suggested 2 year period is considered to be conservative.

### **Option 2.**

Birds to be imported could be:

- i. clinically healthy and come from closed flocks in which outbreaks of mortality have not occurred during the previous 2 years; and
- ii. held in isolation for 4 weeks during which time they are held in close contact with sentinel birds of equal health status from a flock with no history of outbreaks of mortality during the previous 2 years

Birds that die while in quarantine could be submitted to a full post mortem examination including histopathology and immunohistopathology and virus isolation.

### **Option 3.**

Birds to be imported could be:

- i. restricted to clinically healthy birds that come from closed flocks in which outbreaks of mortality have not occurred during the previous 2 years; and
- ii. limited to young recently fledged birds; and
- iii. held in isolation for 4 weeks and birds that die while in quarantine could be submitted to a full post mortem examination including histopathology and immunohistopathology and virus isolation. Identification of reovirus infection could result in disqualification of all birds in the consignment.

Note that the development of suitable diagnostic tests should not be difficult and when these are available additional options could be suggested.

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## 13. Proventricular Dilatation Disease (Macaw Wasting Disease)

### 13.1. HAZARD IDENTIFICATION

#### 13.1.1. Aetiological agent

Unidentified, but believed to be viral (Girling 2004).

#### 13.1.2. OIE list

Not listed.

#### 13.1.3. New Zealand status

In a personal communication to B Simpson, A Johnstone reported on a case that histologically resembled psittacine dilatation disease in a conure (Simpson 2007). No further suspicious cases have been reported. Since this disease can only be diagnosed from histological lesions, a single case may not be considered a definitive diagnosis. For the purposes of this risk analysis it is regarded as an exotic disease.

#### 13.1.4. Epidemiology

Proventricular dilatation disease appears to be specific to parrots and is most commonly seen in macaws, conures, African grey parrots and cockatoos (Snowdon 1995). The condition affects birds of all ages but is most common in young birds, allowing for a prolonged induction period of up to 4 years (Gregory 1995). Clinical signs of depression and weight loss, vomiting and undigested feed in faeces are due to reduced peristalsis caused by the destruction of the intramural ganglia of the proventriculus, gizzard and to a lesser extent, the upper duodenum (Gregory 1995; Gregory et al 1996).

Following its first description in South America, the disease spread to North America and Europe. It has also been reported post-quarantine in Australia in one of a consignment of 102 macaws in 1993 (Sullivan et al 1997), and more recently as a cluster of cases in Queensland (Doneley et al 2007).

Budgerigars, spoonbills, rock pebbles and toucans have shown microscopic changes “suggestive of proventricular dilatation disease” (Gregory 1995). This is the only report located of possible proventricular dilatation disease in budgerigars. Other authors (Girling 2004; Snowdon 1995) state that proventricular dilatation disease is restricted to psittacine birds, with preponderance of cases in larger parrots. Gregory (1995) goes on to state that “It should be noted that lesions suggestive of proventricular dilatation disease have been described in birds that die from other causes and have absolutely no gross or clinical signs of the disease”.

Progressive weight loss, regurgitation and passing of undigested food, especially in association with neurological signs is suggestive of the presence of proventricular dilatation disease. A presumptive diagnosis may be made on clinical signs, aided by contrast radiography but a more definitive ante-mortem diagnosis requires histopathological examination of biopsies of the gizzard, or with less clinical risk, the crop (Gregory et al 1996).

### 13.1.5. Hazard identification conclusion

Proventricular dilatation disease is recognised in an increasing range of psittacine birds; therefore although rare in budgerigars it is considered to be a potential hazard in the commodity.

## 13.2. RISK ASSESSMENT

### 13.2.1. Entry assessment

The induction period of proventricular dilatation disease is 4 years. A closed source flock with no known history of proventricular dilatation disease will have a negligible risk of being a source of proventricular dilatation disease-infected birds. In addition there are no reports of the disease having been diagnosed in budgerigars in the UK, and world-wide it is a disease of large parrots. Since a closed flock of budgerigars in the UK would not have contact with large parrots the likelihood of entry is considered to be negligible.

### 13.2.2. Risk estimation

The likelihood of entry is assessed as negligible. As a result the risk estimate for the proventricular dilatation disease agent is negligible and it is not classified as a hazard in the commodity.

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## 14. Papillomaviruses and Herpesviruses associated with Papillomas

### 14.1. HAZARD IDENTIFICATION

Papillomaviruses of psittacines belong to the genus *Thetapapillomavirus* in the family *Papillomaviridae*. The papillomaviruses of chaffinches belong to the genus *Etapapillomvirus* (De Villiers et al 2005).

#### 14.1.1. OIE list

Not listed.

#### 14.1.2. New Zealand status

A search of electronic databases and conventional media showed no data on the presence of papillomas and papillomatous disease in New Zealand birds.

#### 14.1.3. Epidemiology

Papillomaviruses tend to be host specific and even tissue specific (Dom et al 1993; Quinn et al 2002).

Epithelial papillomas that occur on the skin of finches (Dom et al 1993) and African grey parrots have been shown to be caused by papillomaviruses (Styles et al 2004).

There is strong evidence that internal papillomatosis (mucosal papillomatosis) is associated with infections with herpesviruses of genotype 1, 2 and 3 but not with genotype 4 which is more commonly associated with Pacheco's disease (Styles et al 2004). The amplification of DNA of a novel alpha-herpesvirus from a cloacal papilloma of an African grey parrot has been demonstrated (Styles et al 2005). Internal papillomatosis is a disease of new world parrots (Latimer et al 1997; O'Banion et al 1992; Styles et al 2004) and there is no evidence to suggest that it occurs in budgerigars. A review of the literature revealed that no Papillomavirus group-specific antigens were detected in any of the 41 lesions from (mainly) macaws and Amazon parrots, using immunoperoxidase staining, DNA hybridisation, electron microscopy or inoculation of homogenate into other birds (Snowdon 1995).

A single case of a "palate papilloma" was recorded amongst the 596 cases of enteric disease diagnosed in dead birds (Baker 1996).

#### 14.1.4. Hazard identification conclusion

There is a lack of evidence for the involvement of a transmissible viral aetiology for papillomas in birds other than finches and African grey parrots. In addition internal papillomatosis is a disease of "new world" parrots. Therefore, papillomaviruses and herpesviruses causing papillomas are not considered to be hazards in the commodity.

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References marked\* have been sighted as summaries in electronic databases

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## 15. Arboviruses

### 15.1. HAZARD IDENTIFICATION

Arthropod-borne viruses. The viruses concerned belong to the families *Togaviridae*, *Flaviviridae*, *Rhabdoviridae*, *Bunyaviridae* and *Reoviridae*.

#### 15.1.1. OIE list

Not listed.

#### 15.1.2. New Zealand status

Whataroa virus is the only recognised New Zealand arbovirus of birds (Maquire et al 1967). An orbivirus was found in ticks collected from penguins on Macquarie Island (Duignan 2001).

#### 15.1.3. Epidemiology

Arthropod-borne viruses (arboviruses) are a heterogeneous group but most are maintained in reservoir hosts which are often subclinical carriers of the viruses. Only a small number of known arboviruses cause serious diseases in their maintenance hosts (Buckley et al 2006; Buckley et al 2003) but several cause disease when they are transmitted to species that are not their natural hosts. The only known endemic flavivirus in the UK is louping ill virus which causes disease in sheep and in upland grouse.

There are no published reports of the isolation of West Nile virus (WNV), Usutu Virus (USUV) or Sindbis virus (SINV) in the UK (Buckley et al 2003). There is also no evidence of disease caused by West Nile virus (WNV), Usutu virus (USUV) or Sindbis virus (SINV) in the UK. However, virus-specific neutralizing antibodies have been reported in the sera of resident and migrant birds in the UK, implying that these viruses are occasionally being introduced, possibly by mosquitoes (Buckley et al 2006; Buckley et al 2003). However, no psittacine birds were included in the sample, and no evidence could be found of infection in budgerigars or other psittacine birds. Spur and Sandlant (2004) list budgerigars as being amongst the species of birds known to have been infected with WNV in the USA, but all references were concerned with experimental infection; one such study indicated that budgerigars were not competent hosts of the virus (Komar 2003).

Orbiviruses are members of the virus family *Reoviridae*. The overwhelming majority of these viruses have been isolated from seabirds (Nuttall 1993). The minority that have been isolated from terrestrial birds are transmitted by ixodid ticks more commonly associated with mammals than birds, or by soft (argasid) ticks (Nuttall 1993). Only one report was found of an orbivirus infection in psittacine birds. In this case viruses were isolated from a dead budgerigar and a lovebird. The budgerigar isolate caused a greenish diarrhoea four to eight days after experimental inoculation, but all birds had fully recovered by 28 days post inoculation, and there were no residual lesions. 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).

No reports were found of other arboviruses occurring in birds in the UK.

#### 15.1.4. Hazard identification conclusion

Evidence of West Nile virus and other flaviviruses of birds in the UK is limited to a few reports of seroconversion. There are no reports of infection in budgerigars, except reports of experimental infection from the USA that showed that budgerigars are not competent vectors of WNV. Therefore these viruses are not considered potential hazards in the commodity.

Most reports of orbivirus infections concern seabirds. The sole report of *Orbivirus* infection of psittacines comes from the USA and is 28 years old. There is no evidence to suggest that budgerigars in the UK have ever been infected. Therefore it is considered that orbiviruses are not a hazard in the commodity.

No other arboviruses were identified as potential hazards in the commodity.

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## 16. Rotavirus Infections

### 16.1. HAZARD IDENTIFICATION

#### 16.1.1. Aetiological agent

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

#### 16.1.2. OIE list

*Not listed.*

#### 16.1.3. New Zealand status

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

#### 16.1.4. Epidemiology

Rotaviruses are currently classified into five species and two additional tentative species. Viruses of different species are believed to be unable to reassort their genome segments and are antigenically distinct. Viruses within a species usually have less than 10% sequence variation in their genomes while those from different species have more than 30%. However, since the distinctions between species were not known information in the older literature on classification of species is often lacking. Rotavirus species occur commonly in many countries (McNulty 2003). Rotavirus D, F and G have only been found in birds (McNulty 2003). A virus with characteristics of a rotavirus was isolated from a lovebird but it was not determined whether it was the cause of any pathological changes in the lovebird or was an incidental isolate (McFerran et al 1976; Ritchie 1995). In chickens the virus causes mild and usually not fatal diarrhoea or subclinical infections. The incubation period in experimentally infected birds is 2-5 days and horizontal infection occurs by direct or indirect contact between birds (McNulty 2003). Young birds are most commonly infected but older birds that have escaped infection may be more susceptible. Infected chickens and turkeys excrete large numbers of viruses in their faeces. The virus can be readily demonstrated in the faeces of most birds in infected flocks. Peak virus excretion occurs 3 days post infection and sometimes continued for more than 16 days in turkey poults, but there is no evidence for the occurrence of carriers (McNulty 2003). The best estimate of what is likely to occur in budgerigars must be made by extrapolation of data from chickens and turkeys.

There are no vaccines available for budgerigars.

#### 16.1.5. Hazard identification conclusion

Since no reports could be located on the occurrence of rotaviruses in psittacine birds they are not classified as potential hazards in the commodity.

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## 17. *Salmonella* spp. (Salmonellosis)

### 17.1. HAZARD IDENTIFICATION

#### 17.1.1. Aetiological agents

Modern nomenclature (Davies 2004; Wray and Davies 2002) classifies the genus *Salmonella* into only two species, *S. enterica* and *S. bongori*, with *Salmonella enterica* divided into six subspecies.

Subspecies of salmonellae are classified into serovars. The serovars most commonly causing infections in humans and food animals belong to subspecies 1. The other subspecies are common in reptiles, although some serovars of subspecies *arizonae* are associated with disease in poultry and sheep. According to this latest nomenclature *Salmonella typhimurium* is now known as *Salmonella enterica* subspecies *enterica* serovar Typhimurium (Anonymous 2004). This is cumbersome, and the suggestion of (Old 1992) that the serovar name be written in Roman type is used in this chapter, e.g. *Salmonella* Typhimurium.

#### 17.1.2. OIE list

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

#### 17.1.3. New Zealand status

The following *Salmonella* spp. are unwanted organisms: *Salmonella* Arizonae, *Salmomella* Abortus ovis, *Salmonella* Dublin, *Salmonella* Enteritidis DT4, *Salmonella* Typhimurium DT 44 and DT104, *Salmonella* Gallinarum, *Salmonella* Pullorum, *Salmonella* spp. (exotic affecting animals).

All *Salmonella* isolates identified in New Zealand are referred to the Institute of Environmental Science and Research (ESR) laboratory for detailed typing. Records of all isolates made are available at the ESR public Health Surveillance website (ESR 2007). None of the unwanted organisms occur in New Zealand except for rare isolates of *Salmonella* Typhimurium DT104 in humans (ESR 2007).

The following isolates were made from humans in 2005:

*Salmonella* Typhimurium: 757 isolates including 1 isolate of the definitive phage type DT 104.

*Salmonella* Enteritidis: 157 isolates.

Other isolates: 546 isolates consisting of 84 serotypes.

The following isolates were made from non-human sources:

*Salmonella* Typhimurium 571 isolates with no isolates of DT 104

*Salmonella* Enteritidis 4 isolates

Other isolates: 949 isolates consisting of 62 serotypes

The year 2005 is a typical year and variations from year to year are minimal e.g. the Enteric Reference Laboratory confirmed 1,417 non-human salmonella isolates in 2006 compared with 1,520 in 2005. Prior to 2003 these results were reported in the ESR's Lablink publication (ESR 2003).

In 2006 *Salmonella* Enteritidis DT4 and DT4b were isolated in 4 months from human sources, and there were two isolates of *S* Typhimurium DT104. *Salmonella* Enteritidis phage types 1,1b, 3, 4, 4b, 6, 8, 9A, 13, 13a, 14b, 21, 23 25, and 26 have been reported in New Zealand's human population in the last two years (ESR 2007). Only phage types 4b, 9 and 9a have been isolated from animals (ESR 2007). Isolates of these serotypes have been reported from miscellaneous poultry sources but not livestock, backing up earlier evidence that none have become established in the poultry industry (ESR 2007; MAF 1996; MAF. 1997). Apart from DT9A, which is widespread in animals and humans in this country, isolates of other phage types are mainly associated with returning overseas travellers or foreign visitors.

#### 17.1.4. Epidemiology

*Salmonella* spp. are of concern because they cause animal disease and are also pathogenic for humans. In veterinary literature a distinction is usually made between infections caused by the two non-motile serovars, *Salmonella* Pullorum (pullorum disease) and *Salmonella* Gallinarum (fowl typhoid), which are host-adapted serovars of poultry, and the remainder referred to as paratyphoid salmonellae (Gast 2003a; Gast 2003b). The *arizonae* group of salmonellae (arizonosis) which mainly affects turkeys amongst avian hosts (Gast 2003a) is included within paratyphoid salmonellae. Although there are over 2,400 serotypes of paratyphoid salmonellae (Gast 2003a; Wray and Davies 2002), only about 10% of these have been isolated from poultry, and an even smaller subset account for common poultry isolates (ESR. 2003; Wray and Davies 2002).

The major food borne serotypes are *Salmonella* Typhimurium and *Salmonella* Enteritidis. Although *Salmonella* Typhimurium is common in New Zealand, the definitive phage type (DT) 104 that is of particular concern because of its multiple resistance to common antibiotics, occurs rarely and its low prevalence has remained stable, as can be verified from the ESR databases (see section 16.1.3). This is in contrast to the situation in many countries where *Salmonella* Typhimurium DT104 occurs commonly (Hogue et al 1997; Jones et al 2002).

Gram negative bacteria are less common in the intestinal flora of psittacines than gram positive bacteria (Bangert et al 1988; Flammer and Drewes 1988). *Salmonella* Typhimurium has been isolated from budgerigars and other psittacine birds. One case of pneumonia due to *Salmonella* Typhimurium was recorded amongst 1,525 dead and euthanised birds, including 123 chicks in survey done in the UK (Baker 1996). The author also recorded salmonellosis as an occasional cause of "wet vent" in budgerigars. *Salmonella* Typhimurium was isolated from two budgerigars and a parakeet (Juntilla et al 1988), from 2.3% of 466 aviary psittacine birds and 80 faecal samples (Dorrestein et al 1985), and there were 29 isolates from 533 pet aviary birds examined (Panigrahy et al 1984). Reports of the occurrence of *Salmonella* spp. other than *Salmonella* Typhimurium in budgerigars were not found.

*Salmonella* Pullorum was last diagnosed in New Zealand in 1985, and *Salmonella* Gallinarum and *Salmonella* Arizonae have never been recorded in New Zealand (OIE 2007). The natural hosts of *Salmonella* Pullorum-Gallinarum are poultry, and whilst literature surveys indicate a number of investigations of aviary birds in the vicinity of infected poultry operations, there have been no reports of infection in psittacine birds. *Salmonella* Arizonae has been isolated from sulphur-crested cockatoos, but these birds

were housed with iguanas (reptiles are natural hosts of *Salmonella* Arizonae) (Oros et al 1998). No records of the isolation of *Salmonella* Arizonae from psittacine birds in the UK could be found.

A large number of *Salmonella* serotypes, occur in the UK (Sojka et al 1983).

Diagnosis of *Salmonella* spp. in cases of subclinical infection presents problems. Serological tests are available only for some serotypes. In particular agglutination tests are used for *Salmonella* Pullorum and *Salmonella* Gallinarum in poultry and an ELISA is available for *Salmonella* Enteritidis. These tests are used as flock tests rather than individual animal tests. *Salmonella* can be cultured from faeces or cloacal swabs but in the case of carriers excretion of the organism may be intermittent and culturing should be done on more than one occasion. Flock testing rather than individual testing is recommended.

#### 17.1.5. Hazard identification conclusion

*Salmonella* Pullorum and *Salmonella* Gallinarum are pathogens of poultry and are rare in the UK (Veterinary-Laboratory-Agency 2006). There is no evidence of their occurrence in budgerigars in the UK. *Salmonella* Arizonae are pathogens of turkeys and reptiles and there is no evidence of their occurring in budgerigars in the UK. Therefore the likelihood of these *Salmonella* spp. being present in budgerigars sourced from the UK is considered negligible and they are not considered to be potential hazards in the commodity.

*Salmonella* spp. are rarely isolated from Budgerigars. However, since *Salmonella* spp. are zoonotic agents and responsible for disease in humans and other animals, the introduction of exotic and rare *Salmonella* serotypes should be avoided. Therefore exotic strains of *Salmonella* spp. are regarded as potential hazards in the commodity.

## 17.2. RISK ASSESSMENT

### 17.2.1. Entry assessment

Many *Salmonella* serotypes occur in the UK but salmonellosis is rare in budgerigars. The likelihood of introducing *Salmonella* spp. in budgerigars imported from the UK is considered to be low but non-negligible.

### 17.2.2. Exposure assessment

Since budgerigars will be introduced into New Zealand stud flocks and may be traded and transported to shows, at which they will be in contact with other birds, the imported *Salmonella* serotypes are could be spread widely and infect other birds, animals and people. Therefore, the likelihood of exposure is non-negligible.

### 17.2.3. Consequence assessment

If introduced new *Salmonella* spp. could cause disease in birds and other animals. This could result in losses in production and costs for treatment.

Since humans may be in contact with budgerigars and their faeces, transmission to humans is likely to occur. The occurrence of new types of salmonellosis in humans is likely to cause loss of productivity, expenses for medical treatment and even in rare cases death. Introduction of

antibiotic resistant *Salmonella* Typhimurium DT 104 could result in difficulties in treatment of human cases. All animals are susceptible to infection with *Salmonella* spp., and therefore wild and feral birds and animals could become infected. An outbreak of a new phage type of *Salmonella* Typhimurium (DT160) occurred in sparrows and in humans in 2001 (Alley et al 2002). The outbreak accounted for several hundred deaths in sparrows, but was self-limiting and did not cause lasting damage to the sparrow population (Connolly et al 2006).

The consequences of introduction are therefore considered to be non-negligible.

#### 17.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for exotic *Salmonella* serovars is non-negligible and they are classified as hazards in the commodity. Therefore risk management measures can be justified.

### 17.3. RISK MANAGEMENT

#### 17.3.1. Options

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

- Salmonellosis is rare in budgerigars and introduction of *Salmonella* spp. is much more likely through other pathways
- Since long-term carriers may occur, quarantine on its own is unlikely to be effective.
- Suitable generic serological tests for a wide variety of *Salmonella* spp. are not available for export testing.
- The testing of cloacal swabs from a sample of birds could be used to detect infected flocks. Information is not available for the prevalence of many *Salmonella* spp. in budgerigars and selection of sample size is therefore somewhat arbitrary. To compensate for the fact that cases of infection may occur sporadically or at low prevalence, a sample that is large enough to detect at least one infected budgerigar with a confidence of 95% if the prevalence is 5% could be tested.
- When making cultures from individual animals, isolation of single colonies of *Salmonella* is unusual. Therefore pooling of samples could be permitted in appropriate cases.
- Samples for testing could include cloacal swabs from a sample of birds in the flock and also from the aviary environment such as litter samples, swabs from the floor surface, perches and nest boxes.
- Flock freedom from *Salmonella* infection would provide a high level of assurance of freedom and would compensate for the decreased sensitivity due to intermittent excretion, when testing individual birds.

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

Therefore available options in ascending order of stringency are:

#### **Option 1.**

- i. within the 10 days prior to shipment, cloacal swabs from individual budgerigars could be cultured; and
- ii. all isolates of *Salmonella* spp. could be identified to serovar and in the case of *Salmonella* Enteritidis and *Salmonella* Typhimurium to phage type, and the results forwarded to MAFBNZ for consideration.

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

### Option 2.

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

### Option 3.

A possible variation on option 1 is that swabs could be taken from individual birds before entry into quarantine and twice while in quarantine. All other requirements could be the same as in option 1.

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## 18. Intestinal Spirochaetes

### 18.1. HAZARD IDENTIFICATION

#### 18.1.1. Aetiological agents

*Brachyspira hyodysenteriae*, *Brachyspira pilosicoli*, *Brachyspira* *Brachyspira intermedia*, *Bracyispira innocens*, *Brachyspira murdochii* and *Brachyspira alvinipulli*. The former two are pathogens and *Brachyspira intermedia* is of intermediate pathogenicity. Other *Brachyspira* are non-pathogenic or of doubtful pathogenicity. *Bracyhyspira pilosicoli* is an important pathogen of chickens and pigs (Hampson 2006) and *Brachyspira hyodysenteriae* is the most important pathogen of pigs (Hampson et al 2006).

#### 18.1.2. New Zealand status

*Brachyspira pilosicoli*, *Brachyspira hyodysenteriae* and *Brachyspira innocens* have been isolated (Midwinter 1999).

#### 18.1.3. Epidemiology

*Brachyspira* spp. are considered to be widely distributed, occurring in Australia, the Netherlands, England and the United States (Swayne 2003) and probably most other countries. It occurs in a wide variety of animals, birds and man (Feberwee et al 2007; Hampson 2006; Shivaprasad and Duhamel 2005; Swayne 2003). *Brachyspira dysenteriae* and *Brachyspira pilosicoli* are generally considered to be pathogens while other species are mildly pathogenic or non-pathogenic. At least three species, including the two pathogenic species already occur in New Zealand (Midwinter 1999). An electronic search of three databases found no reference to intestinal spirochaetosis in psittacine birds.

#### 18.1.4. Hazard identification conclusion

*Brachyspira* spp occur commonly in many animals and the pathogenic species are already in New Zealand. As no references were found to them occurring in psittacine species they are not considered to be potential hazards in the commodity.

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## 19. *Coxiella burnetii* (Q Fever)

### 19.1. HAZARD IDENTIFICATION

#### 19.1.1. Aetiological agent

*Coxiella burnetii*, rickettsial organism.

#### 19.1.2. OIE status

Listed as disease of multiple species but no recommendations are given concerning the disease in the *Code*.

#### 19.1.3. New Zealand status

Notifiable organism (MAF 2007).

#### 19.1.4. Epidemiology

*Coxiella burnetii* is known to infect a very wide number of vertebrate species including birds (Martinov et al 2004; Maurin and Raoult 1999; Sakai et al 1998). It is maintained in nature in wild bird populations between which it is transmitted by ticks (Campbell 1994). Organisms can be recovered from liver and spleen of infected chickens for up to 3 months (Barnes 2003). Organisms can be recovered from faeces of infected animals for up to 40 days post-infection (Little 1983), but whether a similar period of excretion occurs in other birds is not known. Infection in birds is subclinical (Barnes 2003). No reports of *Coxiella burnetii* infection of budgerigars in particular, or psittacine birds in general, could be located in reviews of the disease in the UK.

Q fever does not occur in New Zealand but it is endemic in the UK. About 70 cases of Q fever in humans are reported in the UK each year, mainly resulting from occupational exposure (HPA Centre for Infections 2005). No reference to the occurrence of Q fever in psittacine birds was found.

Antibody can be detected in birds using complement fixation, immunofluorescence, microagglutination tests or ELISA (Fournier et al 1998; Maurin and Raoult 1999). The organism can be detected in clinical samples by a PCR or the organism can be isolated in embryonated eggs or cell cultures (Auricu-Bovery et al 2003; Fournier et al 1998; Maurin and Raoult 1999).

In humans the disease usually presents as an asymptomatic or mild infection but rare cases of serious disease including myocarditis, hepatitis, joint infections, and death can occur (Maurin and Raoult 1999). No reports of humans infected or suspected of being infected by contact with psittacine birds were found.

#### 19.1.5. Hazard identification conclusion

Since the organism is exotic to New Zealand (MAF 2007) and occurs endemically in the UK, the organism is considered to be a potential hazard in this risk analysis.

## 19.2. RISK ASSESSMENT

### 19.2.1. Entry assessment

No records of the infection of budgerigars or other psittacine birds with *Coxiella burnetii* could be found. Generally the prevalence of antibody to *Coxiella burnetii* in birds is highest where they have close contact with domestic livestock (Riemann et al 1979; To et al 1998). Therefore the likelihood of importing infected budgerigars from a single source closed budgerigar stud maintained indoors is considered negligible.

### 19.2.2. Risk estimation

The entry assessment for budgerigars imported from the UK is negligible. As a result the risk estimate for *Coxiella burnetii* is negligible and it is not classified as a hazard in the commodity. Therefore risk management measures are not justified

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## 20. Protozoal Blood Parasites (Haematozoa)

### 20.1. HAZARD IDENTIFICATION

#### 20.1.1. Aetiological agents

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

#### 20.1.2. OIE list

Not listed.

#### 20.1.3. New Zealand status

The following species have been recorded in New Zealand. *Haemoproteus danilewsky*, *Leucocytozoon tawaki*, *Leucocytozoon fringillinarum*, *Plasmodium cathermerium*, *Plasmodium elongatum*, *Plasmodium relictum*, *Plasmodium* sp. (McKenna 1998).

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

#### 20.1.4. Epidemiology

A number of species of blood parasites belonging to the genera *Haemoproteus*, *Leucocytozoon* and *Plasmodium* have been described in birds world-wide. Descriptions of these parasites have generally been made by taxonomists who have identified them in clinically normal birds, as in a study conducted in Pakistan (Rukhsana 2005). A large number of organisms have been described. For example, in a single investigation, 112 new host-parasite associations were described (Valkiunas et al 2005). Several examples indicate that the nomenclature of organisms and their reported host associations are unstable (Bennett and Peirce 1990; Gabaldon and Ulloa 1976).

Most haematozoa seem to live in balance with their avian hosts as commensals. Some may be opportunistic pathogens, particularly when they occur in combination with other infections. Association with disease does not necessarily indicate pathogenicity since the disease may have been caused by another unidentified pathogen and the presence of the haematozoan parasite may be incidental. The pathogenicity of the above-listed parasites has seldom been proven by experimental infection. In a study on the pathogenicity of *Leucocytozoon simondi*, a parasite that has often been associated with heavy mortality in ducks and geese, it was found that experimental infection did not cause clinical signs or affect the growth rates of infected American black ducks and mallard (Shutler et al 1999). It appears that the majority of described parasites are non-pathogenic or mildly pathogenic in the species in which they have evolved. However, when transmitted to naïve species they may be pathogenic. The best example of this is the introduction of the mosquito vector (*Culex quinquefasciatus*) and its associated parasite *Plasmodium relictum* into Hawaii. The introduction caused habitat restrictions and extinctions of several Hawaiian bird species (Atkinson and LaPointe 2005). Another example is the susceptibility of penguins to avian malaria. However, some species may adapt to the presence of new species of parasites. Infection with, and recovery from avian malaria provides a

reproductive advantage to the Hawaiian honeycreeper (Kilpatrick et al 2005) and adaptation of Hawaiian bird species to avian malaria may already be occurring.

An extensive survey in which blood smears were examined from 1,234 birds belonging to 186 species collected from pet shops and importers was undertaken in the USA. Before the survey, large numbers of birds had been imported into the USA from Australasia, Africa, South America, and the Caribbean islands. Five new species of *Plasmodium* were identified and other *Plasmodium* spp. were common. Members of the Fringillidae and the Psittacidae families made up the largest proportion of birds investigated but no blood parasites were seen in the latter. Amongst other birds *Haemoproteus* spp. were the commonest blood parasites, followed by *Plasmodium* spp., *Leucocytozoon* spp. and *Atoxoplasma* spp. were very rare (Manwell and Rossi 1975).

Few reports of haematozoa belonging to the *Haemoproteus*, *Leucocytozoon*, *Plasmodium* and *Trypanosoma* genera have been described in the UK. A leucocytozoon-like infection in budgerigars and other antipodean parakeets was reported (Simpson 1991). No reliable information could be found regarding the vector. Two cases (0.43%) of *Leucocytozoon* infection were reported in a survey of euthanased birds in the UK (Baker 1996). No cases of *Haemoproteus* were reported. (Pierce and Bevan 1977) reported the finding of haematozoan parasites in 15% of 117 blood films examined from psittacine birds imported into the UK. The most common parasites were microfilaria and *Haemoproteus*, but *Aegyptianella* and a *Trypanosoma* sp. were also observed. No budgerigars were included in the sample.

### ***Haemoproteus* spp.**

More than 120 species of *Haemoproteus* have been reported from birds, but most species have low pathogenicity (Bermudez 2003). However, outbreaks of disease have been associated with the parasite but it is not known whether there were other complicating infections involved (Resende et al 2001). No reports of *Haemoproteus* infections of budgerigars could be found, although infection has been reported in other psittacine birds being imported into the UK.

*Haemoproteus danilewsky* has been described in blackbird, skylark and song thrush in New Zealand (McKenna 1998).

### ***Leucocytozoon* spp.**

*Leucocytozoon* infections were detected in two euthanased birds in a UK survey (Baker 1996). Disease associated with the infection was not reported. No reports were found where *Leucocytozoon* infection of psittacine birds was associated with disease.

### ***Plasmodium* spp.**

About 65 species of *Plasmodium* from more than 1,000 species of birds have been described but only 35 or fewer are considered valid (Bermudez 2003). The parasites, which are the agents of avian malaria, probably occur world-wide (Bermudez 2003). Pathogenicity of various species ranges from non-pathogenic to virulent. Pathogenic species can cause severe anaemia and death. *Plasmodium relictum* is a serious pathogen of Hawaiian birds that have not evolved with the parasite (Atkinson and LaPointe 2005). 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. *Culex quinquefasciatus* is established in New Zealand (Holder and Brown 1999), and *Plasmodium relictum* is already present in large areas and has caused mortality in some species (Derraik 2006; Tomkins and Gleeson 2006). This parasite has spread southward mirroring the spread of *Culex quinquefasciatus* since the mosquito was introduced into the Auckland region some time prior to 1919 (Tomkins and Gleeson 2006). However, the bird mortalities have been less severe than those documented in Hawaii.

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 could be due to a paucity of suitable vectors. New Zealand's indigenous birds could be presented as a naïve and highly susceptible population in relation to introduction of haematozoa. 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. Each bird was infected with at least two species of *Plasmodium* (Tomkins and Gleeson 2006). However, budgerigars were imported until a few years ago, either directly or via quarantine facilities in Australia, without noticeable effects on indigenous birds.

The diagnosis of avian malaria has been greatly improved by the development of PCR methods which detect several species of malarial parasites with much greater sensitivity than blood smear examination. The PCR method recognises species from the genera *Haemoproteus*, *Leucocytozoon*, and *Plasmodium* (Cosgrove et al 2006; Hellgren et al 2004; Tomkins and Gleeson 2006).

#### 20.1.5. Hazard identification conclusion

Haematozoa are seldom recognised to be of any economic significance and are not listed in import health standards of other countries. The position with regard to the presence of these parasites in New Zealand is not certain and their ability to establish here cannot be predicted. Since various haematozoa have been described in birds in the UK, they are considered to be potential hazards in the commodity.

## 20.2. RISK ASSESSMENT

### 20.2.1. Entry assessment

Since a variety of haematozoa occur in the UK the likelihood that imported budgerigars could be carrying exotic protozoa is considered to be non-negligible.

### 20.2.2. Exposure assessment

Imported budgerigars are unlikely to mix extensively with New Zealand birds. However, although unlikely, escapes of budgerigars and subsequent contact with other birds and parasite vectors cannot be entirely 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 as non-negligible.

### 20.2.3. Consequence assessment

Budgerigars have been imported from the UK in the past with no adverse effects. No descriptions of disease caused by haematozoa in budgerigars could be located. Therefore the likelihood of importing parasites pathogenic to budgerigars is low. However, the introduction of *Plasmodium relictum* to naïve bird populations had serious consequences for native birds in Hawaii and vector systems for at least some protozoal parasites are already present in New Zealand. *Plasmodium relictum* is already endemic in this country (Derraik 2006; Tomkins and Gleeson 2006) but the introduction of other haematozoa might be harmful. Therefore the consequences for native birds are speculative but are assessed as non-negligible.

No evidence was found suggesting that any of the haematozoa of birds infect mammals or reptiles. There would be no consequences for human health since haematozoa of birds are not zoonotic.

### 20.2.4. Risk estimation

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

## 20.3. RISK MANAGEMENT

### 20.3.1. Options

The following points were considered when drafting options for preventing the introduction of haematozoa:

- Since budgies were imported into New Zealand for almost 150 years up to 1997, it could be considered likely that the haematozoa that are associated with budgies have already been introduced into this country.
- Birds are long term carriers of many haematozoa and quarantine would not be a useful measure to prevent their introduction.
- Serological tests are not available to identify carriers of the many possible haematozoan parasites.
- Some avian malarial parasites can be diagnosed by sensitive PCR tests.
- Microscopic examination of blood smears is the only available method of identifying all species of parasites but it lacks sensitivity.
- To improve the sensitivity of both blood smear examination and PCR, blood smears could be examined from a sample of birds from the flock of origin rather than individual birds. Since the prevalence of haematozoa in budgerigars is unknown the sample size could be chosen assuming a low prevalence to provide high flock sensitivity e.g. The sample could be taken from a sufficient number of birds to provide 95 % confidence that the sample will contain at least one infected bird if the prevalence of infection is 5%.
- Strict measures could be implemented to prevent the introduction of haematophagous arthropod parasites that could act as biological or mechanical vectors of protozoal parasites.

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

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

### Option 1.

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

### Option 2.

PCR as primary test with further confirmatory tests on positives

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

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

### Option 3.

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

- i. blood smears from birds to be imported could be examined microscopically for haematozoa. Birds infected with exotic haematozoa could be disqualified; and
- ii. blood from birds to be imported could be examined by PCR for haematozoa. Birds infected with exotic haematozoa could be prohibited from being imported.

### Option 4.

Flock of origin testing

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

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## 21. Protozoa other than Haematozoa

### 21.1. HAZARD IDENTIFICATION

#### 21.1.1. Aetiological agent

Genera considered are *Eimeria*, *Sarcocystis* and *Trichomonas*, *Cryptosporidium* spp. and *Giardia*.

#### 21.1.2. OIE list

There are no protozoal diseases of birds listed in the *Code*.

#### 21.1.3. New Zealand status

Thirty species of protozoa from birds have been reported in New Zealand (McKenna 1998). None have been recorded in budgerigars.

#### 21.1.4. Epidemiology

At least 30 species of intestinal protozoal parasites already occur in birds in New Zealand, including the intestinal parasites known to cause significant disease in budgerigars (McKenna 1998). *Trichomonas gallinae* (Baker 1986; McKeon et al 1997) and *Giardia psittaci* (Box 1981; Filippich et al 1998) are known to infect budgies.

*Giardia psittaci* is a species distinct from *Giardia lamblia* and *Giardia duodenalis* that infest man (Erlandsen and Bemrick 1987). *Giardia* spp. have been described in at least 7 bird species in New Zealand but have not been identified to species level (McKenna 1998). The taxonomy of *Giardia* spp. is confused and has not yet been clearly resolved. It is considered that the likelihood that a new species of *Giardia* that does not already occur in New Zealand birds would be introduced by budgerigars from the UK is negligible.

*Trichomonas gallinae* is endemic.

*Sarcocystis* spp. are two host species with the sexual cycle of the parasite occurring in the gastrointestinal tract of a carnivorous primary host and the cyst form of the parasite occurring in the muscles or organs of the secondary (prey) host e.g. *Sarcocystis falcatula* (Dubey et al 1999). *Sarcocystis* therefore cannot be established unless an infected budgerigar is eaten by a competent carnivorous animal and a host/parasite cycle established. The likelihood of this occurring in captive imported budgerigars is considered to be negligible

Eleven *Eimeria* spp have already been described in birds in New Zealand (McKenna 1998). The commercially important species that occur in poultry are already present. *Eimeria dunsingi* has been described in budgerigars (Todd et al 1977). However, because of the paucity of information on this parasite in budgerigars since that time, it is concluded that it is of no significance as a pathogen of budgerigars. The likelihood that a new *Eimeria* spp. that will cause significant disease in poultry or other birds will be introduced in budgerigars is considered to be negligible

Cryptosporidiosis in cattle and humans caused by *Cryptosporidium parvum* and *Cryptosporidium hominis* occur commonly in New Zealand (Grinberg et al 2003; Learmonth et al 2004). *Cryptosporidium* sp. have been identified in at least four species of birds in New Zealand (McKenna 1998). Only one reference was found to cryptosporidiosis in a budgerigar (Goodwin and Krabill 1989). In this case the infestation was diagnosed histologically but the organism was not isolated or identified to species level. The likelihood that a budgerigar imported from the UK would introduce a *Cryptosporidium* species that does not already occur in New Zealand is negligible.

Most other parasites cause asymptomatic or trivial infestations and can be regarded as scientific curiosities.

#### 21.1.5. Hazard identification conclusion

Intestinal protozoal parasites are not regarded as potential hazards in the commodity.

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## 22. *Encephalitozoon hellem*

### 22.1. HAZARD IDENTIFICATION

#### 22.1.1. Aetiological agent

*Encephalitozoon hellem*

#### 22.1.2. OIE list

Not listed.

#### 22.1.3. New Zealand status

Unknown, not reported.

#### 22.1.4. Epidemiology

*Encephalitozoon* spp. are parasites with complex lifestyles that may be intestinal parasites or cause systemic infections. *Encephalitozoon hellem* was first described in 1991 (Didier et al 1991), when it was found in three patients with AIDS. It was identified in faeces from 85% of 110 samples from AIDS cases (Graczyk et al 2007a). It is clearly a widely disseminated parasite since it has been isolated from environmental sources such as landfill leachate (Graczyk et al 2007b). In immunocompromised patients the infection may become systemic and infect various organs (Didier et al 1991; Scaglia et al 1997; Schwartz et al 1992).

The organism has been identified in a number of different animals and birds including the European brown hare (De Bosschere et al 2007), the Egyptian fruit bat (Childs-Sanford et al 2006), umbrella cockatoo (Phalen et al 2006), gouldian finches (Carlisle et al 2002), budgerigars (Black et al 1997), pigeons and chickens (Sakova et al 2006) and mice have been infected experimentally (Herich et al 2006). Infections in budgies were associated with concurrent infections that included candidiasis, *Macrorhabdus ornithogaser*, avian polyomavirus, cryptosporidiosis, giardiasis and psittacosis (Phalen 2005). It may be considered that *Encephalitozoon hellem* is an opportunistic parasite that occurs in a wide variety of birds and animals and commonly occurs in humans. Descriptions of infections in birds are sporadic and little is known about the epidemiology of the infection, therefore it can be assumed to be of no economic importance. Signs of infection vary depending on whether the infection is enteric or has become systemic and which organs are infected. Signs may include weight loss, stunted growth, loose droppings, conjunctivitis and keratitis and mortality in nestling gouldian finches.

It is probable that transmission occurs by the faecal-oral route in the case of intestinal infections and through urine and other body secretions in systemic infections but this has not been confirmed. The disease can be diagnosed by traditional microscopic identification of the parasites or by PCR (Franzen et al 1998; Katzwinkel-Wladarsch et al 1997).

Treatment of human cases is with albendazole (Didier et al 2005) and treatment of rabbits with *Encephalitozoon cuniculi* is with benzimidazoles (Taylor et al 2007). Albendazole and netobimin, an albendazole precursor, have been used to treat birds (Phalen 2005), but treatments were for protracted periods. Since many cases occur in cases that are

immunosuppressed or have other confounding infections treatment may not be successful (Phalen 2005).

#### 22.1.5. Hazard identification conclusion

Only sporadic cases of infection have been reported in birds and infections are likely to be sub-clinical. The disease is unlikely to be of economic importance and infection in humans is common in AIDS patients and likely to occur at a high rate in a sub-clinical form in humans. The disease is of minor economic importance and the agent is an opportunistic pathogen that occurs widely in the world and there are no meaningful methods that could be employed to prevent the introduction of the organism by humans, birds or other animals. Therefore, *Encephalitozoon hellem* is not considered to be a potential hazard in the commodity.

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## 23. Exotic Fungi and Yeasts

### 23.1. HAZARD IDENTIFICATION

#### 23.1.1. Aetiological agent

Fungi and yeasts considered are

- Dermatophytes - *Microsporum* spp. and *Trichophyton* spp.,
- *Histoplasma* spp.,
- *Cryptococcus* spp.,
- *Candida* spp.,
- *Aspergillus* spp.,
- Zygomycetes
  - *Absidia* spp,
  - *Mortierella* spp.,
  - *Mucor* spp. and
  - *Rhizopus* spp.

#### 23.1.2. OIE list

*Histoplasma farciminosum* is listed by the OIE. However, it causes a disease of horses not birds.

#### 23.1.3. New Zealand status

*Histoplasma farciminosum* (*Histoplasma capsulatum* variety *farciminosum*) is listed in the register of unwanted organisms.

#### 23.1.4. Epidemiology

Fungi are widely distributed in the world and are saprophytes or opportunistic pathogens. The epidemiology of fungi and yeasts has been fully reviewed in relation to birds in the risk analysis for the importation of passerine hatching eggs and it was concluded that fungi and yeasts are not hazards (Simpson 2006) . It has been stated that “Dissemination (of fungi) occurs as a result of their saprophytic lifestyle: infection is dead end, with exception of dermatophytoses, because mycoses are not contagious” (Kunkle 2003). *Candida* spp. occur world-wide and are part of the normal microflora of humans animals and birds (Kunkle 2003). Dermatoses occur worldwide including New Zealand.

#### 23.1.5. Hazard identification conclusion

Fungi and yeasts, except dermatophytoses, are opportunistic pathogens that are not contagious and dermatophytoses occur worldwide. Therefore fungal infections are not considered hazards in the commodity.

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## 24. Internal Parasites

### 24.1. HAZARD IDENTIFICATION

#### 24.1.1. Aetiological agent

The section covers all Nematodes, Trematodes and Cestodes.

#### 24.1.2. OIE list

There are no avian helminths listed by the OIE.

#### 24.1.3. New Zealand status

Twelve nematodes (either species or genera) are listed in the register of unwanted organisms; none of these are parasites of budgerigars. Sixty-one species of nematodes have been identified from birds in New Zealand. Only *Ascaridia* spp have been identified in budgerigars (McKenna 1998). The nematodes *Capillaria* spp., *Syngamus trachei*, *Cyathostoma cacuatua*, *Procynea kea* have been recorded from other psittacine birds in New Zealand (McKenna 1998).

Four trematodes (either species or genera) are listed in the register of unwanted organisms. Thirty one species of trematodes have been identified from birds in New Zealand (McKenna 1998). *Echinostomum revolutum* from the rock pigeon, and *Liperosomum megacotylosum* from the kiwi are the only trematodes from terrestrial birds (McKenna 1998). The rest have come from aquatic birds which are likely to eat snails, slugs and other intermediate hosts of trematodes. No reports were located describing trematodes as important parasites of budgerigars.

Five species or genera of cestodes are listed in the register of unwanted organisms. Twenty three species of cestodes have been identified from birds in New Zealand (McKenna 1998). The cestodes *Pulluterina nestoris* and *Stringopotaenia psittacea* have been recorded from other psittacine birds in New Zealand (McKenna 1998). No reports of tapeworms as significant parasites of budgerigars were located.

#### 24.1.4. Epidemiology

A scan of databases revealed a small number of helminth parasites of species and genera not recorded in New Zealand, but present in psittacine birds, including budgerigars, in other parts of the world.

Searches of online databases indicate that host parasite relationships records for psittacine birds in the UK are inadequate. In particular no records of the pathogenic parasite *Dispharynx nasuta* could be found and it is not known to occur in UK budgerigars (Binks 2006). Trematodes invariably have indirect lifecycles (McDougald 2003). As the intermediate hosts, where these are known, are usually molluscs or tadpoles, it is not surprising that the majority of trematodes are recorded from aquatic birds (McDougald 2003) (see Section 21.1.3). As psittacine birds in captivity and especially exhibition budgerigars have minimal access to potential intermediate hosts, they are unlikely to be infested with these parasites.

Cestodes also have indirect lifecycles (McDougald 2003). Records in the scientific literature for cestodes in budgerigars in the UK are as scant as those in New Zealand. Most cestodes are host specific for a single or few closely related birds (McDougald 2003).

#### 24.1.5. Hazard identification conclusion

Although budgerigars were imported from the UK until 1997, it is not certain that all species of internal parasites that occur in budgerigars in the UK have been imported and established in New Zealand. Therefore internal parasites are considered to be a potential hazard in the commodity.

## 24.2. RISK ASSESSMENT

### 24.2.1. Entry assessment

Given that budgerigars were imported into New Zealand until 1997, it is likely that the significant internal parasites of budgerigars have already been imported. Because of the uncertainties in this area the likelihood of introducing new nematode parasites is considered to be low but non-negligible. Given the negligible risk of exposure to their intermediate hosts in confined facilities, the entry assessment for cestodes and trematodes from single source donor flocks is considered to be low.

### 24.2.2. Exposure assessment

Imported budgerigars are likely to be confined indoors or in aviaries, with limited contact with other birds. Therefore opportunities to transmit parasites to wild and feral birds are limited, but faeces from the aviaries may reach the environment. Some birds will also be moved to shows. Since budgerigars do not survive for long outside heated accommodation, direct contact between escaped birds and wild birds is considered to be unlikely. However, faeces of escaped birds that are contaminated with parasite eggs could survive for longer periods and be a source of infestation. Therefore, the exposure assessment for internal parasites is low but non-negligible.

### 24.2.3. Consequence assessment

Although introduction of new species of parasites is unlikely, any new species introduced could have consequences relating to health, growth and performance of New Zealand birds. Therefore the consequences are considered to be non-negligible.

The possible consequences for indigenous birds are unknown as the susceptibility of indigenous birds to budgerigar parasites is unknown. Therefore the consequences are considered to be non-negligible.

Since internal parasites of budgerigars are not zoonotic, there are no public health consequences that could result from importing budgerigars.

### 24.2.4. Risk estimation

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

## 24.3. RISK MANAGEMENT

### 24.3.1. Options

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

- Since budgies were imported into New Zealand for almost 150 years up to 1997, it could be considered likely that the internal parasites of budgies have already been introduced into this country.
- Anthelmintic treatment of the birds to be imported could be used to eliminate internal parasites. Treatment could be for nematodes (ivermectin or other) and cestodes and trematodes (praziquantel).
- Cleaning of the pre-export quarantine premises prior to entry of the birds would ensure that they were not re-infected. Measures to prevent contact with intermediate hosts of cestodes and trematodes could be implemented.
- The testing of faecal samples prior to export would ensure that adult worms were not present at the time of export. Two tests, one on entry into quarantine and one a week prior to shipment could be used to reduce the risk of importing parasites to a negligible level. Test procedures could include flotation, sedimentation and larval culture method in order to most effectively diagnose all forms of internal parasites.

Available options in ascending order of stringency are:

#### **Option 1.**

It might be assumed that all internal parasites likely to be introduced with birds from the UK are already present in New Zealand, in which case no restrictions would be necessary for imported budgerigars.

#### **Option 2.**

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

#### **Option 3.**

Quarantine, treatment, testing.

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

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

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## 25. External Parasites

### 25.1. HAZARD IDENTIFICATION

#### 25.1.1. Aetiological agent

The parasites considered in this section are:

- Mites, including feather mites and nasal mites
- Fleas
- Lice
- Ticks
- Louse Flies

#### 25.1.2. OIE list

Avian mites, fleas, ticks, lice or louse flies are not listed.

#### 25.1.3. New Zealand status

Seven species of mites are included in the register of unwanted organisms. A literature search has failed to reveal reports of any of these mites from birds. Nineteen species of mite, nine species of nasal mites and 82 species of feather mites have been reported in New Zealand (Bishop and Heath 1998). These include eight feather mites, 7 fleas, one hippoboscid and one tick species on psittacines other than budgerigars, and 3 mite species on budgerigars (Bishop and Heath 1998).

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

Twelve species of tick have been reported from birds in New Zealand (Bishop and Heath 1998). Nine *Ixodes* species and *Ornithodoros capensis* have come almost exclusively from aquatic birds (mainly marine birds). The exceptions are *Ixodes anatis* and *Ixodes eudyptis* on North Island brown kiwi, and *Ixodes auritulus* group ticks from western weka and South Island kaka, and *Haemaphysalis longicornis* from the domestic fowl (Bishop and Heath 1998).

Bishop and Heath (1998) have not listed any species of bird lice as occurring in New Zealand, but it is likely that some species are present.

#### 25.1.4. Epidemiology

In a survey of feather diseases in exhibition budgerigars in the UK, *Dubininia melopsittica* and *Protolichus lunula* were identified as being common (Baker 1996). The latter was not associated with any pathology, but heavy infestations of the former were associated with feather abnormalities in 18% of the 198 birds examined. Other species of mite were apparent but were not identified. *Dermanyssus gallinae*, *Cnemidocoptes* spp and a “considerable number of species of feather mites” was reported as occurring in UK budgerigars and lice have been reported to be a common problem (Binks 2006)..

Data on the effects of ticks on psittacine aviary birds in the UK (Forbes and Simpson 1993; Knott 1993) contains a single record of *Ixodes ricinus* on an Australian king parakeet (*Alisteris* spp.). Generally parasitism of birds by small numbers of ticks does not significantly affect their health unless the ticks are vectors of disease (Forbes and Simpson 1993; Knott 1993).

There are hundreds of different species of mites and lice that infest birds and they cannot be considered individually in this risk analysis. However, many are species specific or infest a limited number of bird species and general principles and treatments are used for controlling or eliminating these parasites.

#### 25.1.5. Hazard identification conclusion

Large numbers of external parasites of birds, some of which have not been identified, occur in the UK. Therefore external parasites are considered to be a potential hazard in the commodity.

## 25.2. RISK ASSESSMENT

### 25.2.1. Entry assessment

Various species of feather mites, lice and possibly ticks are present in the UK and could be imported on budgerigars. Therefore the likelihood of entry is non-negligible.

### 25.2.2. Exposure assessment

If introduced into New Zealand parasites could establish in New Zealand budgerigar studs and be spread by contact during shows and transfers of birds from one aviary to another. Transmission to wild psittacines by contact with escaped budgerigars is unlikely because budgerigars do not survive long outside heated accommodation. However, parasites could be transmitted to the environment when eggs or other life stages of parasites are dumped after cleaning budgerigar's cages. Therefore the likelihood of exposure is non-negligible.

### 25.2.3. Consequence assessment

Feather mites have little economic impact in the poultry industry, but the damage to feathers, resulting from some species of feather mites, is of concern in cage and aviary birds, particularly for owners of show-birds (Greve 1996). Reports of feather mites having negative effects on survival or breeding capabilities of birds, including budgerigars have not been found. The impacts of feather mites and lice on the general health and well-being of show and pet birds are hard to measure but are considered to be non-negligible. Since parasites of birds are unlikely to parasitise humans the effects of introducing bird parasites on people is considered to be negligible. It is unlikely that parasites introduced by budgerigars would infest and become established on wild feral birds but the consequences cannot be predicted with certainty.

### 25.2.4. Risk estimation

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

## 25.3. RISK MANAGEMENT

### 25.3.1. Options.

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

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

Available options in order of ascending stringency are:

#### **Options 1.**

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

#### **Option 2.**

Quarantine, treatment, testing.

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

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