Import Risk Analysis: Psittacine Hatching Eggs

FINAL

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Policy and Risk
MAF Biosecurity New Zealand

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Approved for general release

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Executive Summary

The potential risks to New Zealand's native parrot populations has for many years motivated MAF to adopt a very cautious approach regarding the importation of birds in general and psittacine birds in particular. For at least the past decade there has been no way to import these birds legally, either as live birds or as hatching eggs. This situation has been recognised as one that has encouraged smuggling of these valuable species.

Recognising that the biosecurity risks associated with eggs are considerably lower than with live birds, in 2006 MAF completed an import risk analysis on hatching eggs of passerine birds. Building on the experience gained during that project, this risk analysis considers the biosecurity risks associated with the importation of hatching eggs of birds in the order Psittaciformes. In carrying out this analysis, MAF has made an assumption that allowing imports through a legal and therefore controlled process may provide a greater level of protection overall to New Zealand's endangered species than by maintaining the current ban.

From a preliminary list of organisms considered to be potentially associated with Psittacine birds, those that were considered to require further consideration were identified and subjected to individual risk assessments.

As a result of the individual risk assessments, it was concluded that the following organisms should be regarded as hazards in the commodity:

- Avian Influenza
- Avian paramyxoviruses
- Reovirus
- Proventricular dilatation disease virus
- *Salmonella* Gallinarum-Pullorum, *Salmonella* Typhimurium DT104, and *Salmonella* Enteritidis DT4

Options for managing the risk posed by each of these hazards are discussed.

1. Introduction

This risk analysis examines the biosecurity risks posed by the importation of hatching eggs of birds in the taxonomic Order Psittaciformes.

1.1. COMMODITY DEFINITION

The commodity is defined as hatching eggs of any species of the Order Psittaciformes. The eggs must be clean (free of faeces) when collected, unwashed, and have intact (uncracked) shells. Following collection the eggs must be disinfected using sanitation procedures consistent with those in the OIE *Terrestrial Animal Health Code* (the *Code*) (OIE 2008).

While the commodity definition and the contents of this risk analysis consider the entire Order Psittaciformes, this does not subvert the requirement for approval by the Environmental Risk Management Authority (ERMA) of the importation of any "new organism", as defined in the Hazardous Substances and New Organisms Act 1996 (HSNO). Maintaining this breadth of the commodity definition ensures that all potential hazards identified in psittacines are considered in the risk analysis. This should ensure a more conservative assessment of potential hazards than might be the case if efforts were made to narrow the commodity definition.

1.2. BACKGROUND

The background to this risk analysis is a situation largely unchanged from that described in a *Surveillance* article, which refers to very tight constraints on importation of psittacines and a review of policies relating to the importation of eggs and birds (Smits 1995). The frustration of aviculturists wishing to import new species, or genetic lines of species already present in New Zealand, is referred to along with the encouragement that this has provided for smuggling. The difficulties in ensuring that diseases will not enter New Zealand with legal importations and the need to accept that "zero risk" is not an attainable standard are commented on. It is proposed that illegal importations pose a greater risk than importations through legal channels with biosecurity measures in place to minimise the likelihood of diseases entering the country with those importations. Although 13 years have passed since the article by Smits was published, the principles espoused at that time remain valid. Demands by aviculturists continue and the temptation to smuggle birds or eggs remains. While detection of illegal importation of psittacine genetic material (birds or eggs) is not common, specific examples include smuggling of parrots from a quarantine facility in 1997 after detection of Pachecos'disease (Thornton and Stanislawek 2003) and successful prosecution of a person carrying parrot eggs into New Zealand in 2007 (Anonymous 2007c).

Over recent years there have been some improvements in disease recognition, diagnostic methods and differentiation of strains of some organisms. Knowledge of diseases of psittacine birds, however, remains incomplete and surveillance for diseases in psittacine birds (especially caged birds) in New Zealand remains poor. The commodity definition for this risk analysis is restricted to eggs because the biosecurity risks associated with eggs are far fewer than those associated with live birds and importation of psittacine hatching eggs is considered a practical means of meeting the demands by aviculturists for new genetic material.

The Order Psittaciformes includes those birds commonly grouped as parrots but also includes budgerigars, cockatiels, lovebirds, parakeets, conures, caiques, lorries, lorikeets, pionus, eclectus, African greys, amazons, cockatoos, and macaws. Psittacine birds are regarded as attractive to zoos and collectors and there has been extensive international trade in many of

these species. There are numerous examples of disease arising in psittacines during or shortly after transport. New Zealand's endemic fauna includes a number of psittacine birds, including several for which the population is considered to be under threat. Classifications by the International Union of Conservation (IUCN) are listed below.

Common name	Scientific name	IUCN Classification
Yellow-crowned parakeet	Cyanoramphus auriceps	Near threatened
Forbes parakeet	C. forbesi	Endangered
Orange-fronted parakeet / Malherbe's parakeet	C. malherbe	Critically endangered
Red-fronted parakeet	C. novaezelandiae	Vulnerable
Kaka	Nestor meridionalis	Endangered
Kea	N. notabilis	Vulnerable
Kakapo	Strigops habroptila	Critically endangered

New Zealand places a very high value on the preservation of endemic psittacine species with some (especially kakapo, kea, and kaka) having iconic status and assurances that any importations do not threaten endemic species must have high priority.

Historically, large numbers of birds, previously exotic to New Zealand, have been imported with little or no consideration of biosecurity issues. Importations of psittacine species now established as free-living populations are sulphur crested cockatoos (*Cacatua galerita*), galahs (*Cacatua roseicapilla*), and eastern rosellas (*Platycercus eximius*) (Heather and Robertson 1996). These species were introduced in the early 1900s. In addition, there is a wide range of psittacine species present in captivity in New Zealand. Some psittacine birds in captivity have a high commercial value with individual birds having values in excess of $2,000 and breeding pairs of some species priced in excess of $10,000. An active export trade exists (Anonymous 2007b). Many of these species were imported prior to the recognition of most of the diseases covered in this risk analysis. It is likely that a high proportion of potential hazards that could reasonably be expected to have been imported from Europe and Australia entered New Zealand with the importations that have taken place over the past 150 years. It is probable that the relatively low level of disease surveillance allows a number of these diseases to remain undetected but, for the purposes of this risk analysis, in the absence of their diagnosis they have been regarded as not present in New Zealand.

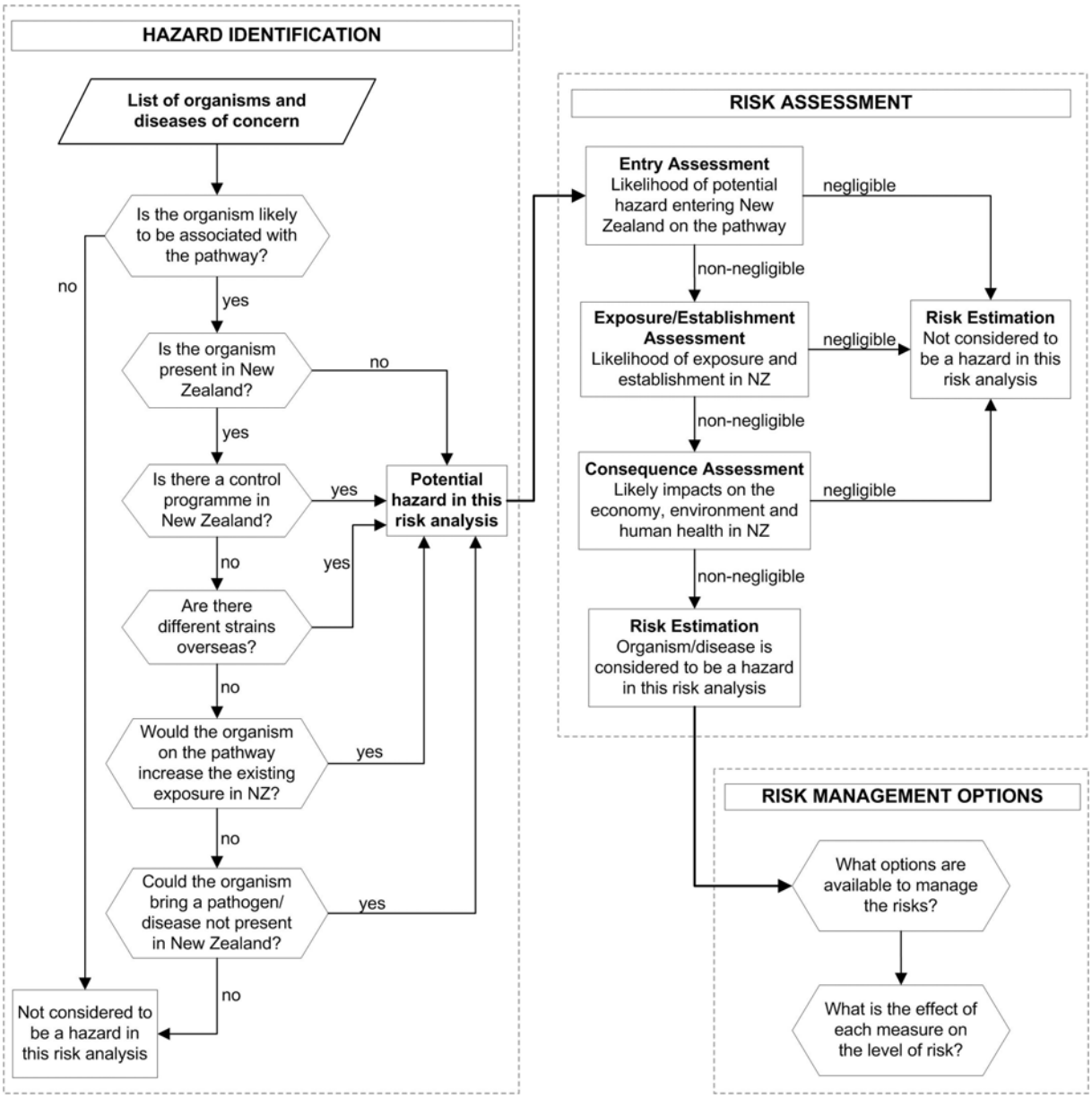
There have been very few evaluations of the diseases or potentially pathogenic organisms carried by bird eggs, other than those of poultry. Much relevant avian disease information comes from poultry species and/or sporadic case reports and/or from local and regional surveys.

Many of the organisms considered in this risk analysis commonly infect birds without causing disease. On occasions, however, they may be associated with incidents of disease. Examples of this include avian influenza viruses, paramyxoviruses, herpesviruses, adenoviruses, poxviruses, polyomaviruses, alphaviruses, bunyaviruses, and *Salmonella* spp. Surveillance for many of these organisms in New Zealand is relatively insensitive so that the lack of recognition of these organisms does not provide a basis for confidence that they are not present. In New Zealand, surveillance information on diseases in psittacine species comes mainly from passive surveillance (i.e. reports of incidents of disease sufficiently pronounced to attract attention and to encourage investment in professional examinations and laboratory investigations) and it is likely that organisms causing sub-clinical disease, or only occasional clinical disease, may remain undiagnosed

1.3. METHODOLOGY

The methodology used in this risk analysis is described in MAF Biosecurity New Zealand's *Risk Analysis Procedures – Version 1*[1] and is consistent with the guidelines in the *Code*. The risk analysis process used by the MAF is summarised in Figure 1.

Figure 1. The risk analysis process.



[1] See: www.biosecurity.govt.nz/

2. Preliminary hazard list

The hazard identification process begins with the collation of a list of organisms likely to be associated with the commodity. Tables 1 and 2 show these organisms, together with some of the key information considered. This list was compiled from those contagious diseases of psittacine birds identified from standard textbooks covering diseases of poultry and caged birds, including psittacines (Harrison and Lighfoot 2006; Ritchie et al 1994; Rosskopf and Woerpel 1996; Saif 2003), literature reviews, and from searches of the international scientific literature including extensive use of electronic databases. Some organisms were included on the basis of initial uncertainty as to whether they might infect psittacine species.

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

Table 1. Organisms considered in this risk analysis

Organism / Disease	Reported from psittacine birds	Associated with disease in psittacines	Associated with disease in other Orders	Recognised as present in New Zealand	Strains of different virulence overseas[2]	Requires further consideration
Viruses						
Avian influenza virus	Yes	Yes	Yes	Yes	Yes	Yes
Avian para-myxoviruses	Yes	Yes	Yes	Yes	Yes	Yes
Pneumovirus	No	No	Yes	No	N.A.	No
Four types of avian pneumovirus (APV) have been identified. Subgroups A and B are the common types in Europe. United States isolates have been shown to have significantly different genetic make-up from the European subgroups and have been classified as subgroup C. Two strains isolated in France were classified as subgroup D (Njenga et al 2003). No reports of pneumovirus in psittacines have been found.						
Psittacine herpesviruses	Yes	Yes	Yes	No	N.A.	Yes
Coronavirus	Yes	Yes	Yes	Yes/No	Yes	Yes

[2] More virulent exotic strains are recognised where either strain typing of New Zealand isolates allows differentiation from more pathogenic types recognised in other countries or where descriptions of the disease in New Zealand allow it to be recognised as less virulent than disease episodes in other countries. Where host-specific strains are recognised overseas but not in NZ, these are treated as "more virulent" in the compilation of this table.

N.A. - Not applicable because assessment of strain variations is not relevant to this process when the organism is not recognised as present in New Zealand.

Organism / Disease	Reported from psittacine birds	Associated with disease in psittacines	Associated with disease in other Orders	Recognised as present in New Zealand	Strains of different virulence overseas[2]	Requires further consideration
Adenovirus	Yes	Yes	Yes	Yes	Uncertain	Yes
Avipoxvirus	Yes	Yes	Uncertain	No	N.A.	Yes
Gyrovirus (chicken infectious anaemia)	No	No	Yes	Yes	No	No
Chicken infectious anaemia virus is established and widespread in New Zealand (Poland 2004; Stanislawek and Howell 1994) and infects only chickens.						
Psittacine beak and feather disease	Yes	Yes	No	Yes	Uncertain	Yes
Infectious bursal disease	No	No	Yes	No	Yes	No
IBD was detected in New Zealand in 1993 in a form less contagious and less persistent in the environment than classical forms (Christensen 1994). A national control programme in commercial flocks (Motha 1996) has lead to there being no sero-positive results since 1999 . IBD has, generally been considered to be a virus of chickens although, outside New Zealand, there have been isolations from other species. Serological testing of 54 psittacine birds (20 captive and 34 free-ranging) in Bolivia for IBD returned negative results (Deem et al 2005). No reports of IBD in psittacines have been discovered.						
Polyomavirus	Yes	Yes	Yes	Yes	No	No
A survey provided positive serological evidence of the presence of avian polyomavirus in two umbrella cockatoos, one sun conure, and one sulphur crested cockatoo in New Zealand (Jakob-Hoff 2003). These findings follow prior speculation that avian polyomavirus was present in this country (Jackson et al 1999; Smits et al 1999), probably as a result of introduction with birds from Australia. Analysis of the genotypes of 20 isolates of Polyomavirus from various locations, times and bird species led Phalen (1999) to conclude that species-specific types had not developed.						
Papillomavirus	Yes	Yes	No?	No	N.A.	Yes
Parvoviruses	Yes?	No	Yes	No	N.A.	No
Both chicken anaemia and psittacine beak and feather disease have been associated with parvovirus-like organisms but their causative organisms are now classified as circoviruses. Other than the early reports of psittacine beak and feather disease, the only reference located suggesting infection of psittacines with parvovirus is one reporting the detection of parvovirus-like particles in the nuclei of hepatic cells (Weissenbock and Fuchs 1995). There was no association between these findings and disease. Goose parvovirus infection (Derzsy's disease) is not recognised as infecting species other than geese, Muscovy ducks, and some hybrid breeds (Gough 2003).						
Alphaviruses	Yes?	No	Yes	Yes / No	Yes?	Yes
West Nile virus	Yes	No?	Yes	No	N.A.	Yes
Japanese encephalitis virus (JEV)	No	No	Yes	No	N.A.	No
JEV, or antibodies to it, has been identified in a number of bird species but no reports of positive virology or serology in psittacines have been discovered. JEV is an important zoonosis, also affecting horses and, to a lesser extent, pigs. Herons and egrets are recognised as carrying the virus and acting as reservoirs for the virus. *Culex* mosquitoes play a major role in virus transmission (Shope 1998).						

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Louping ill	No	No	Yes	No	N.A.	No
No reports of Louping ill virus in psittacines have been located. It is predominantly a disease of sheep, and is transmitted by the tick *Ixodes ricinus*. Red grouse are commonly affected with high mortality rates (Reid 1974; Timoney 1972) and other species of grouse have been infected experimentally.						
Rotavirus	Yes	No	Yes	No (not from psittacines)	Yes	Yes
Orbivirus	Yes	Yes	Yes	No	N.A.	Yes
Reovirus	Yes	Yes	?	No	N.A.	Yes
Bunyaviruses	No	No	Yes	No	N.A.	No
Although the literature contains reference to evidence of infection of birds with Bunyaviruses, no reports associating these viruses with avian disease or with psittacines have been located. With the exception of Hantavirus, Bunyaviruses are transmitted by insects.						
Bornavirus	No	No	Yes	No	N.A.	No
Bornavirus has been reported from ostriches, mallard ducks, and jackdaws (Berg et al 2001; Cooper et al 2004) but reports of infections in psittacines have not been found. Borna disease affects horses, sheep and other mammals in Europe, North America, and parts of Asia (Hatalski et al 1997; Richt 2001). An unrelated avian Bornavirus may be the agent of proventricular dilatation disease, which is considered further in this document.						
Avian encephalo-myelitis virus	No	No	Yes	Yes	No	No
No reports associating AEV with psittacines have been located. Avian encephalomyelitis was recognised in New Zealand prior to 1972 when satisfactory use of a vaccine was reported (Anonymous 1972). Vaccination since then contributes to a high percentage of chickens being serologically positive (Poland 2004).						
Duck hepatitis virus	No	No	Yes	No	N.A.	No
No reports of infection of Psittacines with duck hepatitis virus have been located. Ducks are the only species susceptible to natural infections with duck hepatitis virus.						
Astroviruses	No	No	Yes	No	Yes	No
Searches of the literature have failed to reveal reports of astroviruses in psittacines. A number of agents previously named as Picornavirus, picornavirus-like, enterovirus, or enterovirus-like are now classified, or thought likely to become classified, as astroviruses (Koci 2002). Amongst these are avian nephritis virus (Imanda et al 2000), duck hepatitis type 2 virus (Gough 1986) (both previously classified as picornaviruses), and turkey enterovirus-like agent (Guy 2004). Astroviruses isolated from different species are antigenically distinct and are species-specific (Quinn et al 2002d).						
Proventricular dilatation disease (Macaw wasting disease)	Yes	Yes	No	No	Uncertain	Yes
Avian retroviruses	Yes	Yes	Yes	Yes	No	No
Retroviruses have been proposed as possible causal agents of renal tumours of budgerigars (Gould et al 1993; Neumann and Kummerfeld 1983; Simova-Curd et al 2006). In New Zealand, a hemopoietic neoplasm resembling myeloblastosis has been reported from an incident of ill-thrift and deaths in several budgerigars, the progeny of imported parents. This was attributed to an avian leucosis virus (Anonymous 1999b). Vickers (1991) reported investigations of psittacine						

Organism / Disease	Reported from psittacine birds	Associated with disease in psittacines	Associated with disease in other Orders	Recognised as present in New Zealand	Strains of different virulence overseas[2]	Requires further consideration

erythroblastosis in New Zealand parakeets. This disease behaved as a contagious disease, with high morbidity and mortality. Results of testing suggested that the cause was not one of the avian leucosis-sarcoma complex.

Bacteria

Chlamydophila spp.	Yes	Yes	Yes	Yes	Yes?	Yes

Salmonella spp.	Yes	Yes	Yes	Yes	Yes	Yes

Escherichia coli	Yes	Yes	Yes	Yes	No	No

E. coli are members of the Enterobacteriaceae and are common inhabitants of the intestinal tract of many animals throughout the world. Psittacines are not universally infected with *E. coli;* it was recovered from less than 10% of clinically healthy birds in captivity (Bangert et al 1988a) and in 60% of cockatoos but only 18% of non-*Cacatua* psittacines (Flammer and Drewes 1988). Diet, especially grain intake, may be a factor influencing the prevalence of *E. coli* in the gut of birds (Glunder 2002).

Although specific records of avian pathogenic *E. coli* (APEC) have not been been located, *E.coli*-induced diseases of poultry have been reported in New Zealand (Black 1997).

Verotoxin producing *E. coli* are present in New Zealand and are considered one of the more important causes of enteric disease in humans (Baler et al 1999).

Campylobacter spp.	Yes	No	Yes	Yes	No	No

Reports indicate that the carriage rate of *Campylobacter* spp. in psittacines is very low (Oyarzabal et al 1995; Scope et al 1998; Yogasundram et al 1989) and records associating *Campylobacter* spp. with disease in psittacines have not been located. Both *Campylobacter coli* and *C. jejuni* are present in New Zealand. Campylobacteriosis is the most commonly reported notifiable disease of humans in New Zealand. Poultry products are commonly claimed a major source of human *Campylobacter* infections.

Yersinia spp.	Yes	Yes	Yes	Yes	No	No

Y. enterocolitica and *Y. pseudotuberculosis* are pathogens affecting humans, other mammals, and a range of other animals. Other *Yersinia* species (*Y. frederiksenii, Y. intermedia, Y. kristensenii, Y. rohdei, Y. bercovieri, Y. philomeragia*) are commensals or saprophytes (Wanger 1998) although Quan (Quan 1998) recognises this latter group as occasional opportunist pathogens. Approximately 50 serotypes of *Y. enterocolitica* are recognised but only a small proportion of them are pathogenic. The serotypes that predominate in human illness are O:3, O:8, O:9 and O:5,27. Six serotypes (I to VI) of *Y. pseudotuberculosis* are recognised, each containing pathogenic strains (Weagabt et al 2001).

Both *Yersinia enterocolitica* and *Y. pseudotuberculosis* have been reported from psittacines. *Y. pseudotuberculosis* is the species more commonly associated with disease (Dorrestein et al 1985; Sanekata et al 1991) although mortalities in budgerigars in the UK have been attributed to *Y. enterocolitica* (Dorrestein et al 1985). *Yersinia pseudotuberculosis* has been associated with disease in psittacines in New Zealand (a kaka (*Nestor meriondalis*), one rainbow lorikeet (*Trichoglossus mollucanus*), and one budgerigar (*Melopsittacus undulatus*)) (Cork et al 1999).

In New Zealand, *Y. enterocolitica* serotypes O:2,3, O:3, O:5, O:6,30, O:5,27 and O:9 have been reported, variously, from humans, pigs, dogs, and/or other domestic animals (Fenwick 1997; Gill 1996; Hussein et al 2003). *Y. enterocolitica* biotype 4 has been reported from pigs (Gill 1996), biotype 5 from sheep (Gill 1996), biotypes 1, 2, 3 and 5 from deer (Henderson 1984), and biotype 1a and untypable strains from birds (Cork et al 1995). *Y. pseudotuberculosis* serotypes I, II and III have been reported from livestock, rabbits, guinea pigs, and aviary birds (Hodges et al 1984), and from two of 1370 avian samples from wild birds. *Y. intermedia*, *Y. frederiksenii,* and *Y. kristensenii* have been reported from mammalian and avian sources (Bullians 1987; Cork et al 1995; Henderson 1984).

Klebsiella spp.	Yes	Yes	Yes	Yes	No	No

These organisms are normal inhabitants of the bowel and respiratory tract of humans and other animals (Homes and Aucken 1998). They are also commonly found in soil and water (Homes and Aucken 1998). *Klebsiella pneumoniae* infection has been associated with disease in humans and with mastitis and metritis in cattle, metritis in sows, and pneumonia in foals and primates (Linton and Hinton 1998).

There are few reports of *Klebsiella* spp. in psittacines. Glunder reported that it was very difficult to colonise the intestine of budgerigars with *Klebsiella* spp. and concluded that the presence of either *Klebsiella* or *E. coli* in budgerigars might be

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indicative of poor husbandry (Glunder 2002; Glunder and Martinsen 1981). In New Zealand *Klebsiella* spp. have been reported from cases of bovine mastitis (Anonymous 1976), from omphalitis/peritonitis in Ostrich (Cooke 1998), and a case of pneumonia in a budgerigar (Anonymous 1975a).

Proteus spp.	Yes	No	Yes	Yes	No	No

*Proteu*s spp. are associated with a wide range of clinical conditions in animals. There are very few reports of *Proteus* spp. being isolated from Psittacines and none of them associated the infection with disease.
In New Zealand *Proteus* spp. have been reported from urinary infection in a dog with a urethral defect (Goulden 1969), pooled samples of bovine milk (Elliot et al 1976), cases of bovine mastitis (Orr 1995), suppurative lesions in slaughtered cattle (Elliot 1969), and milk from goats with high somatic cell counts (McDougall 1999). *Proteus* spp. was reported as a predominant component of a mixed bacterial infection in a case of sinusitis in an Antipodes Island parakeet (*Cynaoramphus unicolor*) although it was not suggested that the bacterium had any specific aetiological role (Gartrell et al 2003).

Serratia spp.	Yes	Yes	Yes	Yes	No	No

Serratia spp. have been reported from parrots in association with aspergillosis (Simpson and Euden 1991) and in a single case of disease in a macaw (Quesenberry and Short 1983). In New Zealand there is evidence of *Serratia marcescens* contributing to poor hatchability and poor survivability in juvenile budgerigars (Christensen 2005).
Serratia spp. are found in soil and water with some isolations coming from animals. *S. marcescens* is implicated as an occasional opportunist pathogen in hospital patients (Homes and Aucken 1998). *S. marcescens* has been diagnosed as the cause of mastitis in cattle (Guardo et al 1997; Linton and Hinton 1998). It has also been isolated from other cases of diseased animals including abscesses in ewes (Al-Dughaym 2004), a case of equine abortion (Jores et al 2004), horses with respiratory disease (Kester et al 1993) and a case of equine myocarditis (Ewart et al 1992). From New Zealand, there are reports of the isolation of *Serratia marcescens* from a case of chronic bronchopneumonia in a cat (Anonymous 1974b) and *Serratia* sp. from 11 cases of bovine mastitis (Anonymous 1976).

Morganella spp.	No	No	Yes	Yes	No	No

This organism has been isolated from faeces of healthy (Bangert et al 1988b) and diseased (Tanaka et al 1995) birds but no reports of *Morganella* spp. in psittacines have been discovered. *M. morganii* is common in the intestinal tract and faeces of humans, other mammalian species, and reptiles (Homes and Aucken 1998; Jones 1998). In New Zealand *M. morganii* has been reported from a foal that died at two weeks old (Anonymous 1976).

Enterobacter spp.	Yes	No	Yes	Yes	No	No

Enterobacter spp. have been isolated from the faeces or cloaca of healthy psittacine birds (Bowman and Jacobson 1980; Flammer and Drewes 1988; Glunder 1981) and from dead finches (Prattis et al 1990). In New Zealand, *Enterobacter* spp. have been reported from cases of bovine mastitis (Anonymous 1976) and *E. aerogenes* from the lungs of two cats, each dying after a short febrile illness (Anonymous 1975b).
Enterobacter spp. have been isolated from soil and water and from a wide variety of animals including humans (Jones 1998). *Enterobacter* spp. are a significant cause of nosocomial infections in hospitalised people (Wisplinghoff et al 2004) and *E. sakazakii* can cause serious disease in infants (Iversen and Forsythe 2003). Internationally, *Enterobacter* have been associated with genital infections of mares (Atherton 1975; Atherton and Oerskov 1976) and stallions (*E. aerogenes*) (Atherton and Oerskov 1976), equine abortions (*E. agglomerans*) (Gibson et al 1982), udder infections in cows (*E*. spp.) (McDonald et al 1977), an enteric disorder in a calf (*E. agglomerans*) (Garg 1985), and an inflammatory condition in the skin of sheep (*E. cloacae*) (Jansen and Hayes 1987).

Pasteurella multocida	Yes	Yes	Yes	Yes	No	No

P. multocida is endemic in the New Zealand avian population with documented diagnoses including chronic disease in laying hens (Lohr 1977; Poland 2001), mortalities in turkeys (Anonymous 1990a; Anonymous 1990b), and joint disease in roosters (Anonymous 1999a).
P. multocida was identified during an investigation into mortalities in rockhopper penguins (*Eudyptes chrysocome*) on Campbell Island. The organisms isolated from chicks were classified as capsule serogroup A, somatic serotype 1 which is the main serotype identified in epizootics of avian cholera in wildlife in North America (de Lisle et al 1990).

Organism / Disease	Reported from psittacine birds	Associated with disease in psittacines	Associated with disease in other Orders	Recognised as present in New Zealand	Strains of different virulence overseas[2]	Requires further consideration
Pasteurella gallinarum	No	N.A.	No	No	N.A.	No
Haemophilus paragallinarum	No	N.A.	Yes	No	N.A.	No
Pasteurella-like and *Haemophilus*-like organisms	Yes	Yes	No?	Uncertain	Uncertain	Yes
No reports of either *P. gallinarum* or *H. paragallinarum* infecting psittacines have been located. In New Zealand there is a report of a *Haemophilus* sp. being isolated from turkey poults with respiratory disease (Anonymous 2002) but turkeys are refractory to infection with *H. paragallinarum* (Blackall and Tamamoto 2003). The taxonomy, nomenclature and diagnostic significance of other *Pasteurella*-like and *Haemophilus*-like organisms infecting birds, including psittacines, is not resolved (Bisgaard et al 1999; Christensen et al 2003; Devriese et al 1988; Mouahid et al 1994; Piechulla et al 1985). Some of these organisms are reported as associated with disease but others have been isolated from healthy birds. In New Zealand, the presence or absence of these organisms remains uncertain in the absence of studies of the detailed microbiology of such isolates.						
Riemerella anatipestifer	Yes	No	Yes	Yes	No	No
R. anatipestifer is widely distributed around the world and is recognised, most commonly, when it causes disease in intensively reared ducks. It also causes losses in geese and turkeys. The organism has also been found in pheasants, chickens, guinea fowl, quail, partridges, and other waterfowl (Bisgaard et al 1999; Christensen et al 2003; Devriese et al 1988; Mouahid et al 1994; Piechulla et al 1985; Sandhu 2003). This organism was previously named *Pasteurella anatipestifer* and *Moraxella anatipestifer*. A single report of *R. anatipestifer* in a psittacine has been located, that from a budgerigar of unknown health status (Hinz et al 1998). In New Zealand, an organism tentatively classified as *Pasteurella anatipestifer* was isolated from an incident of disease in ducks involving high mortality and pathology consistent with "new duck disease" in 1974 (Anonymous 1974a). A further case, in which four of 16 ducks died in 1990, was considered consistent with *P. anatipestifer* infection (Orr 1990). Although isolates from these cases were not definitively identified, it appears that *R. anatipestifer* is endemic in New Zealand.						
Ornithobacterium rhinotracheale	No	No	Yes	No	N.A.	No
No reports of *O. rhinotracheale* in psittacines have been discovered. This organism has not been reported from New Zealand and an unknown number of poultry flocks in New Zealand were surveyed for *O. rhinotracheale* using imported ELISA kits with negative results (Christensen 2005). *O. rhinotracheale* is widespread in poultry flocks, outside New Zealand, in the absence of disease and whether it should be regarded as a primary pathogen is doubtful. It can, however, contribute to serious disease incidents (Chin et al 2003; van Empel and Hafez 1999).						
Bordetella avium	Yes	Yes	Yes	No	N.A.	Yes
Mycoplasma spp.	Yes	Yes	Yes?	Yes	Yes	Yes
Mycobacterium spp.	Yes	Yes	Yes	Yes/No	Yes?	Yes

Organism / Disease	Reported from psittacine birds	Associated with disease in psittacines	Associated with disease in other Orders	Recognised as present in New Zealand	Strains of different virulence overseas[2]	Requires further consideration
Francisella tularensis	No	No	Yes	No	N.A.	No

No reports of *F. tularensis* in psittacines have been located. This organism is not known to be present in New Zealand.

Human tularaemia, attributable to *F. tularensis* type B, occurs throughout most of Europe but with marked regional differences and with major differences at different times in history. There is a low level of tularaemia through most of Europe (Tarnvik et al 2004). Past proposals that wild mammals or birds form the reservoir(s) for *F. tularensis* are now questioned. There is epidemiological evidence supporting the proposal that the reservoir for the organism is associated with water (Tarnvik et al 2004). Birds are affected by tularaemia and, commonly, infection results in death. Species most commonly affected are Galliformes species, waterfowl, scavengers, and predatory wild birds (Barnes 2003).

Macrorhabdus ornithogaster (formerly *Megabacterium*)	Yes	No?	Yes	Yes	No	No

Macrorhabdus ornithogaster have been reported from budgerigars and canaries in New Zealand with clinical disease similar to that associated with megabacteriosis in other countries (Anonymous 1999b; Christensen et al 1997; Johnstone and Cork 1993).
Macrorhabosis (megabacteriosis) is predominantly associated with proventriculitis in budgerigars (Quinn et al 2002c) but it has also been found in healthy and diseased birds in a range of species. The epidemiology of megabacteriosis is not known. Scanlon and Graham (Scanlon and Graham 1990) suggested that the organism is part of the normal gut flora of birds. Others consider it to be a pathogen.

Gram positive contaminants (e.g. Staphylococci / Streptococci)	Yes	Yes	Yes	Yes	No	No

Numerous species of *Streptococcus* and *Staphylococcus* are present in New Zealand. Frequently identification of these organisms attracts little attention as they are assigned "secondary", "opportunist", or "contaminant" roles. Typing of *Staphylococcus aureus* beyond coagulase positive or negative is seldom carried out.

Borrelia anserina (Avian spirochaetosis)	Yes	Yes	Yes	No	N.A.	Yes
Borrelia burgdorferi (Lyme Disease)	No	No	Yes	No	N.A.	No

Searches of the scientific literature have failed to identify records of *B. burgdorferi* or Lyme disease in psittacines.
Lyme disease affects dogs, horses, cattle, and humans. These species are incidental hosts to an organism that normally cycles between reservoir hosts (small mammals including rodents, birds, and lizards) and tick vectors. The usual hosts for adult ticks are larger mammals which are maintenance hosts for the ticks but are not reservoir hosts for *Borrelia*. The only competent vectors are ticks, generally of the *Ixodes* genus. In Europe, the main vector is *Ixodes ricinus*, in the eastern United States it is *I. scapularis*, in the western US it is *I. pacificus,* and in Eurasia it is *I. persulcatus* (Barnes 2003).

Brachyspira spp.	No	No	Yes	Yes	No	No

No reports of *Brachyspira* spp. in psittacines have been located. *Brachyspira* (*Serpulina*) *hyodysenteriae*, *B. pilosicoli,* and *B. innocens* have been isolated from pigs with enteric disease in New Zealand (Anonymous 1997; Anonymous 2000). *Brachyspira pilosicoli* is found in the gut of birds and associated with clinical disease. *Brachyspira* (formerly *Serpulina*) species are attracting attention as recently classified inhabitants of the gut of humans, pigs, chickens, and other species which are associated with disease commonly termed intestinal (or colonic) spirochaetosis (Quinn et al 2002b).

Organism / Disease	Reported from psittacine birds	Associated with disease in psittacines	Associated with disease in other Orders	Recognised as present in New Zealand	Strains of different virulence overseas[2]	Requires further consideration
Coxiella burnetii	Yes	Yes	Yes	No	N.A.	Yes
There are two recent reports of *Coxiella* spp. in psittacines (Shivaprasad et al 2008; Woc-Colburn et al 2008). This organism is exotic to New Zealand. *C. burnetii* is the cause of Q Fever, is widely distributed throughout the world and is found in a wide variety of animals and birds. Q fever has been associated with ticks from several genera, however, the role that ticks play in transmission is unclear. The disease seems more likely to be spread by inhaling dust derived from placentas of animals that have aborted.						
Aegyptianella spp.	Yes	Uncertain	Yes	No	N.A.	Yes
Other Rickettsia	Yes	No	Yes/No	Yes/No	Yes/No	No
Only one report of an organism from the genera *Ehrlichia*, *Neorickettsia*, *Rickettsia*, *Anaplasma*, *Eperythrozoon,* or *Haemobartonella* in a psittacine has been located. That was a report of the culture of a rickettsial organism from a parrot (Eb et al 1973). Only the title of the article is available from CAB Abstracts, but there is no reference to the organism coming from a diseased animal.						
Fungi and yeasts	Yes	Yes	Yes	Yes/No	Yes/No	Yes
Internal parasites (Nematodes, cestodes, protozoa)	Yes	Yes	No?	Yes/No	Yes	Yes
External parasites (ticks, mites, lice)	Yes	Yes	Yes	Yes/No	Yes	Yes

Of the organisms identified as requiring further consideration in Table 1, only those that are likely to be transmitted in association with psittacine eggs require further evaluation. The likelihood of transmission of these organisms in psittacine eggs is summarised in Table 2.

Table 2. Organisms considered for their potential to be present in, or on, psittacine eggs.

Organism / Disease	Any evidence of possible transmission through eggs
Avian influenza virus	Yes
Avian paramyxoviruses	Yes
Psittacine herpesviruses	Yes
Coronavirus	No
The coronaviruses of birds include Infectious bronchitis of chickens, turkey coronavirus enteritis, poult enteritis-mortality syndrome, and a respiratory/renal disease of pheasants. No reports of egg-borne transmission of these viruses have been located. The report by Hirai *et al*. of a coronavirus in parrots (Hirai et al 1979a) was subsequently retracted (Hirai et al 1982). Gough *et al*. (Gough et al 2006) reported the identification of a coronavirus from a case of suspect psittacine proventricular dilatation disease in an Amazon parrot (*Amazon viridigenalis cassin*). The relationship of the virus to the disease was considered to be uncertain. No reports indicating that coronaviruses of birds are transmitted through eggs have been located.	
Adenovirus	Yes

Organism / Disease	Any evidence of possible transmission through eggs

Avipoxvirus — No

Psittacinepox virus is recognised as a separate species within the avipoxvirus genus (Buller et al 2005). Fowlpox virus is widespread in New Zealand and pox infections have been diagnosed in a number of other species (Gartrell et al 2003; Howell 1992; Johnstone and Cork 1993; Smits 1995). Avian poxviruses are spread by mechanical transmission and no reports suggesting transmission via eggs have been located.

Psittacine beak and feather disease — Yes

Papillomavirus — No

No reports of Papillomavirus in birds in New Zealand have been located.
Papillomaviruses are, generally, host specific and tissue specific. Lesions in different tissues of the same species are caused by different viral types (Dom et al 1993; Quinn et al 2002e). Papillomaviruses may be latent with no signs of infection until activated during a period of stress. Virus is shed with cells desquamating from the surface of papillomas (Quinn et al 2002e). Internal papillomas are caused by herpesviruses.
Very few reports of characterization of papillomaviruses from birds have been located. Moreno-Lopez et al. (1984) found that individual viruses from chaffinches in Sweden and Holland were closely related but that there was little genetic homology with a bovine papillomavirus. A papillomavirus isolated from an African grey parrot (*Psittacus erithacus timneh*) was found to be distinct from 17 mammalian and one chaffinch viruses tested (O'Banion et al 1992).
No reports suggesting transmission of avian papillomaviruses through eggs have been located.

Alphaviruses — No

Whataroa virus has been identified in southern Westland in the South Island of New Zealand. It infects a number of bird species, particularly passerines (Miles 1973; Miles et al 1971), but there is no reported association with disease. A substantial number of alphaviruses have been identified in birds in various parts of the world. All are transmitted by arthropods, generally mosquitoes. None appear associated with disease in birds but some do cause disease in humans. The equine encephalitis viruses, which also cause disease in humans, are the most important of these.
Serological (but not PCR) evidence of exposure to eastern equine encephalitis was found in four of 56 psittacine birds for which samples were held in the archives at the University of Georgia College of Veterinary Medicine (Gregory et al 1997; Miles 1973; Miles et al 1971) and negative results were found in 54 blue-fronted parrot (*Amazona aestiva*) in Bolivia for serological evidence of exposure to eastern, western, and Venezuelan encephalitis virus (Deem et al 2005).
Searches of the literature have failed to identify reports suggesting egg-borne transmission in birds of any of the alphaviruses.

West Nile virus (WNV) — No

WNV is an arthropod borne flavivirus particularly infecting wet-land birds and transmitted by mosquitoes, ticks, and hippoboscid flies. When first recognised WNV was present in much of Africa, the middle east, and western areas of Asia, and there were occasional incursions into southern Europe. WNV has been identified in a number of mammalian species including humans in which the virus can cause mortality (Hubalek and Halouzka 1999). Disease incidents, particularly affecting horses, have occurred in Israel, Italy, Morocco, and France since 1996. In 1998, WNV caused significant mortalities in migrating birds in Israel (Zeller and Schuffenecker 2004). WNV became evident in the United States in 1999 and spread as a serious epidemic in humans. This epidemic has been marked by significant mortalities in birds, especially American Crows (*Corvus brachyrhynchos*) (Eidson et al 2001a; Eidson et al 2001b). Domestic geese, Canadian geese, chickens, rock dove, and sparrows were amongst other avian species showing serological evidence of infection (Komar et al 2001). A very high degree of homology was found between strains originating from Israel and the New York epidemic (Giladi et al 2001).
There have been relatively few reports of WNV in psittacines. Studies in Madagascar (Fontenille et al 1985; Fontenille et al 1989; Giladi et al 2001; Morvan et al 1990a; Morvan et al 1990b) established that WNV was present in parrots (*Coracopis vasa*), egrets, humans, mosquitoes, and other species. During 2002, WNV was diagnosed in 11 birds from eight species in zoos in Kansas (D'Agostino and Isaza 2004). Affected birds included two psittacines: an African grey parrot (*Psittacus erythacus)* and a thick-billed parrot (*Rhynchopsitta pachyrhncha*).
Searches of the literature have failed to find reports suggesting that WNV might be transmitted through avian eggs.

Rotavirus — Yes?

Organism / Disease	Any evidence of possible transmission through eggs

Orbivirus — No

Orbiviruses are widespread in sea-birds and their associated tick populations. Moss *et al*. commented on the distribution of one sub-group as being from the Arctic to the sub-Antarctic. Reports do not associate these viruses with mortality or disease.

The isolation of viruses classified as Orbiviruses from a cockatiel and a budgerigar in the United States, were reported (Hirai et al 1979b). The authors considered that the viruses were mildly pathogenic in the budgerigars.

No reports suggesting egg borne transmission of Orbiviruses of birds have been located.

Reovirus — Yes

Proventricular dilatation disease / Macaw wasting disease — Uncertain

Chlamydophila spp. — Yes

Salmonella spp. — Yes

Pasteurella-like and *Haemophilus*-like — No

A number of isolates of bacteria fitting within the family Pasteurellaceae, but not within the recognised genera and species, have been isolated from healthy and diseased psittacine birds (Bisgaard et al 1999; Christensen et al 2003; Devriese et al 1988; Hinz et al 1998; Mouahid et al 1994; Piechulla et al 1985). In the absence of classification of these organisms, statements about prevalence must be made with caution and the best guide to epidemiology must be based on that of other members of the family.

It seems likely that isolates of *Pasteurella*-like, *Haemophilus*-like, or *Riemerella*-like organisms are present in New Zealand but their identity remains unclear.

On the basis that no reports suggesting egg-borne transmission of any members of the Pasteurellaceae have been located, it is considered very unlikely that these organisms will be transmitted in this way.

Bordetella avium — No

Bordetella bronchisepticum and *B. parapertussis* are endemic in New Zealand. *B. avium* has not been identified in New Zealand.

B. avium has been identified as a cause of, or contributor to, disease in turkeys in North America and Europe. It is also an opportunist pathogen in chickens. The organism is readily transmitted between birds and can survive in litter for up to six months (Jackwood and Saif 2003; Quinn et al 2002a).

Reports of *B. avium* in psittacines come from parrot finches (*Erythrura psittacea*) and a yellow crested cockatoo (*Kakatoe galleria*) in Germany (Hinz and Glünder 1985), nestling cockatiels and other psittacines in the same establishment in Florida (Clubb et al 1994), and a blue and yellow macaw (*Ara ararauna*) in the eastern United States (Raffel et al 2002).

No reports suggesting that *Bordetella avium* might be transmitted through eggs have been located.

Mycoplasma spp. — Yes

Mycobacterium spp. — Yes

Borrelia anserina — No

Borrelia anserina is the cause of avian spirochaetosis, an acute disease of chickens, turkeys, pheasants, geese, and ducks. It is reliant on *Argas* spp. ticks as vectors (Barnes 2003). One report of Borreliosis in a psittacine has been located, in a grey parrot (*Psittacus erithacus*) (Ehrsam 1977).

Searches of the literature have not revealed suggestions that *B. anserina* might be transmitted through, or on, the eggs of any avian species.

Organism / Disease	Any evidence of possible transmission through eggs

Coxiella burnetii — No

Neither of the recent reports of *Coxiella* spp. infection of psittacines have described infection or pathology of the ovary or oviduct (Shivaprasad et al 2008; Woc-Colburn et al 2008).

Soběslavskỳ and Syrůček (1959) concluded that transovular transmission of coxiellae in domestic fowl is either not the general rule, or that the number and activity of the infective agents released into the egg are so low as to be incapable of causing fresh infection. A more recent study failed to demonstrate egg transmission of *C. burnetii* in experimentally-infected hens (Sethi et al 1978).

Aegyptianella spp. — No

A. pullorum, *A. botuliformis,* and an unidentified *Aegyptianella* sp. have been reported from birds.

A. pullorum is recognised in Africa, Asia, and southern Europe and has been reported from chickens, geese, ducks, quail, and ostrich. The recognised vectors are ticks of the Argas genus. The only report located of an *Aegyptianella* spp. being found in psittacines is from a single *Amazona aestiva* examined at the time of importation into Great Britain (Barnes and Nolan 2008).

No reports suggesting that *Aegyptianella* spp. might be transmitted through, or on, eggs of birds have been discovered.

Fungi and Yeasts — No

A number of fungi have been isolated from healthy and diseased psittacines. New Zealand records have been checked for these fungi and the only fungus/yeast discovered as reported in psittacines and for which no record of recognition in New Zealand could be found is *Candida solani* in the crop of a quaker parrot (*Myopsittacus*) in Argentina (Menchaca et al 1967). This is a rarely reported species of *Candida* but the general habitat of *Candida* spp. is as a saprophyte on leaves and flowers, and in water and soil (Anonymous 2007a).
No reports suggesting that fungi or yeasts might be transmitted through the eggs of any avian species have been located.

Internal parasites — No

This section covers nematodes, cestodes, trematodes, and protozoal parasites. The scientific literature contains many reports of genera and species of internal parasites of birds, including psittacines, which have not been recorded in New Zealand. Some of these parasites have direct lifecycles. The lifecycles of others may involve one, or more, intermediate hosts. They are of varying pathogenicity.
No reports suggesting that these parasites might be transmitted through the eggs of any avian species have been located.

External parasites — No

This section covers ticks, lice, and other arthropod ectoparasites. The scientific literature contains many reports of genera and species of external parasites of birds, including psittacines, which have not been recorded in New Zealand. Some of these parasites have direct lifecycles. The lifecycles of others may involve one, or more, intermediate hosts. They are of varying pathogenicity.
No reports suggesting that these parasites might be vertically transmitted via avian eggs have been located.

From the above, the following organisms/diseases are considered to require further evaluation in this risk analysis:

- Avian influenza virus
- Avian paramyxoviruses
- Psittacine herpesviruses
- Psittacine adenoviruses
- Psittacine beak and feather disease
- Rotavirus
- Reovirus
- Proventricular dilatation disease / Macaw wasting disease

- Chlamydophila spp.
- Salmonellae
- Mycoplasma spp.
- Mycobacterium spp.

2.1.1. Risk analysis for the importation of psittacine eggs

For each of organisms/diseases identified as requiring further evaluation, the epidemiology is discussed, including a consideration of the following questions:

1) whether eggs from psittacine birds could act as a vehicle for the introduction of the organism?
2) if the organism requires a vector, whether competent vectors might be 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,
    a. 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
    b. 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 two 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.

In line with the MAF Biosecurity New Zealand and OIE risk analysis methodologies, for each potential hazard 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.

In assessing the likelihood of exposure to wild birds, caged or aviary birds or poultry in New Zealand, an assumption is made that there is potential for contact between caged or aviary birds and those outside that environment. Such contact might be direct through the walls of enclosures, indirect through transfer of fomites, movement of rodents, insects, or other animals or through escape or release of the imported birds.

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

Knowledge on the epidemiology of a number of organisms within psittacine hosts is limited. For this reason, information on the epidemiology of those organisms, or organisms closely related, in other avian species is used as a basis for determining entry, exposure and/or consequence assessments.

2.1.2. Risk management

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

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

2.1.3. Risk communication

Draft import risk analyses are 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 the draft document, a review of submissions is produced and a decision-making committee determines whether any changes need to be made to the draft risk analysis.

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

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ORGANISM RISK ANALYSES

3. Avian influenza

3.1. HAZARD IDENTIFICATION

3.1.1. Aetiological agent

Avian Influenza (AI) viruses are Influenza A viruses within the family Orthomyxoviridae. These viruses are characterised by antigenic surface glycoprotein haemagglutinin (types H1 – 16) and neuraminidase (N1 – 9) (Spackman 2008). The H and N antigens may be present in any combination (Hx, Ny). Strains of AI are commonly separated into highly pathogenic strains (HPAI) and low pathogenic strains (LPAI) on the basis of their pathogenicity in poultry. All HPAI virus isolates have been subtypes H5 or H7 but not all H5 or H7 isolates have been highly pathogenic. For statutory purposes, the main basis for differentiation on HPAI and LPAI strains has been pathogenicity in susceptible chickens (Alexander 2008; OIE 2008).

3.1.2. OIE list

Notifiable Avian Influenza (NAI) viruses are on the OIE list. NAI refers to any avian influenza virus of H5 or H7 subtypes or any AI virus with pathogenicity above limits set in the *Code* (OIE 2008).

3.1.3. New Zealand status

Avian influenza H5 and H7 are listed as notifiable organisms in the unwanted organisms register.

AI viruses have been isolated from healthy wild mallard ducks in New Zealand (Austin and Hinshaw 1984; Stanislawek 1990; Stanislawek 1992; Stanislawek et al 2002). Subtypes identified have included H4N6, H1N3 and H5N2. The H5N2 isolates were shown to be non-pathogenic (Stanislawek et al 2002).

More recent studies of wild waterfowl have recovered H1, H2, H4, H5, H7, H10 and H11 subtypes of AI in New Zealand (Stanislawek 2008; Tana et al 2007). In 2008 a H5N1 virus was isolated from mallards.
However, this isolate is a low pathogenicity strain (Stanislawek 2008).

3.1.4. Epidemiology

The main natural hosts of AI viruses are birds in the orders Anseriformes (ducks, swans, and geese) and Charadiformes (shorebirds, gulls, and terns). Maintenance of viruses in these populations is aided by water becoming heavily contaminated during periods of congregation (Alexander et al 2003; Stallknecht and Shane 1988; Suarez 2000). Surveillance studies have found infection rates in passerine birds to be comparable with those in Charadiformes (Capua et al 2005) and it has been suggested that passerines might also be reservoir hosts for some subtypes of AI (Ibrahim et al 1990; Panigrahy et al 2003; Papparella et al 1993). These natural hosts act as sources of infection for other species.

Virulent H5 and H7 strains derive from low pathogenic H5 and H7 strains. Mutations arise following transfer of infection from the wild host to poultry and the ability of these mutated viruses to infect multiple tissues results in their high pathogenicity. The propensity for AI

strains to mutate is illustrated by the genetic diversity of isolates within local epidemics (Sims et al 2003).

Swayne and Halvorson (2003) found no evidence for transovarial transmission of AI. Both Lu et al. (2004) and Swayne and Beck (2004) found no evidence of LPAI in eggs of infected birds and a report to the European Food Safety Authority in 2005 (Capua et al 2005) found no evidence of natural infections of poultry with LPAI resulting in infection of the content of eggs. HPAI virus has been isolated from the internal contents of eggs and from egg shells from broiler breeder flocks both in the presence of clinical disease and in infected flocks with no clinical signs (Bean et al 1985; Cappucci et al 1985; Starick and Werner 2003; Swayne and Halvorson 2003). Swayne and Halvorson (2003) commented that, because AI is embryo-lethal, hatching of infected eggs is unlikely and recommended that cleaning of faecal material and disinfection of egg shells might be required to avoid dissemination of virus through hatcheries. This is consistent with the finding that movement of egg trays and associated fomites was a significant risk factor in the spread of AI infection during an epidemic in the Netherlands in 2003 (Thomas et al 2005).

There have been few reports of AI infection of psittacines and the vast majority of those have come from birds being traded either internationally or within countries. Reports of natural infections of psittacine birds were listed in a table identifying them as being "found dead upon arrival in importing countries in quarantine stations or in pet shops" (Kaleta et al 2007) and no references to psittacines were included in a collation of reports of AI in free-living birds (Stallknecht and Shane 1988).

The pathogenicity of AI virus strains varies depending upon the species infected (Mutinelli et al 2003; Perkins and Swayne 2003). Kaleta et al (2007) identified three reports of the use of AI isolates from psittacines, all from sick budgerigars (H3N8 and two of H4N6 from different sources), in transmission experiments. All experiments used budgerigars as the recipient species and results varied from no clinical signs to disease with some deaths. Challenge studies in budgerigars using HPAI subtype H5N1 (A/chicken/Hong Kong/220/97) resulted in mortalities (Perkins and Swayne 2003).

Tests used for the diagnosis of AI include serological tests such as agar gel immunodiffusion, haemagglutination, haemagglutination inhibition test and ELISAs. Virus antigen can be identified by virus isolation, antigen capture ELISA or RT-PCR (Alexander 2008).

No reports of investigations into the possible infection of psittacine eggs with AI have been located.

3.1.5. Hazard identification conclusion

In view of the above, AI viruses are classified as a potential hazard in the commodity.

3.2. RISK ASSESSMENT

3.2.1. Entry assessment

The majority of infections in caged birds are in passerines, with psittacines being infected only rarely (Alexander 2000). Alexander makes no reference to infection in free-living psittacines (Alexander 2000). This is consistent with the lack of reference to infection in psittacines in various reviews (Capua et al 2005; Kaleta et al 2007; Stallknecht and Shane 1988; Suarez 2000; Swayne and Halvorson 2003). Information on seven LPAI isolates from psittacines in the European Union between 1991 and 1994 indicted that none of these came from birds in quarantine (Capua et al 2005). Examination of the reports from which this data came, however, indicates that:

- three of the isolates (H7N2) came from imported birds placed in quarantine in Italy (Papparella et al 1993). H7N2 was said to be endemic in Italy at that time;
- one (H7N1) was from an aviary in the Netherlands with diseased birds (disease not attributed to AI) (Koch 1994);
- one was identified as part of investigations of disease at the property of a caged bird supplier and two isolates of the same subtype (H7N1) and with the same amino acid sequence were from another property (Alexander and Manvell 1994).

The full extent of contact between these birds and potential reservoir hosts is unknown.

Reports of later conferences do not include evidence of AI virus infections of psittacine birds outside quarantine. Capua et al (2005) recognised two reports of individual pet psittacines being infected with AI. Three reports of surveys in populations of wild psittacines gave negative results (Deem et al 2005; Karesh et al 1997; Stone et al 2005). Based on these reports, incidents of AI infection of psittacines other than at times of accumulation of birds in close proximity to birds of other Orders are rare.

The infection of parakeets, originating in Pakistan and dying in, or shortly after release from, quarantine in Japan, with H9N2 virus appears to have followed a course similar to that of entry of other viruses into other countries. Closely related viruses were endemic in Pakistan (Naeem et al 1999) and elsewhere in southern Asia. With 50,000 non-poultry birds being imported to Japan from Pakistan in each of the five years preceding these incidents (Mase et al 2001) exposure of the affected birds to other species prior to or during transport, or during quarantine, seem highly likely. In the apparent absence of any requirement for examinations of healthy birds it is highly likely that healthy infected birds were released from quarantine yet no further reports of this virus in healthy or diseased psittacines have been identified.

No reports of infection of psittacine birds with HPAI have been located.

The most likely sources of AI infection for psittacine birds are other birds, particularly passerines, in close proximity during international trade, quarantine, or in pet shops. Very large numbers of passerine birds are traded internationally and within countries. AI infection of passerines is reported more frequently than from psittacines (Alexander 2000; Capua et al 2005; Fukushi et al 1982; Ibrahim et al 1990; Kaleta et al 2005) and there has been considerable commonality in the monoclonal antibody types (Fukushi, et al 1982; Mase et al 2001; Senne et al 1983) and HxNy subtypes (especially H3N8 and H4N6) (Panigrahy et al 2003) reported in imported passerine and psittacine birds. The isolation of H7N2 viruses from psittacines in Italy in 1991 and of H7N1 from psittacines in the Netherlands and England in 1994 appears to have been isolated incidents with no further similar reports. Collation of data from the laboratories of the European Union from 2002 to 2005 shows that samples from 1,125 birds identified as psittacine were tested for AI with negative results. Several thousand other birds identified as caged, pet, zoo, or exotic birds were tested with negative results and many others recorded as “psittacine and passerine” or “psittacine and other caged birds” were also tested with negative results (Alexander 2002-2005).

The only study identified providing data on persistence of infection in a psittacine bird is that of Hawkins et al (2006). Following supportive therapy, a diseased red-lored Amazon parrot (*Arizona autumnalis autumnalis*) infected with an H5N2 AI virus recovered clinically and subsequent virological examinations (at days 8 and 42 after initial referral) were negative.

In summary

- AI virus has seldom been isolated from psittacine birds (Alexander 2000; Alexander 2002-2005; Deem et al 2005; Kaleta et al 2007; Karesh et al 1997; Stallknecht and

Shane 1988; Stone et al 2005; Suarez 2000; Swayne and Halvorson 2003) and therefore psittacine birds are unlikely to act as reservoir hosts for AI.

- Infection of psittacines is usually acquired during periods of very close mixing with birds of other orders, generally during the process of trading, either internationally or within countries. Birds most likely to infect psittacines are passerines since they are more likely to be infected with AI than psittacines (Alexander 2000; Capua et al 2005; Kaleta et al 2005; Senne et al 1983; Stallknecht and Shane 1988). However, AI virus has occasionally been isolated from psittacine birds (Capua et al 2005).
- One report indicates that a red lored Amazon parrot shed the virus for only a few days after infection (Hawkins et al 2006). However, this single study cannot be regarded as conclusive and must interpreted with caution since turkeys may carry the virus for up to 72 days (Swayne and Halvorson 2003)
- Eggs laid by infected birds may carry contamination on the shell. However, the commodity definition requires disinfection of eggs such that the likelihood of live virus in this location is negligible.
- Virus has been found in the albumen and yolk of eggs of naturally infected poultry (Cappucci et al 1985). According to Swayne and Halvorson (2003) Beard, Brugh and Johnson found that most eggs laid on days 3-4 days after experimental infection contained virus. However, AI viruses are lethal for embryos and hatching of infected eggs has never been demonstrated.

The likelihood of AI infection in birds from which eggs are to be collected is very low and, even if birds were to be infected, the likelihood of viral infection in eggs is again very low. However, since the virus has been isolated from psittacines and is found in chicken's eggs, the likelihood that AI virus could be introduced in the commodity is considered to be non-negligible.

3.2.2. Exposure assessment

Although imported psittacine birds are likely to be held in aviaries or cages the enclosures in which they are kept may not preclude contact with wild birds. Faeces removed from aviaries or cages may act as a pathway for infection of wild birds or poultry. And close contact with humans or cats could result in the infection of these animals which are susceptible to some strains of AI virus, particularly the H5N1 strain that has caused a world-wide pandemic in recent years. Therefore the exposure assessment is non-negligible.

3.2.3. Consequence assessment

H5Nx and H7Nx strains of AI virus must be reported as notifiable avian influenza (NAI) strains. HPNAI may cause catastrophic outbreaks of avian influenza in poultry and some strains are occasionally pathogenic for man and other animals such as cats. The effect new AI strains would have on native and introduced birds is not known and may vary for different strains and bird species. The consequences for domestic poultry, native and wild birds and human health are therefore assessed to be non-negligible.

3.2.4. Risk estimation

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

3.3. RISK MANAGEMENT

There are no *Code* recommendations that apply to psittacine eggs.

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

Option 1

Ensuring that the birds from which eggs are to be collected are from countries, zones or compartments that are free from NAI disease as defined in the *Code* (OIE 2008).

Option 2

Testing a sample of birds from each potential source flock for NAI with negative results (see comments above regarding test procedures).

Option 3

Maintaining birds from which eggs are to be collected in pre-export isolation prior to and during pre-export testing and egg-laying.

Option 4

Hatching the eggs and maintaining the hatchlings in quarantine and

a) testing material from all embryos/chicks dead-in-shell and from any hatchlings dying.
b) testing a sample of hatchlings prior to clearance.

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4. Avian paramyxoviruses

4.1. HAZARD IDENTIFICATION

4.1.1. Aetiological agent

Nine "prototype" virus strains of paramyxovirus are recognised in birds, which are differentiated on serological grounds and identified as avian paramyxoviruses 1 to 9 (APMV-1 to 9) (Alexander 2003).

Pathogenic strains of APMV-1 cause Newcastle disease (ND) and strains have, in the past, been differentiated on the basis of their ability to cause chick embryo mortality (Hanson and Brandly 1955). The OIE criteria for reporting an outbreak of ND provide for differentiation of isolates of APMV-1 on the basis of either intra-cerebral pathogenicity in day-old chicks or demonstration of specific amino acids at specific locations on the F1 and F2 proteins in the virus (Alexander 2008).

APMVs 1 to 9 show varying degrees of host specificity with APMV-1, APMV-2, APMV-3, and APMV-5 having been identified in psittacines (Alexander 2003; Leighton and Heckert 2007).

4.1.2. OIE list

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

4.1.3. New Zealand status

APMV-1 (exotic strains) (Newcastle disease) is listed as notifiable in the unwanted organisms register.

APMV-2, 3, and 5 are listed as "other exotic organisms" in the unwanted organisms register.

Newcastle disease has never been diagnosed in New Zealand. APMV-1 has been isolated from mallard ducks, chickens and one parrot in New Zealand, and all isolates have been demonstrated to be avirulent (Pharo et al 2000).

In addition to the APMV-1 isolations, APMV-4 was identified in samples from 17 ducks. Serological tests in ducks were positive for APMV-1, 2, 3, 4, 6, 7, 8, and 9 but, because of the cross reactivity that occurs between the prototype strains (Alexander 2003), only APMV-1, 4 and 6 could be concluded to be present. This did not preclude the possibility that infection with other strains might also have occurred. Testing did not include APMV-5 (Stanislawek et al 2002).

Stanislawek et al (2001) interpreted serological results from caged birds, wild birds, and poultry as indicative of the presence of APMV-1 in all categories and suggestive (but not confirmatory) of the presence of APMV-2 in caged and wild birds, including psittacines. Because of cross reactivity between APMVs, the presence of other APMVs in caged or wild birds could not be excluded.

4.1.4. Epidemiology

Newcastle disease virus (NDV) is distributed in poultry throughout the world with clinical disease being largely controlled, in more developed areas, through the widespread use of vaccines (Alexander 2003). Transmission between birds may be through either inhalation or ingestion. Geographic spread may be aided by movement of live birds, contact between

animal groups, movement of people and/or fomites, and spread in aerosols. Contamination of waterways, ponds, and surface water has also been proposed as means of spread of NDV (Alexander 1988; McFerran et al 1968). Infection in groups of animals may present with signs varying from high morbidity and high mortality to inapparent infections depending upon viral strain and host strain or species. There is anecdotal evidence that ND causes mild transient conjunctivitis and occasionally fever in humans. Reports of human to human transmission have not been identified (Alexander 2003).

Mutation of a NDV of low virulence was proposed as the most likely source of high virulence virus that caused a disease outbreak in Australia (Gould et al 2001; Kirkland 2000).

One review found that APMV-1 infection had been reported from 241 species of birds from 27 orders with differences in clinical presentation even between species within the same genus (Kaleta and Baldauf 1988). It has been proposed that the majority (if not all) birds are susceptible to infection (Alexander 2003).

APMV-2 (also called Yucaipa virus), most commonly infects turkeys and passerine birds. In these species it most commonly causes mild respiratory disease, although more severe disease has been reported in turkeys (Bankowski et al 1981). In wild and caged birds APMV-2 has been recorded from Europe, Asia, Africa, and America with most isolations being from passerine birds and a lesser number of reports from psittacine species (Alexander 2003; Leighton and Heckert 2007).

APMV-3 was first identified in turkeys in the United States and subsequently in other countries. In turkeys it affects egg production. There have been no reports of natural infections of chickens. A strain of APMV-3, distinct from that found in turkeys (Anderson et al 1987), has been isolated relatively frequently from caged and quarantined birds, mainly psittacines but also passerines (Alexander 2003; Leighton and Heckert 2007).

APMV-5 (Kunitachi virus) was reported from pet budgerigars in Japan between 1974 and 1976 (Yoshida et al 1977) and similar viruses have been identified from diseased budgerigars in Australia (Gough et al 1993; Mustaffa-Babjee and Spradbrow 1974; Mustaffa-Babjee et al 1974) and the United Kingdom (Gough et al 1993). Ritchie et al (1994b) stated that a similar virus had been isolated from free-ranging rainbow lories in Australia but this is not supported by the references provided. It is clear from the paper on APMV from budgerigars (Mustaffa-Babjee and Spradbrow 1974) that in the earlier paper on enteritis in lorikeets (Mustaffa-Babjee 1973) (paper not available) virological examinations were not carried out.

4.1.5. Hazard identification conclusion

APMV-1, 2, 3 (caged bird strain), and 5 are considered to be potential hazards in the commodity.

4.2. RISK ASSESSMENT

4.2.1. Entry assessment

APMV-1

Although surveys of free-living psittacines have found few birds with evidence of infection (Deem et al 2005; Garnett and Flanagan 1989; Gilardi et al 1995; Karesh et al 1997; Stanislawek et al 2001; Stone et al 2005), psittacines are considered to be amongst the most common reservoir hosts for APMV-1 (Ritchie et al 1994a). There are numerous reports of APMV-1 in captive psittacine birds, especially following international transport or during quarantine (Ashton and Alexander 1980; Clavijo et al 2000; Panigrahy et al 1993; Senne et al

1983). The virulence of isolates of APMV-1 from psittacines varies as does the susceptibility of different species of birds. Lorikeets were considered to be refractory to infection while cockatoos and Amazon parrots were regarded as highly susceptible (Ritchie et al 1994a). An epizootic was reported of velogenic Newcastle disease virus in caged psittacines. The epizootic occurred across six states within the United States and velogenic viruses were found in psittacines intended for importation (Panigrahy et al 1993). Parrots, parakeets, cockatiels, and conures were infected but the source of the virus was not determined.

APMV-2

Most reports of surveys of free-living psittacines have failed to provide evidence of APMV-2 infection (Deem et al 2005; Karesh et al 1997; Stone et al 2005), although three of 17 rainbow lorikeets (*Trichoglossus haematodus*) sampled in New Zealand had low antibody titres to the virus (Stanislawek et al 2001). These results were interpreted with caution because of low antibody titres to APMV-1 and -3 in these birds. Non-specificity in the testing was considered to be a likely explanation. Reports of identification of APMV-2 in passerine birds are relatively common (Fleury and Alexander 1979; Goodman and Hanson 1988; Nymadava et al 1977; Tumova et al 1979) and APMV-2 was isolated eight times more frequently than APMV-3 in finches being imported to the United States (Senne et al 1983). Passerine birds were considered to be the primary hosts for APMV-2 and psittacines became infected when in close proximity (Alexander 1986).

AMPV-3 (caged bird strain)

Discovered reports of surveys of free-living psittacines have failed to provide evidence of APMV-3 infection (Deem et al 2005; Karesh et al 1997; Stone et al 2005). One of the 17 rainbow lorikeets (*Trichoglossus haematodus*) sampled in New Zealand had a low antibody titre to the virus (Stanislawek et al 2001) but this result was interpreted as being due to either cross reactivity or non-specificity and it was concluded that it was not evidence of APMV-3 infection. In pet birds in quarantine on entering the United States, APMV-3 was isolated eight times more frequently from psittacines than from passerine birds (Senne et al 1983). If the proposition that psittacine birds are the primary hosts for APMV-3 (caged bird strains) (Alexander 1986) is correct, then the actual reservoir in free living birds has yet to be identified.

APMV-5

Reports of APMV-5 (or APMV-5-like) viruses have come only from budgerigars (Gough et al 1993; Mustaffa-Babjee et al 1974; Nerome et al 1978). The geographic dispersion, however, is wide, with cases being reported from Australia, Japan, and the UK.

The occurrence of true vertical transmission of NDV is controversial, at least in part because birds infected with pathogenic strains commonly cease laying and also because infection of eggs commonly results in death of the embryo (Alexander 2003). Lethality of NDV in embryonated eggs is, however, used as measure of virulence of virus isolates (Alexander 2008), so the comments by Alexander are interpreted as referring to transmission of virulent NDV during outbreaks of disease. Chen and Wang (Chen and Wang 2002), on the basis of epidemiological evidence and results from experimental infection of chicken embryos, concluded that egg borne transmission of NDV was possible. Yucaipa virus and Bangor virus strains (both members of APMV-2) isolated from finches grew in eggs and some embryos survived (Chen and Wang 2002; McFerran et al 1974). While there may be doubt whether true transovarial vertical transmission of APMV occurs, there are fewer doubts that APMV can penetrate either cracked or intact egg shells after laying. While there is no information available on the role of the egg in transmission of APMV in psittacines and the evidence for

egg borne transmission is limited, the likelihood of such a means of spread is considered to be non-negligible.

In view of the above, the entry assessment for APMV-1, 2, and 3 (cage bird strains) in the commodity is considered to be non-negligible.

The entry assessment for APMV-5 in budgerigar eggs is considered to be non-negligible, whereas the entry assessment for APMV-5 in the eggs of species other than budgerigars is considered to be negligible.

4.2.2. Exposure assessment

Although little is known of the epidemiology of APMVs other than APMV-1, all can be expected to behave similarly. Should infected eggs be imported, and lead to infected birds that are given biosecurity clearance, spread to other susceptible species is likely. The extent of that exposure would be limited as imported psittacines are likely to be of high value and held in cages or aviaries. In addition, transmission of infection will depend on the host specificity of the strain of virus and the host range that might be exposed. It is relevant that the velogenic strain of APMV-1 affecting psittacine birds across six of the United States did not spread to other avian orders and that the poultry industry was unaffected (Panigrahy et al 1993). Although a significant number of pet birds were killed during the eradication of Newcastle disease from California in 2003, testing of these indicated that only two psittacines that were kept in close physical contact with game birds were infected, suggesting they had little (if any) role in the transmission of infection to other flocks (Pharo 2003).

While the likelihood of transmission of virus beyond the hatchlings from imported eggs and their direct contacts is low, the exposure assessment for APMV-1, 2 and 3 (cage bird strains) in psittacine eggs and for APMV-5 in budgerigars is considered to be non-negligible.

4.2.3. Consequence assessment

APMV-1

The potential consequences of introduction of new strains of APMV-1 to New Zealand vary greatly. The lentogenic strain present in New Zealand is reported to spread relatively slowly in poultry and introduction of a strain that spread rapidly could disrupt current sero-surveillance (Christensen 2005). Otherwise such an introduction would be of no consequence unless it subsequently mutated to a more pathogenic form.

The introduction of a velogenic strain would have serious consequences for the poultry industry and it could result in significant mortalities in wild and/or caged birds. Although there are anecdotal reports of APMV-1 causing disease in humans, these reports have not been confined to velogenic strains. Given the presence of a lentogenic strain of APMV-1 in New Zealand and the mild and transient nature of the disease reported anecdotally as being caused by APMV-1 in humans (Alexander 2003), any consequence to human health is considered negligible.

The consequences of APMV-1 would depend upon the virulence of the strain entering the country but, even with strains not virulent in poultry, disease in wild or caged birds could occur and, with genetic changes, the likelihood of serious disease in poultry cannot be excluded.

The consequences of introducing of APMV-1 are considered to be non-negligible for the poultry industries and many species of both free living and caged birds. Any consequences to human health are considered minor, if not negligible. The consequence assessment for AMPV-1 is considered to be non-negligible.

APMV-2

APMV-2 has been isolated from poultry species, especially turkeys and chickens, in many parts of the world but reports of disease are scarce. Although there have been reports of severe respiratory disease in turkeys in both the United States and Israel associated with APMV-2 (Alexander 2003) an epidemiological study of the relationship between APMV-2 infection and acute respiratory disease syndrome in the US did not indicate an aetiological connection and experimental infections of both turkeys (Bankowski et al 1981) and chickens (Bankowski and Corstvet 1961) resulted in only mild disease. Reports of disease associated with APMV-2 in natural infections of psittacine, passerine, or other avian orders have not been located and the virus is generally considered to be non-pathogenic in non-poultry species. However, decreased activity in recently experimentally infected finches suggested that the behavioural changes could result in increased susceptibility of wild birds to disease (Goodman and Hanson 1988).

The consequences of APMV- 2 in the commodity would be restricted to the New Zealand poultry industry where there would be a low likelihood of disease.

The consequences of introducing of APMV-2 are considered to be non-negligible for the poultry industry, especially the turkey industry. The potential effect on a naive population of native psittacine birds is not known. The consequences for other sectors of the economy, and human health are considered to be negligible.

The consequence assessment for APMV-2 is considered to be non-negligible.

APMV-3 (Caged bird strains)

APMV-3 has been isolated from caged birds with a number of reports being from birds involved in international or internal trade (Alexander 2003). Alexander recognised three reports of disease in caged birds associated with APMV-3. These were:

- A case of large numbers of deaths of lovebirds (*Agapornis roseicollis*) in the United States, shortly after receipt from an importer. The birds were emaciated with hepatosplenomegaly. Other psittacine and passerine birds housed nearby were not affected (Goodman and Hanson 1988; Hirai et al 1982; Hitchner and Hirai 1979).
- Deaths of parakeets (*Neophema* spp.) in a number of aviaries over a period of ten years in the Netherlands (Smit and Rondhuis 1976). Clinical signs of central nervous system disease resembling the nervous form of Newcastle disease preceded death in *Neophema* spp. while psittacines of other genera sharing the same cages were seldom affected. Occasional cases of less severe disease in passerines were confirmed as having the same cause. Experimental challenge confirmed the susceptibility of *Neophema* spp. and of red-rump parakeets (*Psephotus haematonotus*), but budgerigars (*Melopsittacus undulatus*) and cockatiels (*Nymphicus hollandicus*) were not infected.
- Cases of steatorrhea and pancreatic atrophy in captive psittacines in Belgium (Uyttebroek et al 1991).

It has been suggested that proventricular dilatation disease of psittacines (previously Macaw wasting syndrome) might be caused by a paramyxovirus but the evidence is not strong and a number of alternative aetiologies have been proposed (Gregory et al 1995) and more recently an avian Bornavirus has been reported to be the cause of this condition.

The introduction of APMV-3 strains with imported psittacine eggs could result in disease in caged psittacines but the likelihood of disease in free-living birds is considered remote.

The consequences of introducing of APMV-3 are considered to be non-negligible for caged psittacines. The susceptibility of native or endemic species is unknown but the potential for disease cannot be excluded. The consequences for the economy or human health are considered to be negligible. The consequence assessment for AMPV-3 is considered to be non-negligible.

APMV-5

APMV-5 has been recognised only in budgerigars in which high mortality rates may occur. The introduction of APMV-5 with budgerigar eggs could result in deaths in budgerigar aviaries.

The consequences of the introduction of APMV-5 would be restricted to budgerigars. There is a low likelihood that episodes of high mortality rates could arise.

APMV-5 in budgerigar eggs may result in occasional episodes of mortality in budgerigar aviaries. While most budgerigars are readily bred and available for sale at $10 to $15 each, the economic consequences for breeders of high value show budgerigars are likely to be non-negligible. High value show birds are the only budgerigars likely to be imported.

4.2.4. Risk estimation

Since the entry, exposure, and consequence assessments are non-negligible, the risk estimate for APMV-1, 2, 3 (caged bird strains) and 5 (only in budgerigars) is non-negligible, and they are classified as hazards in the commodity. Therefore, risk management measures can be justified.

4.3. RISK MANAGEMENT

The recommendations relating to APMVs and hatching eggs in the *Code* are specifically relevant to Newcastle Disease and poultry eggs. They are:

a. When importing from ND free countries, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the hatching eggs come from establishments or hatcheries situated in a Newcastle Disease free country and which are regularly inspected by the Veterinary Authority.

b. When importing from countries considered infected with ND, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the hatching eggs:
    i) have been disinfected in conformity with procedures established by the OIE;
    ii) come from establishments or hatcheries which are regularly inspected by the Veterinary Authority;
    iii) come from establishments or hatcheries free from ND and not situated in an ND infected zone;
    iv) come from establishments or hatcheries in which birds were not vaccinated against ND; or
    v) come from establishments or hatcheries in which birds were vaccinated against ND (the nature of the vaccine used and the date of vaccination shall also be stated in the certificate).

Cultural methods for detection of APMVs have been described (Alexander 2008). Any APMV isolate recovered could be serotyped, paying particular attention to the antigens and antisera used to avoid erroneous identification. Alternatively, on the basis of the epidemiology

and entry assessments in this risk analysis, it is reasonable to assume that any APMV isolated would be either APMV-1, 2, or 3 (cage bird strain) or APMV-5 (from budgerigars).

Haemagglutination, haemagglutination inhibition tests and ELISAs are used in the serological diagnosis of Newcastle disease (Alexander 2008). Validation of tests has mainly focussed on APMV-1 in poultry and Alexander (Alexander 2003) comments on the need for care in reagent selection.

The use of sentinel specific-pathogen-free chickens in contact with hatchlings may allow detection of some strains of APMV-1 and 2 but it is likely that chickens may not be susceptible to infection by other strains. This procedure may not allow the detection of APMV-3 as, although one-day-old chickens have been shown to be susceptible to experimental infection with one isolate of APMV-3 this was not a cage-bird strain and there are no reports of natural infection of chickens with APMV-3.

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

Option 1

Ensuring that the birds from which eggs are to be collected are from flocks in areas recognised as free from notifiable Newcastle disease as defined in the *Code* (OIE 2008).

This will provide a high level of assurance that the birds are not carrying velogenic APMV-1.

Option 2

Testing a sample of birds from each potential source flock for APMV with negative results (see comments above regarding test procedures).

Option 3

Maintaining birds from which eggs are to be collected in pre-export isolation prior to and during pre-export testing and egg-laying.

Option 4

Hatching the eggs and maintaining the hatchlings in quarantine and

a) testing material from all embryos/chicks dead-in-shell and from any hatchlings dying.

b) testing a sample of hatchlings prior to clearance.

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5. Psittacine herpesviruses

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological agent

Psittacine herpesviruses (PsHV) are a heterogenous group of viruses in the family Herpesviridae and the subfamily Alphaherpesvirinae. The group displays both serological and genetic heterogeneity.

5.1.2. OIE list

No PsHVs infections are OIE listed diseases.

5.1.3. New Zealand status

Although Pacheco's disease virus is included in the register of unwanted organisms as an exotic organism, there remains some uncertainty surrounding New Zealand's status in regard to this virus.

Pacheco's disease was diagnosed in two incidents of mortalities in parrots in the South Island of New Zealand in 1977. One of these, in a Christchurch aviary, involved birds that had been moved from the North Island 2 weeks earlier, and the other case was in an aviary in Oamaru. The diagnoses were based on clinical signs, pathology and on viral isolation and confirmation of the virus as a herpesvirus (Durham et al 1977). In a later attempt to verify these diagnoses, in 1997 material from formalin fixed, paraffin embedded tissues from the 1977 cases was tested for evidence of Pacheco's disease virus, using *in situ* hybridisation, with negative results (Loth 2003).

When Pacheco's disease was diagnosed in 5 of 129 parrots in quarantine shortly after their import from the UK in early 1997, the MAF position was that there was insufficient information to treat Pacheco's disease other than as an exotic disease. Although the 129 imported parrots were condemned in quarantine, about half of them were illegally removed before they could be destroyed. While some of these missing birds were traced, most were never recovered and their disease status remains unknown. The MAF position as regards Pacheco's disease was unchanged as a result of this investigation (Thornton and Stanislawek 2003).

In 2001 a serological survey was carried out for a number of diseases in a range of New Zealand birds, including 26 wild parrots and 70 captive parrots. Testing carried out in the USA resulted in one Pacheco's virus neutralisation test out of 19 samples taken from one aviary. However, several months after the initial sampling, the bird that returned the positive serological test was resampled (blood and cloacal swab) and both serological and cultural tests carried out at the Ministry of Agriculture and Forestry laboratory were negative. The results of this survey did not change the MAF position that New Zealand is considered free of Pacheco's disease virus (Loth 2003).

5.1.4. Epidemiology

Herpesviruses are considered to be the causal agents for Pacheco's disease (Simpson and Hanley 1977), mucosal papillomas (Johne et al 2002; Styles et al 2004), and Amazon tracheitis (Gerlach et al 1998; Helfer et al 1980; Suarez et al 2003; Winteroll and Gylstorff

1979). Of these, Pacheco's disease has been reported most frequently, and studied most intensively.

In his review of the evolution of herpesviruses, Davison (2002) states that, in nature, each herpesvirus is closely associated with a single host species and that some host species may be infected by more than one distinct herpesvirus. He also suggests that the number of herpesviruses is likely to be much greater than the number so far identified. Herpesviruses are highly host adapted, establishing life-long infections but causing little or no disease in their evolutionary hosts. Infection in non-adapted species may result in disease and Davison (2002) comments that high pathogenicity of herpesviruses in man and farmed animals is, invariably, the result of "disequilibrium" arising from human activity. In non-fatal infections the herpesvirus may be incorporated into host DNA and then become reactivated, particularly during periods of stress. At times of reactivation clinical signs may reappear (Davison and Clements 1998; Fenner et al 1993). These features of herpesviruses mean that it is difficult to interpret many of the studies of herpesviruses of psittacines because the viruses are commonly identified and studied on the basis of their pathogenicity, or history of causing disease, in particular hosts which may not be their natural hosts.

Pacheco's disease is characterised by focal hepatic necrosis, with associated inflammatory lesions, and death. Morbidity rates vary. Mortality rates amongst birds showing clinical signs are commonly high. The disease is present in psittacine birds in many countries (Cho and McDonald 1980). Previously it was proposed that Pacheco's disease was caused by a single avian herpesvirus (probably a betaherpesvirus) with three serotypes (Kaleta 1990). More recently it has been established that avian herpesviruses conform to the criteria for Alphaherpesviridae (Tomaszewski et al 2001; VanDevanter et al 1996). Within the psittacine herpesviruses (PsHV) wide diversity in both serotype (Gravendyck et al 1996; Tomaszewski et al 2003; Vindevogel et al 1980) and genotype (Schroder-Gravendyck et al 2001; Tomaszewski et al 2001; Tomaszewski et al 2003) have been reported.

Many of the incidents of Pacheco's disease occur in birds in quarantine (Gough and Alexander 1993; Horner et al 1992; Senne et al 1983) and, commonly, other reports refer to the presence of more than one species of psittacine in close proximity and/or histories of presence in pet shops or introduction of new birds to aviaries (Durham et al 1977; Gravendyck et al 1998; Gunther et al 1997; Krautwald et al 1988; Simpson and Hanley 1977). It is likely that the psittacine species in which severe disease is demonstrated are not the natural hosts for that genotype of the virus.

Serological surveys for evidence of PsHV found 11% of free-ranging dusky-headed parakeets (*Aratinga weddellii*) in Peru to be positive to a complement fixation test for herpesvirus, whereas samples from Tui parakeets (*Brotogeris sanctithomae*) were negative (Durham et al 1977; Gilardi et al 1995; Gravendyck et al 1998; Gunther et al 1997; Krautwald et al 1988; Simpson and Hanley 1977). All 411 psittacines, including free-living and captive birds, tested in Australia were negative to a serum neutralisation test (SNT) for PsHV (Raidal et al 1998) and in Costa Rica two of 128 samples from captive scarlet macaws (*Ara macao*) were positive to an SNT for Pacheco's disease virus (Herrera et al 2001). Beyond providing positive evidence that a PsHV was present in the populations of free-living *Aratinga weddellii* in Peru and captive *Ara macao* in Costa Rica, these surveys tell us little about the epidemiology of the virus(es). The ability of herpesviruses to become incorporated into host DNA and persist in the absence of serological evidence means that the prevalence of infection may be greater than indicated by the serological test results, and that negative results are not evidence of the absence of the virus.

PsHVs cause mucosal papillomas in psittacines (Johne et al 2002; Styles et al 2004). Lesions are found most commonly in the cloaca and upper gastrointestinal tract (Schmidt 1996a;

Schmidt 1996b). Proliferative cutaneous lesions have been described on the legs and feet, mainly in macaws and cockatoos and their aetiology ascribed to herpesvirus although reports of virus isolation or characterisation have not been located.

Respiratory disease, to which the name "Amazon tracheitis" has been ascribed, has been reported from *Amazona*, *Neophema*, *Neopsephotus*, *Platycercus*, and *Oreopsittacus* species (Schmidt 1996a; Schmidt 1996b). Histories of transport and then housing in quarantine stations are common. A herpesvirus similar to, but distinct from, the virus causing infectious laryngotracheitis in chickens is thought to be the cause (Gerlach et al 1998; Winteroll and Gylstorff 1979).

5.1.5. Hazard identification conclusion

PsHVs are considered to be potential hazards in the commodity.

5.2. RISK ASSESSMENT

5.2.1. Entry assessment

Literature searches revealed only one report of vertical transmission of an avian herpesvirus. Burgess and Yuill (1981) reported that clinically healthy ducks infected with strains of duck virus enteritis virus (DVEV) laid eggs with decreased hatchability and this was attributable to DVEV. Some hatchlings died within two weeks while survivors beyond that time carried infections of DVEV and excreted virus. The significance of these findings in the epidemiology of the disease remained unknown because the quantity of virus shed by surviving hatchlings was low and the authors were uncertain whether exposure to such levels of virus would result in infection of other birds. Virus has not been recovered from eggs during naturally occurring outbreaks of duck virus enteritis (Sandhu and Shawky 2003).

With respect to other herpesvirus infections of birds, vertical transmission of pigeon herpesvirus is considered unlikely (Vindevogel and Pastoret 1980), vertical transmission of turkey herpesvirus has not been demonstrated (Witter and Schat 2003), egg borne transmission of Marek's disease virus does not occur (Witter and Schat 2003), and it has not been demonstrated in infectious laryngotracheitis (Guy and Bagust 2003). No other reports of vertical transmission of avian herpesviruses, whether associated with natural or experimental infections, have been located.

In view of the above, the likelihood of transmission through eggs is considered to be negligible. Therefore the entry assessment is considered to be negligible.

5.2.2. Risk estimation

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

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6. Psittacine adenoviruses

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Three genera of adenoviruses have been reported from birds; Aviadenoviruses (previously Group I), Siadenoviruses (previously Group II) and Atadenoviruses (previously Group III) (Benko et al 2005; McFerran 2003).

6.1.2. OIE list

No avian adenovirus infections are OIE listed of diseases.

6.1.3. New Zealand status

Avian adenoviruses are not included in the register of unwanted organisms.

A number of the aviadenoviruses of fowl (FAdV) are endemic in New Zealand poultry (Saifuddin et al 1992). Serological reactions to avian adenoviruses are found routinely in flock surveillance programmes (Poland 2004). A serological survey of pigeons for aviadenovirus revealed positive titres to be common in all geographic areas from which samples were collected (Stanislawek 2008).

A suspect adenovirus infection has been reported in a Jardine's parrot in New Zealand (Stone 2005) and records are available of suspect adenovirus infections in a rainbow lorikeet, a cockatiel, a red-tailed black cockatoo, and a parrot (Gartrell 2007). However, adenoviruses of psittacines have not been isolated or characterised in this country.

6.1.4. Epidemiology

6.1.4.1. Aviadenoviruses

Aviadenoviruses have been reported from a wide range of birds including species in the Orders Galliformes, Columbiformes, Anseriformes, and Psittaciformes (Gerlach 1994). They are considered to be ubiquitous in populations of chickens and widespread in turkeys and geese (McFerran and Adair 2003b). A survey of 293 budgerigars in Japan found evidence suggestive of adenoviral infection in almost 60% of the birds (Okita 1989). Although there are reports of FAdV infecting species other than chickens, there is evidence of host specificity, or host preference, for some aviadenoviruses with recognition of five species of Fowl adenovirus and one species of Goose adenovirus. Duck adenovirus B, Pigeon adenovirus and Turkey adenovirus B have also been given tentative recognition (Benko et al 2005)

The role of aviadenoviruses as primary pathogens has not been clearly established and many birds are infected in the absence of disease. Horizontal and transovarial transmission of aviadenoviruses is described (McFerran and Adair 2003b).

There are a number of reports of aviadenoviruses (or suspect aviadenoviruses) in a wide range of psittacines species. These infections are, most commonly, detected as intranuclear inclusion bodies in hepatocytes, the epithelium of renal tubules and/or intestinal epithelium without associated pathology (Desmidt et al 1991; Gomez-Villamandos et al 1992; McFerran and Adair 2003b; Mori et al 1989; Okita 1989; Pennycott 2004; Scott et al 1986; Tsai et al 1994; Weissenbock and Fuchs 1995). Some reports ascribe a specific pathogenic role to

adenoviruses, particularly as a cause of hepatitis (Capua et al 1995; Droual et al 1995; Scott et al 1986), necrotising enteritis (Droual et al 1995; Mackie et al 2003), or nephropathy (Okita 1989) but other factors contributing to the disease are not commonly reported. Relatively few reports of isolation and/or characterisation of adenoviruses in psittacines have been located but aviadenoviruses resembling FAdV serotypes 2 (McFerran et al 1976), 3 (Capua et al 1995), 4 (Gassmann et al 1981), 8 (McFerran et al 1976), and both 2 and 11 (Gassmann et al 1981) have been identified from psittacines.

Recently there have been reports of infection of psittacines with aviadenoviruses distinct from those previously recognised in either psittacine or other species. Wellehan et al (2005) differentiated an aviadenovirus associated with characteristic inclusion body hepatitis in a Meyer's parrot (*Piocephalus meyeri*) from other adenoviruses using PCR methodology and proposed that it be called Meyer's parrot adenovirus. A virus isolated from Senegal parrots (*Piocephalus senegalus*), with a hepatopathy typical of that associated with aviadenovirus, was characterised as being a group I adenovirus of a serotype not previously reported and designated it as psittacine adenovirus (PsAdV) (Raue et al 2005) and aviadenoviruses were isolated from *Poicephalus* spp., *Cacatua* sp., *Amazona* sp., and *Psittacula* sp. with inclusion body hepatitis in a single psittacine collection (Luschow et al 2007).

6.1.4.2. Siadenoviruses

Turkey adenovirus A is the only species of siadenovirus recognised as infecting birds (Benko et al 2005). Isolates have commonly been differentiated on the basis of the species from which they have been isolated - avian adenovirus splenomegaly virus (from chickens), marble spleen disease virus (from pheasants), and turkey haemorrhagic enteritis virus (from turkeys) (Benko et al 2005; Pierson and Fitzgerald 2003). A virus recovered from psittacines with haemorrhagic enteritis and focal necrosis of the spleen was reported as a Group II adenovirus (Siadenovirus) (Gomez-Villamandos et al 1995). This is the only report located suggesting infection of psittacines with an adenovirus other than an aviadenovirus.

There is no evidence that Siadenoviruses are transmitted via eggs.

6.1.4.3. Atadenoviruses

Duck adenovirus A is the only species of atadenovirus recognised as infecting birds. This virus causes egg drop syndrome in chickens and it is thought that ducks and geese may act as reservoir hosts (Benko et al 2005; McFerran and Adair 2003a). No reports suggesting that duck adenovirus A, or any other atadenovirus, might infect psittacines birds have been located.

6.1.5. Hazard identification conclusion

6.1.5.1. Aviadenovirus

It is concluded that aviadenovirus-associated disease occurs in psittacine species in New Zealand and that there is no basis for suspecting that the viruses causing such disease are less pathogenic than those in other countries. Therefore aviadenoviruses are not considered to be potential hazards in the commodity.

6.1.5.2. Siadenovirus

On the basis of the scarcity of reports in psittacines and the absence of evidence that siadenoviruses might be transmitted via eggs, it is concluded that they are not a potential hazard in the commodity.

6.1.5.3. Atadenovirus

On the basis that there is no evidence that atadenoviruses infect psittacines, it is concluded that these viruses are not a potential hazard in the commodity.

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7. Psittacine beak and feather disease (PBFD)

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

PBFD is caused by a circovirus with a range of genotypes with varying degrees of host specificity. Genotypes in psittacines of Australian and African origin differ (Heath et al 2004; Kloet and Kloet 2004)

7.1.2. OIE list

No infections caused by Circoviridae are OIE listed diseases.

7.1.3. New Zealand status

No avian members of the Circoviridae are included in the register of unwanted organisms.

Psittacine beak and feather disease virus (PBFDV) is present in free-living eastern rosellas (*Platycercus eximius*) and sulphur crested cockatoos (*Cacatua galerita*) in New Zealand (Mander et al 2003; Ha et al 2007). It causes disease in both captive and imported psittacine species (Anonymous 1997; Anonymous 1999; Jakob-Hoff 2003; Ritchie et al 2003). It was identified in 21 of 25 captive psittacine birds in New Zealand, from 8 of the 10 species sampled (Ritchie et al 2003). Genotype clustering of viruses within related psittacine species was observed, with one cluster infecting cockatoos and another infecting lorikeets. An isolate from a budgerigar was placed in a separate lineage. The cluster pattern observed was similar to that seen in Australia (Bassami et al 2001) and it is considered likely that PBFDV was introduced into New Zealand in psittacines imported from Australia.

A survey of 169 wild native parrots and 143 captive native parrots found a pair of red-crowned parakeets and two Antipodes Island parakeets from different captive facilities that were infected with PBFDV (Ha et al 2009). PBFDV has also been recovered from wild specimens of an endemic New Zealand parrot, the red-fronted parakeet (Ortiz-Catedral et al 2010).

7.1.4. Epidemiology

PBFD is considered one of the most serious viral diseases of free-living and captive psittacines. First recognised in the early 1970s as causing deformities of beaks and feathers in large numbers of free-living cockatoos in Australia (Anonymous 2004; Jakob-Hoff 2003; McOrist et al 1984), it has subsequently been recognised as the cause of major economic losses in the pet trade (Woods and Latimer 2003). An annual mortality rate of 10 – 20% in South African psittacine breeding stock has been attributed to PBFD (Heath et al 2004) and it has affected at least two threatened species in that country (Warburton and Perrin 2002). Surveys have revealed seroprevalences from 41% to 91% in flocks of free-living psittacines in New South Wales (Raidal et al 1993), and 40%, 24% and 21% in captive budgerigars, cockatoos and African grey parrots respectively in Japan (Sanada and Sanada 2007). The disease has been reported from at least 61 psittacine species and from many countries around the world (Cross 1996).

There is considerable genetic diversity amongst PBFDVs (Bassami et al 2001). Phylogenetic analysis has resulted in groupings of viral strains that parallel the phylogenetic groupings of their psittacine hosts. Several strains have been identified with differences within and between

viruses infecting psittacine species of Australian and African origin (Heath et al 2004; Kloet and Kloet 2004; Ritchie et al 2003). However, an examination of the entire genome of Australian PFDB isolates led to the conclusion that differences in pathogenicity between PBFD isolates were not significant (Bassami et al 2001). This conclusion is supported by the findings of several other phylogenetic studies of isolates in diseased lories and African grey parrots (Schoemaker et al 2000; Raue et al 2004).

It is concluded that while there are different strains of PBFDV infecting different psittacine species, host specificity is not absolute. Kloet and Kloet (2004) concluded that the relationship between PBFDV strain, psittacine species and pathogenicity was very complex and likely to be influenced by other factors such as age of bird and the presence of secondary infections. Although the pathogenicity of the virus in particular host species may vary, "it must be assumed that all psittacine bird species are potentially susceptible to each genotype" of PFBD virus (Khalesi et al 2005).

7.1.5. Hazard identification conclusion

Since pathogenic PBFDV genotypes are widely distributed in psittacines in New Zealand and there is no evidence of significant differences in pathogenicity between PBFD isolates, it is concluded that PBFDV is not classified as a potential hazard in the commodity.

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8. Rotavirus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Rotavirus is a genus within the Reoviridae family. Members of this genus cause diarrhoea in intensively reared animals worldwide (Quinn et al 2002). Rotaviruses have been differentiated on the basis of a group antigen. The vast majority of both mammalian and avian rotaviruses fall within the conventional group A, whereas Groups B, C, and E rotaviruses are found in mammals and groups D, F, and G in birds (McNulty 2003).

8.1.2. OIE list

Avian rotaviruses infections are not OIE listed diseases.

8.1.3. New Zealand status

There are no rotaviruses listed in the unwanted organisms register.

Species from which rotaviruses have been reported in New Zealand include cattle, foals, dogs, cats, pigs, chickens, rabbit, deer, and humans (Black and Orr 1966; Fu 1987; Fu et al 1989; Holdaway et al 1982; Saifuddin et al 1989; Schroeder et al 1983; Townsend 1994).

8.1.4. Epidemiology

Avian rotavirus infection has been described in turkeys, chickens, pheasants, partridges, ducks, guinea fowl, pigeons, and lovebirds. Virus is excreted in faeces, contaminates the environment, and leads to horizontal transmission. Both infection and disease are most common in young birds. Infection in the absence of disease is common. Strain variations in pathogenicity occur and viral strains are generally, but not exclusively, host specific (McNulty 2003).

Egg transmission of rotavirus in turkeys was postulated on the basis of detection of infection in three-day-old poults (Theil and Saif 1987). Supporting evidence has not been forthcoming in the 20 years since that report and the development of clinical rotaviral infections in calves in the very early days of life (Theil and Saif 1987) suggests that egg transmission is not required as an explanation for the early development of disease in chickens.

Searches of the literature have not identified any reports of rotavirus in psittacines beyond one report in lovebirds in 1988 (Gough et al 1988).

8.1.5. Hazard identification conclusion

Rotaviruses are considered to be potential hazards in the commodity.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

The scarcity of reports of rotavirus in psittacine birds does not exclude the likelihood that sub-clinical infections may occur, however, there is no evidence that avian rotavirus infection is transmitted through eggs.

The combined scarcity of reports of rotaviruses in psittacines and the lack of evidence that rotaviruses are transmitted through eggs means that the likelihood of infection arising from the importation of clean psittacine eggs is negligible.

The entry assessment for rotaviruses in the commodity is considered to be negligible.

8.2.2. Risk estimation

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

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9. Reovirus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Orthoreovirus (Ritchie 1995; Chappell et al 2005). Ritchie (1995) describes 11 serotypes of avian orthoreoviruses.

9.1.2. OIE list

Avian reovirus infections are not OIE listed diseases.

9.1.3. New Zealand status

Not listed in the unwanted organisms register.

Avian orthoreoviruses have been recovered from multiple avian host species in many countries (Doyle 1997; Ritchie 1995; Jones 2000; van den Brand et al 2007). Avian reovirus was first identified in New Zealand poultry in 1976 (Green et al) and vaccination against this virus in poultry is now common (Anonymous 1999; Howell 1992; Poland 2004; Saifuddin et al 1989).

No reports of psittacine reovirus infections in New Zealand have been located. While there has been speculation that the occurrence of reovirus-related disease in Australian king parrots (*Alisterus scapularis*), exported from New Zealand to Italy, might have been due to infection of those birds prior to export (Conzo et al 2001), closer inspection of this report suggests the birds exported from New Zealand were infected by a shipment of African grey parrots (*Psittacus erithacus erithacus*) from Zaire that were being held in the same Italian quarantine facility.

9.1.4. Epidemiology

Orthoreoviruses are recovered most commonly from the gastrointestinal tract of clinically normal birds. A few strains have been associated with disease in several species of gallinaceous birds, psittacine birds, and waterfowl (Ritchie 1995). Several incidents of fatal disease associated with reovirus (or reovirus-like) infections in psittacines have been reported and the reports of disease in psittacines have multiplied as the worldwide market for imported birds has grown (Sanchez-Cordon et al 2002). When clinical signs are seen, these are usually limited to depression, anorexia, dyspepsia and nasal discharge. The prognosis for old world psittacines is usually poor, especially for African grey parrots and cockatoos, whereas new world psittacines usually respond to treatment (Ritchie 1995). Common pathological findings are splenomegaly and multifocal hepatocellular necrosis.

The exact routes of reovirus transmission in companion and aviary birds is unclear. In chickens, reoviruses are primarily transmitted horizontally, principally by direct contact or indirect contact with contaminated faeces. Egg transmission has been documented to occur only occasionally in ducks, geese, turkeys and chickens. This route of transmission has not been confirmed in companion birds (Ritchie 1995).

Most reports of psittacine reovirus disease relate to birds recently moved, birds in pet shops, or birds in (or recently released from) quarantine (Ashton et al 1984; Conzo et al 2001; Graham 1987; Meulemans et al 1983; Sanchez-Cordon et al 2002; Senne et al 1983; Wilson et al 1985). Non-clinical infections of birds may occur (Senne et al 1983). Reports of two

epizootics of reovirus-associated disease in psittacines have been located. In 2002/03 there were numerous deaths of budgerigars in aviaries in Scotland (Pennycott 2004). PAGE and PCR analyses of isolates from these incidents showed the viruses to be distinct from mammalian and other avian reoviruses with which they were compared. From 2002 to 2004 numerous fatalities of psittacines in the Netherlands were noted. Reoviruses were consistent findings from these birds and testing with a range of monoclonal antibodies showed isolates to be distinct from chicken reoviruses but similar to viruses from the Scottish budgerigar outbreak (van den Brand et al 2007). There was a known history of recent introduction of birds to almost 50% of the aviaries affected in the Netherlands and it was thought that stress associated with movement of birds between cages within aviaries may have contributed to other cases.

Some reoviruses recovered from companion and aviary birds have been shown to be related to strains found in poultry, whilst others have been shown to be serologically distinct (Ritchie 1995). Psittacine reovirus infections have not been described in New Zealand. For the purposes of this risk analysis, psittacine reoviruses are considered to be exotic to New Zealand.

9.1.5. Hazard identification conclusion

It is concluded that reoviruses of psittacines are distinct from reoviruses affecting poultry species and, as psittacine reoviruses have not been reported in New Zealand, they are classified as a potential hazard in the commodity.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Reports of studies or epidemiological observations on the likelihood of egg-borne transmission of reoviruses in psittacines have not been discovered. Natural egg-borne transmission of reovirus in poultry has not been described although infection of eggs laid by hens experimentally infected with high doses of reovirus has been demonstrated.

It was found that experimental infection of eggs resulted in high embryo mortality rates but at lower doses some infected embryos hatched normally. More than 50% of embryos were killed by a virus dose of 14 PFU (Menendez et al 1975). When hens were experimentally infected with a high dose of reovirus (10,000 PFU) by the tracheal, oesophageal and nasal routes, the egg infection rate was low, with virus found in only four eggs of 226 tested.

Infection of eggs was reported to occur from 8 to 17 days after infection of the bird, but not before or after those times (van der Heide and Kalbac 1975). Because of lack of sterilisation of the surface of eggs, it was not clear whether the infections occurred transovarially or through contamination of the eggs in the cloaca. Also, the rates of infection of eggs could not be ascertained from this report because all testing was done on pools of eggs.

More recently, Al-Muffarej et al (1996) experimentally infected hens with a dose of $5x10^{5.5}$ $TCID_{50}$ of two strains of reovirus, given intravenously and intranasally. Only one infected chick was hatched from the 120 eggs laid by hens infected with a trypsin-sensitive reovirus (strain TR1) whereas six infected chicks and a further 13 infected dead embryos were recovered from the 99 eggs laid by hens infected with a trypsin-resistant reovirus (strain R2).

On the basis that the epidemiology of psittacine reoviruses is likely to be similar to that of reovirus of chickens, the likelihood of entry for reoviruses in the commodity is considered to be very low but non-negligible.

9.2.2. Exposure assessment

Reports of psittacine reovirus infections indicate that it behaves as a contagious disease. Specific information on the mechanisms of spread of psittacine reoviruses is not available but, in chickens, excretion in faeces appears to be the major source of virus and the extent of lateral spread appears to vary between viral strains (Rosenberger 2003).

The exposure assessment is considered to be non-negligible.

9.2.3. Consequence assessment

Although it has been suggested that species may vary in their susceptibility (Conzo et al 2001; Manvell et al 2004; Spenser 1991), reovirus-associated disease has been reported from a wide range of psittacine species. Following introduction of psittacine reovirus to New Zealand, the extent of infection and disease would be dependent upon the degree of contact between infected birds and others. So far as is known, the psittacine population in New Zealand is naïve to reovirus infection and in such a situation the likelihood of infections causing mortality would be high, especially in captive bird populations where stress is likely to increase susceptibility to disease.

The consequence assessment is considered to be non-negligible.

9.2.4. Risk estimation

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

9.3. RISK MANAGEMENT

As avian reovirus is not listed by the OIE, there are no international standards for this virus in birds or eggs.

Reoviruses have been isolated from a small number of cloacal swabs from clinically healthy and diseased psittacine birds during quarantine (Senne et al 1983). Testing of cloacal swabs by electron microscopy or virus isolation are likely to be the most sensitive test available for detecting infected birds.

In experimentally infected chickens, cloacal contamination (as measured by ability to isolate virus from swabs) had peaked and decreased to a very low level prior to the period during which infected eggs were laid (Menendez et al 1975).

The period of time during which reovirus infection could be detected in chicken eggs was restricted to a maximum of 19 days post infection
(Menendez et al 1975; van den Brand et al 2007).

Risk management options include various strategies for reducing the likelihood that birds from which eggs are to be collected are infected with reoviruses. One or a combination of the following options could be considered in order to effectively manage the risk:

Option 1

Cloacal swabs, from birds from which eggs are to be collected, could be cultured for reoviruses or examined by electron microscopy.

Option 2

Establishments from which eggs are to be collected could be required to have had no evidence suggestive of reovirus infection during the preceding 12 months. Examination of cloacal swabs could be undertaken on a monthly basis to demonstrate source flock freedom.

Option 3

Birds from which eggs are to be collected could be required to come from groups/flocks to which there have been no introductions of new birds for a period of time (six weeks is arbitrarily suggested as a suitable period).

Option 4

Prior to the period of egg collection, birds from which eggs will be collected could be isolated from other birds for four weeks. During that period, all birds should have remained clinically healthy.

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10. Proventricular Dilatation Disease (Macaw wasting disease)

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

The aetiology of proventricular dilatation disease (PDD) is remains unknown. For almost 40 years a viral aetiology has been suspected, and in July 2008 the first report appeared implicating an avian Bornavirus (Anonymous 2008; Kistler et al 2008).

10.1.2. OIE list

PDD is not included in the list of diseases notifiable to the OIE

10.1.3. New Zealand status

PDD is not included in the register of unwanted organisms.

Although no published reports of PDD in New Zealand have been located, there is a record of "lympho-plasmacytic infiltrate of the neural and perineural elements (and sometimes smooth muscle) in gizzard, proventriculus and intestines" in tissues from a conure submitted to a diagnostic laboratory in 1996 (Johnstone 2007). The scientific literature regards this pathology as pathognomic for PDD (Ritchie 1995). Although other cases indicative of PDD in New Zealand have not been discovered, this may be explained by the low level of surveillance for diseases of caged psittacines, together with the requirement for very specific samples to be submitted for laboratory examination. There may be parallels with the situation in Australia where a single case of PDD was diagnosed in a legally imported macaw that had been released from quarantine in 1993, and yet by 2007 there was evidence that the disease was widely distributed in caged psittacines in Australia (Doneley et al 2007; Sullivan et al 1997) . However, in the absence of positive evidence of the presence of this disease in this country, it is considered for the purposes of this risk analysis to be exotic to New Zealand.

10.1.4. Epidemiology

PDD was first reported in the late 1970s and early 1980s in macaws and other large psittacines. The characteristic pathology of PDD is a mononuclear cell inflammatory process (lymphoplasmacytic ganglioneuritis) affecting the nerves and ganglia supplying muscles of the proventriculus, crop, ventriculus, and small intestine. Gastric dilatation is the most common cause of clinical signs which may include weight loss, regurgitation, anorexia, lethargy, and death.
Signs consistent with disease of the central nervous system may also be observed.

PDD has now been recognised in at least 50 psittacine species and is as being distributed within the populations of captive psittacines in North America and Europe. Similar diseases have been described in Canada geese (Daoust et al 1991), spoonbills, toucans, canaries, honey creepers, and weaver finches (Gregory et al 1998). PDD has been reported from adult birds more frequently than from juveniles (Gregory et al 1995). No reports of PDD affecting free-living psittacines have been located.

A viral aetiology has been suspected for almost 40 years, and suggested causal agents have included paramyxovirus, polyomavirus, herpesvirus, togavirus, adenovirus, coronavirus, and

eastern equine encephalitis virus. However, the most likely cause of the condition is now considered to be an avian Bornavirus (Anonymous 2008; Kistler et al 2008).

Circumstantial evidence has been summarised that indicates that the incubation period could vary from weeks to years (Ritchie 1995).

Although many of the reports of PDD refer to the disease in isolated cases (Clark 1984; Doneley et al 2007; Gough et al 1996; Lutz and Wilson 1991; Sullivan et al 1997; Vice 1992), situations in which multiple birds within aviaries have been infected are also described (Berhane et al 2001; Clark 1984) and an incident of high morbidity and mortality has been described on a breeding farm in Israel (Lublin et al 2006). In this latter incident, PDD was first recognised about one year after the introduction of a breeding pair of blue and gold macaws (*Ava ararauna*). Rosskopf and Woerpel (1996) described an incident in a purchased bird that died of PDD, which was followed eight months later by the death of four other in-contact birds of three different species.

It has been suggested that egg-borne transmission of PDD may occur (Ritchie 1995) but no evidence was provided while others state that it is unclear whether such transmission takes place (Doneley et al 2007). Some factors to be considered in assessing the likelihood of PDD being transmitted through eggs and the role that this might play in the wider epidemiology of the disease are:

- The occurrence of PDD in five species within a collection of psittacines and within a short period of time (Lublin et al 2006) is consistent with horizontal spread of the disease. With experimental evidence that clinical signs may develop shortly after infection (Gregory et al 1994), there is no need for a proposition of transovarial spread to explain the appearance of disease in birds from 10 weeks of age.
- The majority of cases are in adult rather than young birds.

Although an etiological agent for the disease may have been recently identified as a bornavirus (Kistler et al 2008), the only method of diagnosis remains histological examination of proventriculus, ventriculus, brain and spinal chord. In live animals a biopsy from the crop can be examined histologically. A positive result is of diagnostic value but since the sensitivity of the test may be around 66% a negative diagnosis is not reliable (Ritchie 1995).

10.1.5. Hazard identification conclusion

The disease is likely to be an infectious disease caused by an avian Bornavirus. It is not known whether the virus is transmitted through the egg. Since it is exotic and occurs in countries from which psittacine eggs may be imported, it is regarded as a potential hazard in the commodity.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

The disease is probably an infectious disease and occurs in countries from which psittacine eggs may be imported. Since the incubation period may be long, the importation of infected, subclinically infected birds in the incubation period of the disease could occur. Therefore, the likelihood of entry is considered to be non-negligible.

10.2.2. Exposure assessment

There is circumstantial evidence that the disease can be transmitted between birds and imported birds may be housed with other psittacine birds. Therefore, the likelihood of exposure is considered to be non-negligible.

10.2.3. Consequence assessment

Since large parrots have high economic value and the disease is invariably fatal the introduction of the disease agent is likely to have serious economic consequences for breeders and owners of psittacine birds. It is not a zoonotic disease and there would be no consequences for human health. The effect the introduction of the disease agent could have on native psittacine birds is not known but it is likely that they would be as susceptible as other psittacines. Although contact between wild native psittacines and introduced psittacines is likely to be minimal, it could occur. Therefore, the consequences of introduction of the infectious agent for psittacines kept in captivity and for wild birds that may have contact with introduced psittacines are considered to be non-negligible. If the causative agent were to establish in native psittacine populations, the consequences would be high.

10.2.4. Risk estimation

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

10.3. RISK MANAGEMENT

The OIE *Code* does not include any recommendations relating to proventricular dilatation disease.

Options for effective management of the infectious agent in the commodity should recognise that there are no suitable tests for diagnosis in individual birds and that the incubation period may be protracted.

Following the recent publication of a report implicating avian borna virus as the cause of PDD (Kistler et al 2008) further diagnostic possibilities may become available for this disease/virus at some point in the future.

Risk management options include various strategies for reducing the likelihood that birds from which eggs are to be collected are infected with avian Bornavirus. One or a combination of the following risk management options could be considered in order to effectively manage the risk:

Option 1

Introduction of eggs from birds from flocks with a 4 year history of freedom from the disease (4 years is the maximum suggested incubation period).

Option 2

Histological examination of crop biopsies from a sample of birds in the flock from which eggs are collected.

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11. *Chlamydophila* spp. (ornithosis)

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

The aetiologic agent for avian chlamydiosis is *Chlamydophila psittaci*. Eight serovars, distinguished using monoclonal antibodies and with differences in their predominant host ranges, are recognised (Gilardi et al 1995).

Six serovars (A to E) of *Chlamydophila psittaci* are recognised in birds (Everett et al 1999) :

- Serovar A is endemic in psittacines,
- Serovar B in Columbiformes, with some presence in turkeys,
- Serovar C has been isolated most frequently from Anseriformes with reports also from turkey and partridge,
- Serovar D is most common in turkeys (Phasianiformes) with single isolates being identified from a seagull and a budgerigar,
- Serovar E has been reported from ducks, pigeons, and ratites,
- Serovar F has been reported only from a single psittacine (parakeet).

11.1.2. OIE list

Avian chlamydiosis is included on the OIE list.

11.1.3. New Zealand status

Avian chlamydiosis is not listed in the unwanted organisms register.

Chlamydophila psittaci infection is endemic in psittacine and pigeon populations in New Zealand (Bell and Schroeder 1986; Cairney 1954; McCausland et al 1972). Testing of 54 clinically normal feral pigeons from three distant sites revealed infection at all sites (Motha et al 1995). Infection was found in healthy New Zealand keas shortly after importation into the United Kingdom (Johnson et al 1984). Following a diagnosis of psittacosis in an adult Takahe (*Porphyrio mantelli*) on Mana Island, evidence of infection was found in 73 of 121 faecal samples from captive and wild endangered and threatened native birds. In a follow-up investigation on Kapiti Island inconclusive evidence of chlamydial infection was found in two kaka and three weka (Motha et al 1995).

Given the patterns of host preference of *C. psittaci* serotypes, the evidence of widespread infections of pigeons and psittacines in New Zealand is consistent with the presence of serotypes A (Psittaciformes) and B (Columbiformes) in the New Zealand avian population.

11.1.4. Epidemiology

Documented avian hosts of *Chlamydophila* spp. include nine species of poultry and 460 species of wild and pet birds (Kaleta and Taday 2003).

Serovars C and D are considered the most serious zoonoses, particularly affecting slaughterhouse workers and others in close contact with birds (Andersen 1991; Duan et al 1999; Everett et al 1999; Fukushi et al 1987; Vanrompay et al 1993).

Reports of psittacine isolates from serovars other than A are uncommon, and other than A or B rare. Other reports discovered were:

- one budgerigar from Texas with an infection typed as "serovar – turkey" and likely to have been serovar D (Andersen 1991),
- one Calopsitte budgerigar from Essonne, France, typed as serovar E (Duan et al 1999) and
- a single isolate from a parakeet (location unknown) designated as in serovar F (Everett et al 1999).

11.1.5. Hazard identification conclusion

As they are present in New Zealand, *C. psittaci* serotypes A and B are not classified as potential hazards in the commodity. As they are occasionally found in psittacines, serovars D, E, and F are classified as potential hazards in the commodity.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

The sources of infections of psittacines with chlamydial serovars other than A or B are unknown. However, the fact that they are only single records is consistent with these cases being adventitious infections from other reservoir hosts. In the case of the "serovar – turkey" isolate from a budgerigar in Texas, given the size of the turkey industry in that state, turkeys are the most likely source. Serovar E, which infected the Calopsitte budgerigar in France is uncommon and no particular source can be hypothesised. Similarly, there is no basis for proposing any source for the unique identification of serovar F from a parakeet.

Given the unique status of each report of *C. psittaci* serovars "turkey" (D), E, and F from psittacine birds, the likelihood of their presence in psittacine birds is considered to be negligible.

The entry assessments for *C. psittaci* serovars D, E, and F are considered to be negligible.

11.2.2. Risk estimation

As the entry assessment is negligible, the risk estimate for *C. psittaci* serovars D, E, and F is negligible and they are not classified as hazards in the commodity. Therefore, risk management measures are not justified.

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12. Salmonellae

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

As members of the Enterobacteriaceae, Salmonellae are motile Gram-negative rods that ferment glucose and other sugars and are oxidase negative.

The Salmonella genus contains over 2,400 serotypes. Nomenclature places most Salmonellae of veterinary and public health relevance in the sub-species *Salmonella enterica* subspecies *enterica*. Over 2,300 serotypes fall within this subspecies. The commonly used names (e.g. *Salmonella* Typhimurium) identify serotypes within the *Salmonella enterica enterica* sub-species. Some of these serotypes are further partitioned on the basis of phage type. Most salmonella species are considered to be relatively non-host specific. Nomenclature of *Arizona* spp. or *Salmonella arizonae* has changed over the years but *Salmonella enterica arizonae* and *Salmonella enterica diarizonae* are now considered subspecies within *Salmonella enterica*. *Salmonella enterica arizonae* contains over 300 serotypes.

12.1.2. OIE list

Salmonella serotypes other than *S.* Gallinarum-Pullorum are not included in the OIE list of notifiable diseases.

12.1.3. New Zealand status

S. Gallinarum, *S.* Pullorum, *S.* Abortusovis, *S. arizonae*, *S.* Dublin, *S.* Typhimurium definitive phage type (DT) 104, *S.* Typhimurium DT 44, *S.* Enteritidis phage type (PT) 4, and *Salmonella* spp. (exotic, affecting animals) are listed in the unwanted organisms register.

S. Gallinarum has not been diagnosed in New Zealand and, as a result of an extensive eradication programme operated by the commercial poultry industries, *S.* Pullorum has not been diagnosed since 1985.

S. Abortusovis, *S. arizonae*, *S.* Dublin, and *S.* Typhimurium DT 44 are not present in New Zealand.

S. Typhimurium DT 104 has been isolated relatively infrequently from human and non-human sources in New Zealand (Anonymous 2006).

S. Enteritidis PT 4 is one of the more common *S.* Enteritidis phage types isolated from humans in New Zealand. Isolations from non-human sources in New Zealand are infrequent and there are no records of such isolations during the period 2003 to June 2007 (Anonymous 2007). It is thought that most human infections arise during international travel (Anonymous 1999).

From 1999 to mid 2007, typing of Salmonella isolates from humans in New Zealand yielded over 140 Salmonella serotypes/phage types. During the same period typing of isolates from animals, animal feeds and their environment yielded over 80 serotypes/phage types (Anonymous 2007). As many Salmonella infections are subclinical, the full range of serovars and phage types present in New Zealand and the extent of introductions to the country is unknown.

An epidemic of *S.* Typhimurium DT160, commencing in the winter of 2000, resulted in the death of a large number of sparrows (Alley et al 2002). Infections were also diagnosed in a

small number of psittacines. Two sulphur-crested cockatoos (*Cacatua galerita*) which ate affected sparrows yielded *S*. Typhimurium DT160 and a captive kaka (*Nestor meridionalis*), from a zoological park frequented by sparrows, was also infected. This organism had not been identified in New Zealand prior to isolation from a human in 1998. Introduction to New Zealand with a human carrier was considered a possibility but there was insufficient evidence to draw any firm conclusion. Other reports of salmonellae in psittacine birds in New Zealand have not been located.

12.1.4. Epidemiology

12.1.4.1. *S.* Gallinarum-Pullorum

The natural host for *S.* Gallinarum-Pullorum is chickens. In episodes of infection within flocks both morbidity and mortality can be highly variable and the age group most affected depends upon the pattern of infection within the flock. Transovarian infection does take place and resulting chicks may die in incubators. Clinical signs in adult birds may vary from none to severe with high mortality. Transmission can occur both horizontally and vertically with carrier birds playing an important role in spreading the disease (Shivaprasad 2003).

Reports of isolation of *S*. Gallinarum from two individual psittacine birds have been located (Georgiades and Iordanidis 2002; Liow 1978). Positive serology for *S*. Pullorum was reported in wild and captive blue-fronted Amazon parrots in Bolivia (Deem et al 2005). Natural infections with *S.* Pullorum, of species other than chickens or turkeys, have usually resulted from exposure to infected chickens (Asterino 1996; Snoeyenbos 1991). This proposal is supported by the effective eradication of *S.* Pullorum from New Zealand through the implementation of a programme directed solely at commercial poultry. Although there are very few reports of *S*. Gallinarum-Pullorum infecting psittacines the likelihood of such infection cannot be excluded.

12.1.4.2. *S*. Abortusovis

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

12.1.4.3. *S.* Dublin

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

12.1.4.4. *S. arizonae*

There are very few reports of isolations of *S. arizonae* from avian species other than commercial turkeys and chickens. However, *S. arizonae* has been reported from two individual cases of diseased caged psittacines in the United States (Panigrahy et al 1979) and Spain (Oros et al 1998). The latter case followed shortly after the introduction of Iguanas to the premises. These also became diseased and were found to be infected with the same organism.

12.1.4.5. *S*. Typhimurium PT 44

Few reports of the isolation of *S*. Typhimurium PT 44 have been found. Searches of data from national salmonella surveillance programmes available on the internet revealed reports of *S.* Typhimurium PT 44 from Australia but not from any other country. In the reports discovered (Andrews et al 1997; Anonymous 2005a; Anonymous 2005b; Kirk 2001; Mackie et al 1996;

Oros et al 1998; Sumner 2002), all isolations have been from humans or cattle and they have come from most states in Australia. The numbers of cases per year in both cattle and humans are small. No reports of *S.* Typhimurium PT 44 in birds have been located.

12.1.4.6. *S.* Typhimurium DT 104

This organism has a broad host range including cattle, sheep, goats, pigs, poultry, humans, dogs, cats, horses, and a number of other species (Hogue et al 1997; Rabsch et al 2002; Smith-Palmer et al 2003; Threlfall 2000). The first isolations of the multi-antibiotic-resistant strain (ACSSuT) were from a migratory gull and an imported parrot in 1984, with further isolations from imported exotic birds during 1985 and 1986 (Davies 2001). In Britain, these isolations were followed by an epidemic of multi-resistant DT 104 involving cattle, sheep, pigs, poultry, and other species that peaked in 1996 and has since declined (Anonymous 2003; Davies 2001). Cattle are considered to be the reservoir host.

Twenty two isolates from "non-domestic" birds in the south-east USA were examined and multi-resistant DT104 identified from two captive psittacines originating from the same owner. However, these psittacine isolates were negative for *sefC,* a fimbrial gene found primarily in the avian-adapted salmonellae (Hudson et al 2000).

12.1.4.7. *S.* Enteritidis PT 4

Two reports of *S.* Enteritidis in psittacines have been located. One of those was from Poland with information available limited to an abstract (Wasyl et al 1999), which indicated that a number of birds in the *Psittacula* genus were infected with *S.* Enteritidis of unknown phage type. Two incidents in which birds were infected with *S.* Enteritidis PT 4 have been reported (Orosz et al 1992). One incident involved infection of two lilac-crowned parakeets (*Amazona finschi Schlater*) in a collection of ten psittacines and in the other case a single diseased bird of the same species was infected. In both cases other pathogens were present and the authors were uncertain of the role of the salmonellae in the aetiology of the disease.

12.1.4.8. Other salmonellae

The epidemiology of different Salmonella serotypes follows broadly similar patterns. Spread is mainly via the faecal-oral route, with the organism able to survive for varying periods of time in different environmental niches. Host specificity or host preference varies between Salmonella serotypes. It has been thought that some serotypes, especially *S.* Typhimurium, have very little host preference. This view is being revised with the recognition that genetic determinants are contributing to substantial variations in the breadth of host range for many strains (Hattman et al 1976; Rabsch et al 2002; Tsolis et al 1999).

Relatively few reports of salmonellae in psittacines are available but amongst them are:

- A majority of reports of single (or small numbers of) captive birds being affected in any one incident with some reports including a collection of such incidents (Dorrestein et al 1985; Hudson et al 2000; Panigrahy et al 1984; Sawa et al 1981; Simpson and Euden 1991). The majority of such reports are of *S.* Typhimurium with no phage type specified but there is one report of *S.* Typhimurium PT 36 in a bird with pulmonary disease (Simpson and Euden 1991).
- A 1981 report of large numbers of cases of *S.* Typhimurium U286 infection in African grey parrots (*Psittacus erithacus*) in the United Kingdom (Anonymous 1981). These isolates were from birds developing disease shortly after importation. The author proposed that poor hygiene, poor management, and stress from the time of capture to importation contributed to these cases.

- A report by Shima and Osborne (1989) of an epidemic of *S.* Typhimurium in a collection of rainbow lorikeets (*Trichoglossus haematodes*) and black winged lories (*Eos cyanogenia*), and
- A report by Ward et al (2003) in the United States of an outbreak of *S.* Typhimurium in a zoological collection of 45 Lories and Lorikeets. In neither of these latter two cases was a source for the infection identified.
- A PCR method was used to detect *Salmonella* DNA in 280 captive psittacine birds of 13 species, with 13% being positive. However, no organisms could be isolated from the PCR positive samples (Allgayer et al 2008). Allgayer et al also reviewed literature that indicates that salmonellae have been isolated from both diseased and healthy birds.
- In India, *S.* Saint-Paul was isolated from one of 82 free-flying psittacines sampled (Sharma et al 1980).

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

12.1.5. Hazard identification conclusion

S. Gallinarum-Pullorum, *S. arizonae*, *S.* Typhimurium DT104, *S.* Enteritidis PT 4, and "other salmonellae" are classified as potential hazards in the commodity.

S. Abortusovis, *S.* Dublin, and *S.* Typhimurium PT 44 are not classified as potential hazards in the commodity.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

Vertical transmission of salmonellae infecting poultry can take place either through internal infection in eggs, or through external contamination of egg shells during lay. It is recognised that *S.* Gallinarum-Pullorum may be transmitted in these ways, although transovarial transmission is considered the most important route (Shivaprasad 2003).

Transovarial transmission requires that the organism infects the ovary and/or oviduct of the bird (De Buck et al 2004). That such infections are restricted to only specific Salmonellae was illustrated by artificial infection of chickens using six Salmonella serovars (not including S. Gallinarum-Pullorum) with *S.* Enteritidis being the only serovar resulting in infection of tissues of the reproductive tract (Okamura et al 2001). Infection of eggs following experimental infection of chickens with *S.* Typhimurium DT104 was demonstrated by Williams et al (1998). The Salmonella serovars infecting the reproductive tracts of chickens and turkeys are highly host adapted. However, since there is no evidence relating to psittacines it must be assumed that serovars that can infect chicken eggs can also infect psittacine eggs.

Therefore, the entry assessments for *S.* Gallinarum-Pullorum and *S.* Enteritidis DT4, and *S.* Typhimurium DT104 in the commodity are considered to be non-negligible.

The entry assessments for *S. arizonae*, and "other salmonellae" are considered to be negligible.

12.2.2. Exposure assessment

Birds hatched from imported eggs are likely to have contact with other psittacines, humans and other animals. There may be contact with wild and feral birds through contact with disposed faeces and other materials. The likelihood of exposure of animals and humans is therefore non-negligible.

12.2.3. Consequence assessment

The consequences of introduction of new serovars of *Salmonella* have already been demonstrated by what occurred after the emergence of a new phage type of *S.* Typhimurium DT 160. The organism caused mortalities in sparrows and other birds (including psittacines) and became the most commonly isolated phage type in humans (Alley et al 2002; ESR 2007). Therefore, the consequences of introduction are assessed to be non-negligible.

12.2.4. Risk estimation

Since the entry, exposure, and consequence assessments are non-negligible, the risk estimate for *S.* Enteritidis DT 4, *S.* Typhimurium DT104, and *S.* Gallinarum-Pullorum is non-negligible, and they are classified as hazards in the commodity. Therefore, risk management measures can be justified.

Since the entry assessments for *S. arizonae* and "other salmonellae" are negligible, the risk estimate for these organisms is negligible and they are not classified as hazards in the commodity.

12.3. RISK MANAGEMENT

Although *Salmonella* spp are classified as hazards in the commodity, the likelihood of psittacine donors of eggs being infected with either *S.* Enteritidis DT4, *S.* Typhimurium DT104, or *S.* Gallinarum-Pullorum is low and the likelihood of intact surface sterilised eggs being infected is also very low. Therefore, it could be argued that the likelihood of introducing the organism in the commodity is negligible. This is reflected as one of the options presented.

The OIE *Code* provides recommendations in Article 6.6.3, relating to *S.* Enteritidis and *S.* Typhimurium, for importation of poultry hatching eggs (OIE 2009).

Article 6.6.3.

Veterinary Authorities of *importing countries* should require:

for *hatching eggs*

the presentation of an *international veterinary certificate* attesting that the *hatching eggs*:

1. come from an *establishment* which is regularly monitored for the presence of Salmonella in conformity with the provisions of Chapter 6.4. (see Article 6.4.9.);
2. come from a *flock* of birds within the *establishment* in which no evidence of *Salmonella enteritidis* or *Salmonella typhimurium* has been detected and have had no contact with *hatching eggs* or material from poultry *flocks* which do not comply with this standard;
3. come from an *establishment* which complies with the hygiene and disease security procedures referred to in Chapter 6.4.;

4. were shipped in clean and unused packages.

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

Option 1

Surface sterilised eggs from healthy donors could be imported without restriction.

Option 2

Individual donors could be tested by culture of faeces samples before collection of eggs for export. The birds could then be isolated from contact with other birds until collection of eggs has been completed.

Option 3

A sample of the eggs collected could be sacrificed and cultured for *Salmonella* spp. Any *Salmonella* strains isolated could be fully identified and MAFBNZ could decide whether importation of the eggs should proceed.

Option 4

A sample of birds from the premises from which eggs are to be collected for export could be tested by culture of faeces samples.

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13. *Mycoplasma* spp.

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Mycoplasma spp. are micro-organisms in the class Mollicutes (Quinn et al 2002).

Table 3 summarises the avian *Mycoplasma* spp. and their usual hosts as listed by Kleven (Kelven 2003).

Table 3. Avian *Mycoplasma* spp. and their usual hosts

Mycoplasma sp.	Usual host
M. gallinarum	Chicken
M. gallinaceum	Chicken
M. glycophilium	Chicken
M. iners	Chicken
M. lipofaciens	Chicken
M. pullorum	Chicken
M. gallorale	Chicken
M. synoviae	Chicken, turkey
M. anatis	Duck
M. imitans	Duck, goose, partridge
M. anseris	Goose
M. columbinasale	Pigeon
M. columbinum	Pigeon
M. gallopavonis	Turkey
M. iowae	Turkey
M. meleagridis	Turkey
M. cloacale	Turkey, goose
M. gallisepticum	Turkey, chicken, house finch, other
M. sturni	European starling
M. laidlawii	Various
M. corogypsi	Black vulture
M. buteonis	Buteo hawk
M. gypis	Griffon vulture
M. falconis	Saker falcon

13.1.2. OIE list

M. gallisepticum is listed by the OIE.

13.1.3. New Zealand status

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

M. gallisepticum is endemic in New Zealand (Lohr 1975; McCausland 1972; Pohl 1966).

Positive serology has been reported from routine surveillance for *M. gallisepticum* in chicken and turkeys, *M. synoviae* in chickens, and *M. meleagridis* in turkeys. Clinical disease has been associated with all three *Mycoplasma* species (Anonymous 1994). Reports of other avian *Mycoplasma* spp. in New Zealand have not been located.

The only information discovered on the presence or absence of *Mycoplasma* spp. in native or wild birds in New Zealand is a report of an unidentified *Mycoplasma* sp. isolated from a duck (Hemsley 1996). No evidence of *Mycoplasma* spp. was found in 10 captive Kiwi from four properties (Christensen 1996).

13.1.4. Epidemiology

Clinical presentation of mycoplasmosis varies with host species and differs between *Mycoplasma* species. Many infections are sub-clinical. Each of the *Mycoplasma* spp. appears restricted to a limited host range and most have a host preference for a single species. Spread between birds is by direct or indirect contact and transmission between groups occurs with fomites. Vertical transmission via eggs occurs (Kelven 2003). No reports of human infections with *Mycoplasma* spp. that infect birds have been located.

There is little published information on mycoplasmosis in psittacine birds. Mycoplasmosis is described as a common cause of upper respiratory disease in captive budgerigars and cockatiel flocks (Kelven 2003; Rosskopf and Woerpel 1996; Spira 1996). Many of the diagnoses of mycoplasmosis in these birds are based on response to therapy, as they are resistant to antibiotics that act by disrupting cell wall synthesis (Fudge 1996; Kelven 2003; Spira 1996).

Searches of the scientific literature and the internet have revealed only one specific report of *M. gallisepticum* in psittacines, which was isolated along with *M. iowae*, an unidentified *Mycoplasma* spp., and a number of opportunist pathogens from the respiratory tract of yellow-napped Amazon parrots from a flock in which 200 of 1,100 had died with upper respiratory disease. The authors did not consider that the *Mycoplasma* spp. were primary pathogens in the disease. Following challenge studies in budgerigars and chickens the authors concluded that it was likely that the organism may not establish persistent infections in budgerigars and that it was less pathogenic in this species than was a control strain of *M. gallisepticum* (Bozeman et al 1984).

Other references to Mycoplasma infections in psittacines include

Cases diagnosed as mycoplasmosis in macaws, cockatiels, and cockatoos (details of the bases of these diagnoses are not available) (Gaskin 1987),

M. gallisepticum in five birds. While parrots were included in the study, the information available does not identify whether the parrots were amongst those infected with Mycoplasma (Oladele et al 1999),

Positive serum plate agglutination test results to both M. gallisepticum and M. synoviae in the majority of six clinically healthy “parrots” (Mall et al 1975).

Other studies have failed to isolate Mycoplasma spp. from psittacine birds (Poveda et al 1990; Shimizu et al 1979).

Reports of Mycoplasma infections in psittacines most commonly refer to *M. gallisepticum* which is endemic in New Zealand. Reports discovered, included only one in which *M. gallisepticum* was associated with disease in psittacines and *M. iowae* was also isolated in this case. The authors suggested that the disease was caused by a mixed infection of organisms amongst which the mycoplasmas may have made a contribution (Bozeman et al 1984). It is concluded that *Mycoplasma* infections in psittacines are uncommon and present as upper

respiratory disease. Infections other than *M. gallisepticum* are rare and there is no evidence that psittacines act as reservoir hosts for *Mycoplasma* spp.

13.1.5. Hazard identification conclusion

It is concluded that *Mycoplasma* spp. are not classified as a potential hazard in the commodity.

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14. *Mycobacterium* spp.

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Mycobacterium spp. are aerobic, non-motile, acid-fast bacilli (Fulton and Thoen 2003).

14.1.2. OIE list

M. bovis in bovines and *M. paratuberculosis* in any species are included in the OIE list of notifiable diseases.

14.1.3. New Zealand status

Mycobacterium spp. (exotic strains) are included in the register of unwanted organisms

M. bovis is included in the register of unwanted organisms as "reportable" and is the subject of a national pest management strategy.

M. tuberculosis is endemic in NZ and predominantly a disease of humans. It is a notifiable disease under the provisions of the Tuberculosis Act 1948 administered by the Ministry of Health. Over recent years approximately 350 new cases of human tuberculosis have been diagnosed each year (Anonymous 2006; Hoop et al 1996)

M. avium is endemic in New Zealand birds (De Lisle 1987; Montgomery 1999) and causes some of the tuberculosis infections of deer (De Lisle 1987). Most cases occur in older free range fowls (Black 1997a). Diagnoses of avian tuberculosis in other species have been reported from a captive Kiwi (Davis et al 1984), a harrier hawk (Orr 1995), ostriches (Black 1997b), Fischer lovebirds (Anonymous 1999), and peacocks (Anonymous 2000). Such diagnoses are treated as routine and further laboratory examinations to confirm the specific organism involved is seldom done.

M. genavense has been isolated three times from one human patient in New Zealand. The patient was immuno-compromised, was originally from Africa and had been in New Zealand for 14 years prior to recognition of the infection. The report on this case (Vaughan 2004) states that it is thought that the infection was acquired in New Zealand and that, with the technology now available, further cases of *M. genavense* infection will be recognised in New Zealand in the future.

14.1.4. Epidemiology

The most comprehensive picture of *Mycobacterium* spp. infecting pet birds comes from Hoop et al (1996) who diagnosed mycobacterial infection in 204 birds on the basis of histological findings. They attempted to culture mycobacteria from 110 of these cases with success in 48. Thirty four isolates were identified as *M. genavense*, eight as *M. avium*, two as *M. fortuitum*, two as *M. tuberculosis*, and one each as *M. gordonae* and *M. nonchromogenicum*. The avian orders from which these isolates were obtained were not reported but the difficulty in culturing and identifying mycobacteria from avian lesions which are characteristic of tuberculosis and in which acid-fast organisms are observed is also reported by others (Keymer et al 1982; Portaels et al 1996).

M. genavense has been reported as the cause of granulomatous lesions in birds, including psittacines and other avian species. Most reports are of infection in individual pet birds (Ferrer et al 1997; Hoop et al 1993; Kiehn et al 1996).

M. avium has been diagnosed as the cause of a number of cases of disease in psittacines (Reed and Johnson 1994; Stanz et al 1995).

M. tuberculosis is a contagious disease of humans that usually is spread, predominantly by aerosol, to people in close contact with clinical cases. *M. tuberculosis* has been reported from psittacine birds (Ackerman et al 1974; Hoop 2002; Steinmetz et al 2006; Woerpel and Rosskopf 1984). All reports identified were of individual pet birds with no suggestion of bird to bird spread. One report (Steinmetz et al 2006) was of a green-winged macaw (*Ara chloroptera*) that had been in prolonged close contact with an owner known to be infected with *M. tuberculosis*. Although the sources of infection of many cases of *M. tuberculosis* in psittacines cannot be traced, it is considered that humans are the most likely source (Hoop 2002; Steinmetz et al 2006).

No reports of *M. bovis* infecting birds have been located.

14.1.5. Hazard identification conclusion

Mycobacterium spp. are classified as a potential hazard in the commodity.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

No reports of investigations into the role of vertical transmission of mycobacteria in psittacines have been located, nor have reports of the investigation of the contamination of eggs from infected psittacines been located. The literature contains a number of reports of *M. avium* infection in the eggs of infected chickens (Fulton and Thoen 2003).

The entry assessment for *Mycobacterium* spp. in psittacine eggs is considered to be non-negligible.

14.2.2. Exposure assessment

Chickens hatched from the eggs of hens infected with *M. avium* failed to develop tuberculosis (Fulton and Thoen 2003).

Based on the lack of evidence of vertical transmission of *Mycobacterium* spp. in chickens or other birds, the exposure assessment for *Mycobacterium* spp. in the commodity is considered to be negligible.

14.2.3. Risk estimation

Because the exposure assessment is negligible, the risk estimate for *Mycobacterium* spp. is negligible and they are not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

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