



Australian Government

Biosecurity Australia

Policy Review for the Importation of Preserved Duck Eggs from Taiwan

October 2007

The Australian Government acting through Biosecurity Australia has exercised due care and skill in the preparation and compilation of the information in this publication. Notwithstanding, Biosecurity Australia, its employees and advisers disclaim all liability, including liability for negligence, for any loss, damage, injury, expense or cost incurred by any person as a result of accessing, using or relying upon any of the information in this publication to the maximum extent permitted by law.

Cite this report as:

Biosecurity Australia (2007) *Policy Review for the Importation of Preserved Duck Eggs from Taiwan*.

Biosecurity Australia, Canberra, Australia.

Postal address:

Biosecurity Australia
GPO Box 858
CANBERRA ACT 2601

Internet: www.biosecurityaustralia.gov.au

© Commonwealth of Australia 2007

This work is copyright. Apart from any use as permitted under the *Copyright Act 1968*, no part may be reproduced by any process without prior written permission from the Commonwealth. Requests and inquiries concerning reproduction and rights should be addressed to the Commonwealth Copyright Administration, Attorney General's Department, Robert Garran Offices, National Circuit, Barton, ACT, 2600 or posted at <http://www.ag.gov.au/cca>

Contents

List of tables	iii
List of figures	v
Glossary of terms and abbreviations	vii
Summary	1
1 Introduction	3
2 Background to this review	5
2.1 Proposal to import preserved Taiwanese duck eggs	5
2.2 Scope	5
2.3 Australia's current quarantine policy for the importation of eggs and egg products	6
2.4 Domestic arrangements	8
2.5 Potentially affected Australian industries	8
2.6 Taiwanese poultry industry	11
3 Method	15
3.1 Hazard identification	15
3.2 Risk assessment	16
3.3 Method for risk management	29
4 Hazard identification	33
4.1 Initial hazard list	33
4.2 Disease agents excluded from list	34
4.3 Hazard refinement	34
4.4 Effect of processing on potential hazards	41
4.5 Conclusions: hazard identification	47
5 Risk assessments – salted and heat-treated eggs	51
5.1 <i>Ornithobacterium rhinotracheale</i>	51
6 Risk assessments – alkalised eggs	55
6.1 Notifiable avian influenza viruses	55
6.2 Newcastle disease virus	67
6.3 <i>Salmonella Gallinarum/Salmonella Pullorum</i>	75
6.4 <i>Salmonella</i> Enteritidis/Multi-drug resistant <i>Salmonella</i> Typhimurium	81
6.5 Duck enteritis virus	87
6.6 <i>Ornithobacterium rhinotracheale</i>	95
7 Risk management	97
7.1 Salted and heat-treated eggs	97
7.2 Alkalised eggs	97
8 Quarantine measures	99
8.1 Salted and heat-treated eggs	99
9 Review	101
10 Appendix 1	103
Highly pathogenic notifiable avian influenza (HPNAI)	103
10.1 Risk assessment – alkalised eggs	103

List of Tables

Table 1.	Nomenclature for qualitative likelihoods	17
Table 2.	The assessment of direct or indirect impacts on a national scale ¹	26
Table 3.	A matrix of 'rules' for combining descriptive likelihoods	27
Table 4.	Risk estimation matrix: estimation of the risk of entry and exposure	28
Table 5.	Initial hazard list	33
Table 6.	Hazard refinement	35
Table 7.	Heat susceptibility of disease agents in liquid egg and egg products.....	41
Table 8.	Resistance of agent to physical and chemical action.....	56
Table 9.	Resistance of NDV to physical and chemical action.....	67
Table 10.	Disease agents requiring risk management	97

List of Figures

Figure 1.	Likelihood components of risk assessment	16
Figure 2.	Release pathways for imported preserved eggs from Taiwan.....	19
Figure 3.	Exposure pathways for imported preserved eggs from Taiwan	20

Glossary of Terms and Abbreviations

ABARE	Australian Bureau of Agricultural and Resource Economics
ABS	Australian Bureau of Statistics
ACT	Australian Capital Territory
AI	Avian influenza
AIV	Avian influenza viruses
ALOP	Appropriate level of protection
APMV	Avian paramyxovirus
AQIS	Australian Quarantine and Inspection Service
AUSVETPLAN	Australian Veterinary Emergency Plan
BAA	Biosecurity Australia Advice
Biosecurity Australia	a prescribed Agency within the Australian Government Department of Agriculture, Fisheries and Forestry portfolio
BAPHIQ	Bureau of Animal and Plant Health Inspection and Quarantine
BAPM	Biosecurity Australia Policy Memorandum
bp	Backyard poultry (refers to the low biosecurity poultry exposure group)
CFU	Colony forming units
DAFF	Australian Government Department of Agriculture, Fisheries and Forestry
DEW	Australian Government Department of the Environment and Water Resources
DEV	Duck enteritis virus
DHV	Duck hepatitis virus
DoHA	Australian Government Department of Health and Ageing
DVE	Duck virus enteritis
EHEC	Enterohaemorrhagic <i>Escherichia coli</i>
EID	Egg infectious dose
FAO	Food and Agriculture Organization of the United Nations
FSANZ	Food Standards Australia New Zealand
JE	Japanese encephalitis
HA	Haemagglutinin
HI	Haemagglutination Inhibition
HP	Highly pathogenic
HPNAI	Highly pathogenic notifiable avian influenza
IBDV	Infectious bursal disease virus
ICPI	Intra-cerebral pathogenicity index
IVPI	Intravenous pathogenicity index
ICON	AQIS Import Conditions database
ILT	Avian infectious laryngotracheitis virus
IRA	Import risk analysis
LPAI	Low pathogenicity avian influenza (subtypes other than H5 and H7)
LPNAI	Low pathogenicity notifiable avian influenza (H5 and H7 subtypes)

MD	Marek's disease
NAI	Notifiable avian influenza
NSW	New South Wales
NDV	Newcastle Disease virus
OIE	World Organisation for Animal Health (formerly known as the Office International des Epizooties)
OIE Code	OIE Terrestrial Animal Health Code
PCR	Polymerase chain reaction
PALEES	Partial annual likelihood of entry, exposure, establishment and spread
PAREES	Partial annual risk of entry, exposure, establishment and spread
PFU	plaque forming units
PLE	Partial likelihood of exposure
PLEES	Partial likelihood of entry, exposure, establishment and spread
PLES	Partial likelihood of establishment and spread
SPS	Sanitary and Phytosanitary
SPS Agreement	WTO Agreement on the Application of Sanitary and Phytosanitary Measures
US	United States
vvIBD	Very virulent infectious bursal disease
wb	Wild Birds (refers to the wild birds exposure group)
WNV	West Nile virus
WHO	World Health Organisation
WTO	World Trade Organization

Summary

Quarantine policy currently exists for the importation into Australia of a variety of egg products, and for hatching eggs of domestic ducks, hens and turkeys.

This policy review considers quarantine risks that may be associated with the importation to Australia from Taiwan of two types of traditional preserved duck eggs - salted and heat-treated eggs, and alkalisated eggs. It includes assessment of all potential disease agents that may be introduced to Australia via the importation of these types of duck eggs from Taiwan, and whether the processing they have undergone addresses these risks.

Salted and heat-treated duck eggs are produced from commercial flocks of Tsaiya ducks (*Anas platyrhynchos*). They are processed as follows:

- Shell eggs are washed
- Eggs are soaked for 30 days in saturated saline (20% to 25% salt solution)
- Eggs are pooled and steamed for 45 minutes at 100 °C, to reach a core temperature of 85 °C.

During processing, the internal temperature of the eggs will be at or above 80 °C for approximately 30 minutes when steamed at 100 °C for 45 minutes, and the final temperature of the egg yolk reaches 85 °C. In addition, the egg shell will be directly exposed to the effect of the steam and will heat much more rapidly than material deeper within the egg albumen or yolk. The external surface of the egg is considered to be at or near 100 °C for greater than 30 minutes during the steaming process.

Alkalisated duck eggs are produced from commercial flocks of Tsaiya ducks (*Anas platyrhynchos*) and are processed using the following steps:

- Shell eggs are washed
- Eggs are soaked within the shell for 35 days in alkali solution with a pH of 13, to achieve an internal pH of 9.5 or higher
- Eggs are then pooled, washed, dried and packed.

A draft policy review was issued for public comment on 10 May 2007 and the 60 day consultation period closed on 9 July 2007. Four stakeholders provided comments and the issues raised in submissions have been taken into account in finalising the review.

The policy review concludes that the manufacturing process for salted and heat-treated eggs addresses the diseases of quarantine concern and no additional risk management measures are recommended, beyond ensuring that the manufacturing of the eggs is carried out as advised by Taiwan's Bureau of Animal and Plant Health, Inspection and Quarantine (BAPHIQ), and that post-processing contamination is prevented by good handling practices. To ensure that these prerequisites are met, an import permit will be required, and the product must be accompanied by an official health certificate.

For alkalisated preserved eggs, the following diseases were identified as hazards:

- Notifiable avian influenza viruses

- Newcastle disease virus
- *Salmonella* Pullorum/*Salmonella* Gallinarum
- *Salmonella* Enteritidis /Multidrug resistant *Salmonella* Typhimurium
- Duck enteritis virus
- *Ornithobacterium rhinotracheale*

Further risk assessment concluded that the risk presented by notifiable avian influenza viruses, Newcastle disease virus, and *Salmonella* Enteritidis and multi-drug resistant *S* Typhimurium, exceeded Australia's appropriate level of protection (ALOP). For these disease agents, risk management measures are required. This could include treatment to ensure destruction of the pathogen (such as heating) or measures to decrease the likelihood of entry of the disease, such as country or zone freedom, or flock accreditation. Taiwan has advised that heat treatment is not an acceptable risk management measure for these products. Therefore, risk management will need to be based on country or zone freedom, or flock accreditation, or similar.

Assessment of any of these approaches to risk management needs to be done on a case-by-case basis. A detailed submission will need to be provided by the veterinary authority of Taiwan and Australia will conduct an on-ground assessment of the proposed zone, compartment or flock accreditation scheme.

1 Introduction

Biosecurity Australia is a prescribed Agency within the Australian Government Department of Agriculture, Fisheries and Forestry (DAFF). Biosecurity Australia is responsible for developing quarantine policy for imports and for liaising with overseas veterinary authorities to determine their requirements for exports of Australian animals and animal products.

In late 2000, the BAPHIQ informally enquired about market access for traditional preserved duck eggs into Australia. Formal discussion on access commenced in 2001, and since then Taiwan has provided technical information in support of its request.

Quarantine policy currently exists for the importation into Australia of a variety of egg products, and for hatching eggs of domestic ducks, hens and turkeys. A generic IRA is underway to assess the biosecurity risks of the complete range of edible eggs and egg products. This work has a very broad scope and will continue. A *Technical Issues Paper for the Importation of Non-viable Eggs and Products Containing Egg*¹ was released for stakeholder comment in 2001. Also of relevance is a *Draft Generic Import Risk Analysis Report for Chicken Meat*² (Biosecurity Australia 2006) that was released for stakeholder comment on 28 June 2006.

Biosecurity Australia Policy Memorandum (BAPM) 2006/17 of 21 June 2006 advised stakeholders of the commencement of a policy review of the biosecurity risks associated with the importation of preserved duck eggs from Taiwan. On 10 May 2007, Biosecurity Australia issued a draft policy review for 60 days public comment. Four stakeholders provided comments: Australian Poultry Veterinary Alliance and NSW Department of Primary Industries (NSW DPI); Queensland Biosecurity and BAPHIQ. A range of issues were raised and these have been considered in finalising the policy review. Minor amendments have been made to the policy review although the changes have not altered the risk assessments or conclusions.

As the initial step in the policy review process, Biosecurity Australia identified and categorised hazards potentially associated with traditional preserved duck eggs from Taiwan. Where required, detailed risk assessments of these disease agents were conducted estimating the likelihood of entry, establishment or spread and associated consequences to determine an unrestricted risk estimate for each hazard.

The possible risk management measures were then identified for each disease agent that did not meet the ALOP for Australia. These risk management measures form the basis for recommendations to Australia's Director of Animal and Plant Quarantine for a policy determination. Following the determination, the Australian Quarantine and Inspection Service (AQIS) will take the review into account when considering import permit applications.

This policy review report contains the following:

- background to this review of policy and an outline of Australia's current quarantine policy for the importation of eggs and egg products;
- methodology and results of hazard identification and risk assessment;

¹ Available at: <http://www.biosecurityaustralia.gov.au/>

² Available at: <http://www.biosecurityaustralia.gov.au/>

- recommended risk management measures; and
- quarantine measures.

2 Background to this review

2.1 Proposal to import preserved Taiwanese duck eggs

This policy review is being undertaken in response to a request from the Government of Taiwan for access for traditional Taiwanese preserved duck eggs into Australia. In late 2000, Taiwan requested access for two types of traditional preserved eggs of ducks - salted and heat-treated (cooked) eggs, and alkalised eggs.

Over a number of years, Taiwan has provided technical information to support its access proposal. In February 2006, officers from Biosecurity Australia visited Taiwan for a program of meetings and industry visits. Taiwan has subsequently provided additional information on its animal health status and manufacturing processes. As a result, in June 2006 Biosecurity Australia commenced this review of existing biosecurity policy to consider whether salted and heat-treated (cooked) eggs, and alkalised eggs from Taiwan could be safely imported into Australia. This review was conducted in cooperation with the IRA team that is conducting the chicken meat and egg and egg products import risk analyses.

2.2 Scope

This policy review considers quarantine risks that may be associated with the importation into Australia from Taiwan of two types of traditional preserved duck eggs - salted and heat-treated eggs, and alkalised eggs. It includes assessment of all potential disease agents that may be introduced to Australia via the importation of these types of duck eggs from Taiwan, and whether the processing they have undergone addresses these risks.

This policy review is being conducted in accordance with Australia's rights and obligations under the World Trade Organization's (WTO) Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement).

Under Australian administrative arrangements, Biosecurity Australia provides advice to Australia's Director of Animal and Plant Quarantine in relation to the biosecurity risks to animals and plants associated with the importation of animal and plant products. Biosecurity Australia consulted with the Australian Government Department of Health and Ageing (DoHA) and Food Standards Australia New Zealand (FSANZ) on public health issues and with the Australian Government Department of Environment and Water Resources (DEW) on environmental issues associated with the importation of Taiwanese preserved duck eggs, during the preparation of this policy review report. Products intended for human consumption may undergo a separate risk assessment by FSANZ to determine public health risks.

The two types of traditional Taiwanese preserved duck eggs are defined as follows:

2.2.1 Salted and heat-treated duck eggs

Salted and heat-treated duck eggs are produced from commercial flocks of Tsaiya ducks (*Anas platyrhynchos*). They are processed as follows:

- Shell eggs are washed

- Eggs are soaked for 30 days in saturated saline (20% to 25% salt solution)
- Eggs are pooled and steamed for 45 minutes at 100 °C, to reach a core temperature of 85 °C.

Washing and salting will have little effect on the survival of any pathogens which may be in or on the raw material. However, heating in steam will have a significant effect on pathogen survival.

Grijpspeerdt and Herman (2003) demonstrated that when eggs are placed in boiling water, the temperature of the egg yolk reaches approximately 80 °C within 10-12 minutes. The rate of heating of eggs in steam can be assumed to be similar, indicating that the temperature of egg yolks during processing will be at or above 80 °C for approximately 30 minutes when steamed at 100 °C for 45 minutes. This is consistent with the final temperature of the egg yolk reaching 85 °C as indicated by BAPHIQ. The effectiveness of this heat treatment as a risk management measure will be discussed further as part of the hazard identification stage and in individual risk assessments.

2.2.2 Alkalised duck eggs

Alkalised duck eggs are produced from commercial flocks of Tsaiya ducks (*Anas platyrhynchos*) and are processed using the following steps:

- Shell eggs are washed
- Eggs are soaked within the shell for 35 days in alkali solution with a pH of 13, to achieve an internal pH of 9.5 or higher
- Eggs are then pooled, washed, dried and packed.

As is the case for salted and heat-treated eggs, washing will have little effect on the survival of any pathogens which may be in or on the raw material. The pH may have an effect on some pathogens, but the effect of pH will vary from organism to organism. The effectiveness of pH treatment as a risk management measure will be discussed further as part of the hazard identification stage and in individual risk assessments.

2.3 Australia's current quarantine policy for the importation of eggs and egg products

For human consumption, a wide variety of egg-based commodities containing less than 10% egg content can be imported without an import permit but with a manufacturer's declaration stating that the product contains less than 10% egg ingredients (by dry weight) and contains no discernible pieces of egg.

Import requirements for products containing more than 10% egg have been developed for commodities such as egg powders, mayonnaise, moon cakes, noodles and cake mixes. Eggs and egg products can also be imported in some pet foods and for laboratory use with an import permit and subject to conditions. Commercially prepared and packaged, cooked cakes, biscuits or bread (but not mooncakes or cheese cakes) are permitted entry with egg content greater than 10% with an import permit.

Egg shell ornaments do not require permits, but are subject to inspection and must be accompanied by documentation showing any processing, or can be treated on arrival.

Specific import requirements for some types of eggs and products containing eggs are:

- Egg pasta/noodles with less than 20% egg content – the pasta/noodles must be cooked by a process sufficient to raise the core temperature to:
 - 87 °C for 2 minutes and 30 seconds
 - 75 °C for 15 minutes
 - 60 °C for 5 hours or
 - 60 °C for 30 minutes followed by 54 °C for 5 hours
- Mooncakes containing egg – eggs must be salted in a solution of 1 kg salt per 2 litres of water for a period of not less than 20 days, then the egg yolk must be removed from the eggs and oven cooked at 180 °C for a period of not less than 15 minutes, with the cooked yolks and other ingredients baked at not less than 180 °C for a period of not less than 30 minutes
- Pasteurised egg products from New Zealand – the following time and temperature parameters must be met:
 - liquid whole eggs – 64 °C for a minimum of 2.5 minutes
 - liquid egg yolk –
 - : 60 °C for a minimum of 3.5 minutes or
 - : 60.5 °C for a minimum of 3 minutes
 - egg whites
 - : 55.5 °C for a minimum of 9.5 minutes
- Whole boiled eggs from New Zealand – eggs must attain a minimum core temperature of 80 °C or they must be cooked in water where the water is maintained at a temperature of at least 97 °C for at least 17 minutes
- Egg waffles – must be baked at 250 °C for at least 140 seconds
- Spray dried egg white/albumin – product must be spray dried and then ‘hot boxed’ in its final packaging to a minimum core temperature of 70 °C for 7 days or 62 °C for 10 days. The product must not be distributed, sold or used for veterinary purposes including stock feed, veterinary therapeutic or vaccine manufacture or environmental use associated with livestock
- Whole spray dried whole egg or egg yolk products from Canada, Denmark, the United States (US) and Belgium must be heated to a minimum core temperature of not less than 70 °C for 120 minutes.
- Hermetically sealed canned and retorted egg products are permitted entry provided:
 - the final product is canned or retorted to a minimum core temperature of 100°C, and obtaining an F₀ value of at least 2.8, and/or
 - the final product is in a hermetically sealed (airtight) container and has been heat-treated (retorted within this container so that the final product is shelf stable (not requiring refrigeration); and
 - the final product contains egg of avian origin only.

2.4 Domestic arrangements

The Australian Government is responsible for regulating the movement of animals and their products into and out of Australia, but the State and Territory governments have primary responsibility for animal health controls within their jurisdictions. Legislation relating to resource management or animal health may be used by State and Territory governments to control interstate movement of animals and their products.

Egg products may move freely in trade between all States and Territories within Australia. Restrictions have existed from time to time, due to outbreaks of exotic disease such as virulent Newcastle disease (in certain areas in New South Wales (NSW) between 1998 and 2002 and in Victoria in 2002), or avian influenza (most recently in NSW in 1997). These outbreaks were managed by stamping out or, in the case of Newcastle disease, stamping out and vaccination.

2.5 Potentially affected Australian industries

2.5.1 The Australian duck industry

The duck egg industry

The duck egg industry is small and produces for specialist outlets. There are six commercial enterprises with laying ducks. The enterprises also raise broilers or breeding stock and are situated in Queensland, NSW and Victoria. The producers have farm level biosecurity plans.

Chinese and other Asian restaurants provide the biggest market for duck eggs. Eggs are marketed as fresh, salted fresh, pickled and pickled salted as well as balut eggs, which are fertile eggs that have been incubated for about 18 days and are sold for eating after boiling.

Duck eggs also provide an alternative for people who are allergic to chicken protein.

Most producers of duck eggs send the eggs to specialist processors close to the main markets in Sydney (NSW) and Melbourne (Victoria).

The duck meat industry

Duck production occurs in most States, with two companies producing most of the Pekin-type duck for the restaurant and hospitality sectors.

All Australian States have a small game-bird industry, with NSW and Victoria being the largest production centres. The majority of Australia's game bird population, including duck, turkey, quail, squab, guinea fowl, pheasant, partridge and geese, are processed in domestic processing plants.

2.5.2 Other potentially affected Australian animal industries

The Australian egg industry

The egg industry in Australia is dominated by egg production from the domestic chicken. However, there are niche markets for duck, goose, quail and pigeon eggs. In contrast with the

chicken egg industry, non-chicken eggs are produced by small operators or as a sideline to a meat industry.

There is a trend away from the sale of eggs in shell through retail outlets towards the processed food and food service sectors. There is also a trend to increased egg products manufacturing. An estimated 65% of eggs are sold in shell form through supermarkets and other retail outlets, 20% are sold to the food service sector and 15% are transformed into manufactured egg products (Australian Egg Corporation Limited 2005). Egg products with an estimated value of \$6.542m were imported in 2005 (Australian Egg Corporation Limited 2006)

Processed egg products make up 15% of market share (Australian Egg Corporation Limited 2005). Liquid, frozen and spray dried products are manufactured at processing plants. These products may be in whole or separated form. Small volumes of specialised egg products are also manufactured. There are five major plants in Australia located at Newcastle and Griffith (NSW), Melbourne (Victoria), Perth (Western Australia), and Adelaide (South Australia).

A relatively small proportion of shell eggs and egg products is exported.

Domestic retail sales of shell eggs were estimated at \$199.3 million between 10 July 2005 and 8 July 2006. The value of exports (shell egg and egg products) was \$2.264m in 2005 (Australian Egg Corporation Limited 2006).

The Australian chicken meat industry

The chicken meat industry produces a range of fresh, frozen and cooked products. Raw products in the form of fresh and frozen whole birds account for 50% of the market. Raw value-added products in the form of cut up chickens – breasts, legs, thighs and other specialty lines are produced both bone-in and de-boned for a total market share of 30%. Other value-added products include ready-to-cook and fully cooked products and represent the remaining 20% of the market (Jeff Fairbrother, Australian Chicken Meat Federation, personal communication 2002; Andreas Dubs, Australian Chicken Meat Federation, personal communication 2005).

The industry supplies its products to four market segments:

- supermarkets 40%
- take away outlets 25%
- food service industry 25%
- other 10%

The food service industry includes restaurants, hotels, caterers, hospitals, armed services, canteens and similar type operations. The others category includes butcher shops, specialty chicken shops, and other small retail outlets.

Australian poultry meat production was 817 kilotonnes in 2005 (ABARE, 2007). The Australian Bureau of Agriculture and Resource Economics (ABARE) estimate for production in 2007-08 is 847 kilotonnes (ABARE 2007). In Australia, chicken meat consumption per person is projected to be 39.1 kg per person in 2007-2008 (ABARE, 2007).

In 2000/01, exports accounted for only 1.8% of turnover in the chicken meat industry (Anonymous 2002), with some export markets closing in response to Newcastle disease outbreaks in NSW and Victoria in 1998, and 2000. Poultry meat exports were forecast to

reach 22.0 kilotonnes in 2007-08 (ABARE 2007). Major export markets are Hong Kong/China, South Africa and the Pacific Island nations.

There is a growing export market for Australian breeding stock. Potential growth of this market, however, will depend, among other things, on the continued absence of major poultry diseases in the Australian breeder flock.

Turkeys and other game birds

Approximately 17 million game birds were processed for meat in Australia in 2001/02, with duck, quail and turkey accounting for 95%. Duck production occurs in most States, with two companies producing most of the Pekin type duck for the restaurant and hospitality sectors. The majority (77%) of Australia's 4.7 million turkeys are produced by large, vertically-integrated chicken meat companies, with the remainder being produced by large independent growers or smaller producers in each state. A single NSW company accounts for about 75% of the 6.5 million quail processed in Australia each year, with smaller producers in NSW, Victoria and South Australia. Squab producers are located in Queensland, NSW and Victoria, while pheasant, guinea fowl, partridge and geese producers are concentrated mainly in NSW and Victoria (Leech et al. 2003).

The retail value of the game bird market was estimated at \$290 million per year (Leech et al. 2003). Export markets have been severely compromised by outbreaks of Newcastle disease in NSW and Victoria in recent years.

Ostrich industry

While relatively small compared with the chicken meat and egg industries, the Australian ostrich industry has grown in recent years. Significant export markets had been developed for ostrich meat, before restrictions on access due to outbreaks of Newcastle disease in NSW and Victoria. Australia is now recognised by OIE as free of this disease.

Pigeons

While it is not a large or well-organised industry in Australia, there are a number of individuals who have put considerable resources into developing international markets for racing and show pigeons. Restrictions on exports from NSW and Victoria due to outbreaks of Newcastle disease have caused financial losses to some pigeon breeders.

Avicultural community and zoological gardens

The aviculture community in Australia covers a wide spectrum of the population, from individuals with a single pet bird, to commercial enterprises worth millions of dollars. The most recent available figures from the Australian Bureau of Statistics (ABS) on pet ownership in Australia (1994) indicate that 16% of households in Australia keep pet birds, with 35% of bird-owners keeping three or more birds (Australian Bureau of Statistics 1995).

Zoological gardens keep a wide range of avian species, many of which are of great conservation and commercial value, including some which are listed as endangered species.

Native birds and the environment

Australia has significant populations of native birds, many of which do not occur naturally

elsewhere. The conservation value of native birds is extremely high, but is difficult to measure. Some of Australia's native species have been shown by overseas experience to be susceptible to major exotic diseases of poultry. The potential effects of an outbreak of an exotic disease in our wild bird populations are difficult to estimate.

2.6 Taiwanese poultry industry

2.6.1 Structure of the industry

Most duck farms are situated in southern Taiwan. Poultry and pigs are also located in this area. There are approximately 1,000 duck farms and most farms have an average of 3,000 to 5,000 adult ducks. Farms have introduced general biosecurity measures and commenced protecting duck ponds with nets to minimise the risk of introduction of highly pathogenic notifiable avian influenza (HPNAI). Taiwan has imported day-old ducks from Great Britain and France. A total of 7,459 day-old ducks were imported from Great Britain in 2003 and 2006, and 5,382 birds were imported from France in 2005.

BAPHIQ advised in 2006 that eight premises currently produce preserved duck eggs in Taiwan. Of these establishments, two processors intend to export preserved duck eggs to Australia. The first company operates in a premises constructed in 2005, and mainly produces alkalised eggs. Twenty thousand eggs are processed daily, with eggs collected from five farms every three to five days. The company follows standard operating procedures dealing with hygiene. A government agency conducts microbiological testing on a monthly basis. Documentation and labelling enables recall of product if necessary.

The second company also uses a newly established factory, although it also uses other premises to produce preserved eggs. Ten farms supply this company and a veterinarian is engaged to check the health of ducks on farms. All eggs are washed off-site. Sixty thousand salted and heat-treated eggs and 750,000 alkalised eggs are produced monthly. The company also produces flavoured and coloured eggs and egg yolk (300,000 per month). A government agency tests monthly for *Salmonella* spp., bacterial count, and residues including antibiotics (chloramphenicol, penicillin, chlortetracycline, oxytetracycline, tylosin, erythromycin, sulphamezathine, sulphamonomethoxine), lead and copper. Batch number and date are recorded on packaging, for recall of product if necessary. A quality control program is operating at this factory. Product is distributed to wholesalers (50%), retailers and traditional markets (30%), export to US, Japan, Canada and South Africa (10%) and the remainder to other outlets such as restaurants and schools for lunches.

The Provincial Municipalities and Counties – Public Health Bureau and Municipalities – Department of Health inspects facilities regularly to ensure compliance with sanitary standards. Where companies request approval to export, BAPHIQ checks requirements of the importing country and requests the Bureau of Health of Local Government to inspect the premises and report against importing country requirements.

2.6.2 Avian health status

Taiwan has active and passive disease surveillance systems where the active surveillance system concentrates on exotic disease pathogens. BAPHIQ, Animal Health Research Institute and Livestock Disease Control Centers (LDCCs) work cooperatively to conduct national surveillance and sampling according to the OIE Terrestrial Animal Health Code.

Taiwan claims country freedom from HPNAI and duck enteritis virus (DEV).

To support its claim of freedom from HPNAI, Taiwan has provided information about its active national surveillance program of HPNAI that has been conducted since 1998. The surveillance is based on serum and swab samples from poultry farms. In 2005, 113 duck farms, 68 goose farms and 661 chicken farms were sampled for surveillance. In addition, 4,506 faeces samples from migratory birds were also tested for evidence of virus. No HPNAI virus has been isolated. However, some cases of low pathogenic avian influenza (LPAI) have been recorded. In a letter of May 2005, Taiwan stated that it had had some cases of LPAI between January and March 2004, although Taiwan did not indicate that LPAI were reported in ducks.

For DEV, a serological survey in ducks and geese was conducted in the years 2000 and 2001. In total, 5,203 samples were tested and no antibody against DEV was found in the two-year survey. Since then, surveillance for DEV has been passive and is based on ruling out suspect cases. No cases have been detected. Australia considers that further, more recent data would be necessary to provide confidence of complete freedom from this disease agent. However, it is accepted that if the disease is present the prevalence is extremely low, since population densities in concentrated duck-producing areas result in rapid spread of DEV, and it would be expected to spread rapidly once introduced to a laying flock.

Taiwan relies on passive surveillance system for non-exotic diseases such as Newcastle disease (ND), infectious bursal disease and LPAI, in which the farmers and local veterinarians are required to notify LDCCs of any suspicious cases. LDCCs then conduct investigations into the cases and report their findings to BAPHIQ through the Disease Notification System. Taiwan has stated that very virulent infectious bursal disease (vvIBD), ND and duck viral hepatitis do occur in Taiwan, but that IBD and ND have never been reported in ducks. However, ducks can be infected with IBD and not exhibit clinical signs. Water birds are also the least likely to show clinical signs of infection with NDV. In addition, ducks can be infected with LPAI and not exhibit clinical signs, or only display mild respiratory and reproductive disease. Biosecurity Australia considered that these diseases may exist in ducks in Taiwan but may have not been reported to LDCCs by farmers and local veterinarians given the asymptomatic nature of these diseases in ducks. Vaccines for duck viral hepatitis and goose parvovirus are available but their use is not mandatory.

The National Animal Industrial Foundation conducted monitoring for *Salmonella* spp in preserved (salted/heated and alkalisied) duck eggs from retail markets in 2003 and 2004, with negative results.

2.6.3 Reference list

1. ABARE. 2007. *Australian Commodities 7 March Quarter 2007*. Web page, [accessed April 2007]. Available at http://www.abareconomics.com/interactive/ac_mar07/htm/meat.htm
2. Agriculture and Resource Management Council of Australia and New Zealand. 1996. *AUSVETPLAN: Enterprise Manual Poultry Industry*, Canberra, Australia.
3. Anonymous. 2002. *Poultry Farming (Meat) in Australia*, IBISWorld Pty Ltd. A0141.
4. Australian Bureau of Statistics. 1995. "Australian Social Trends 1995 – Culture and Leisure Special Feature: Household Pets". Web page, [accessed 2003]. Available at <http://www.abs.gov.au/ausstats/abs@.nsf/0/AF01B3D0CA8D9C9DCA2569EE0015D8CA?Open&Highlight=0,pets>.
5. Australian Chicken Meat Federation. 2005. "*Chicken Meat Industry*". Web page, [accessed March 2006]. Available at <http://www.chicken.org.au/page.php?id=37>.
6. Australian Egg Corporation Limited, Editors. 2006. "Australian Egg Industry Overview". Web page, [accessed April 2007]. Available at [http://www.aecl.org/Images/2006%20egg%20industry%20statistics%20\(2\).pdf](http://www.aecl.org/Images/2006%20egg%20industry%20statistics%20(2).pdf)
7. Australian Egg Corporation Limited, David Witcombe (david@aecl.org). 7 October 2005. "Egg Products". E-mail to Kathy Gibson (Kathy.gibson@daff.gov.au).
8. Dubs, A. 2005. Chicken meat and egg industries: an overview.
Notes: Presentation given by A. Dubs, Executive Director of the Australian Chicken Meat Federation, Exercise Hermes, May 2005, Sydney, Australia
9. Fairbrother, J. 2004. Future directions for Australian chicken meat. In *Outlook 2004 Intensive Livestock Session: Speakers Papers* Canberra, Australia: ABARE.
Notes: ABARE product code 12691
10. Food Regulation Standing Committee. 2006. *Australian Standard for Construction of Premises and Hygienic Production of Poultry Meat for Human Consumption*, Australia and New Zealand Food Regulation Ministerial Council. FRSC Technical Report No. 1 AS 4465:2005. CSIRO Publishing, Victoria, Australia.
11. Gilchrist, P. 2005. Involvement of free-flying wild birds in the spread of the viruses of avian influenza, Newcastle Disease and infectious bursal disease from poultry products to commercial poultry. *World's Poultry Science Journal* 61: 198-214.
12. Larkin, J. T., S. G. Heilbron, T. Murphy, and P. Bradbery. 2000. "Report for 1999–2000". *Benchmarking and Value Chain On-going study program for the Australian Chicken Meat Federation*, J.T.Larkin & Associates; S.G. Heilbron Pty Ltd, Canberra, Australia.
13. Larkin, J. T., S. G. Heilbron, T. Murphy, and P. Bradbery. 2001. "Year 2 Report: Part 1– Value Chain Research". *Benchmarking and Value Chain On-going study program for the Australian Chicken Meat Federation*, J.T.Larkin & Associates; S.G. Heilbron Pty Ltd, Canberra, Australia.
14. Leech, A., P. Shannon, P. Kent, G. Runge, and B. Warfield. 2003. *Opportunities for exporting game birds, RIRDC Publication No 03/106*. Rural Industries Research Development Corporation, Canberra, Australia.
15. Primary Industries Ministerial Council, 2002. "Primary Industries Ministerial Council. Record and Resolutions. 1st Meeting, Hobart, 2 May 2002" Web page, [accessed May 2006]. Available at http://www.mincos.gov.au/pdf/pimc_res_01.pdf

16. USDA-FAS, 2006. "Livestock and poultry: World markets and trade, March 2006". Web page, [accessed June 2006]. Available at http://www.fas.usda.gov/dlp/circular/2006/06-03LP/poultry_sum.pdf

3 Method

This policy review is conducted according to the principles outlined in the OIE Terrestrial Animal Health Code (OIE Code) for undertaking risk analysis. The risk analysis process consists of a number of steps. These are

- Hazard identification
- Risk assessment, incorporating
 - Release assessment
 - Exposure assessment
 - Consequence assessment
 - Risk estimation
- Risk communication

This policy review report incorporates the consultation step with release of the draft policy review report in May 2007 and together with the consideration of stakeholder comments, form part of the risk communication process.

3.1 Hazard identification

Hazard identification is defined in the OIE Code as the process of identifying the pathogenic agents that could potentially produce adverse consequences if introduced in an imported commodity. Hazard identification is a classification step, identifying pathogenic agents as potential hazards or not.

The OIE Code states that, to be identified as a potential hazard, a pathogenic agent:

- should be appropriate to the animal species to be imported, or from which the commodity is derived;
- may be present in the exporting country; and
- should not be present in the importing country. If present, the pathogenic agent should be associated with a notifiable disease, or is subject to an official control or eradication program.

In this policy review, hazard identification was initiated by generating a preliminary list of potential pathogenic agents, or 'potential hazards'. The list consisted of pathogenic agents associated with each of the OIE-listed diseases, and those other diseases relevant to the importation of whole duck eggs. The review considered the diseases listed in the *Technical Issues Paper for the Importation of Non-viable Eggs and Products Containing Egg* (Animal Biosecurity Policy Memorandum 2001/01)³, *Conditions for the importation from approved countries of fertile eggs (domestic duck)* (BAPM 2006/25), and the *Draft Generic Import Risk Analysis Report for Chicken Meat* (BAPM 2006/18)⁴. The list was refined by applying the criteria stated above to each disease agent, and was further modified by considering whether the disease agent was likely to be present in or on eggs laid by infected ducks. As a final step in the hazard identification, the effect of the processing of the product on the survival of the

³ Available at: <http://www.biosecurityaustralia.gov.au/>

⁴ Available at: <http://www.biosecurityaustralia.gov.au/>

disease agents was considered. If reasons for the inclusion or exclusion of particular pathogenic agents were not clear-cut, these agents were retained on the list and were examined further in the risk assessment.

3.2 Risk assessment

Risk assessment is defined in the OIE Code as:

‘an evaluation of the likelihood and the biological and economic consequences of entry, establishment or spread of a pathogenic agent within the territory of an importing country.’

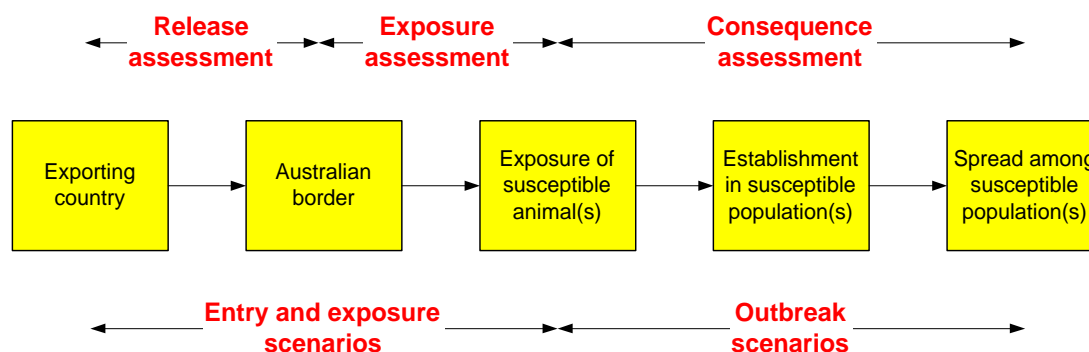
The likelihood that a pathogenic agent will enter an importing country, and the likelihood that susceptible animals will be exposed to, and infected by, that agent, are determined through a ‘release assessment’ and an ‘exposure assessment’, respectively.

The likelihood of establishment and spread, and the biological and economic consequences of introducing a pathogenic agent, are determined through a ‘consequence assessment’.

The risk assessment for each identified agent concludes with ‘risk estimation’ - the combination of the likelihoods and consequences - and yields the ‘unrestricted risk estimate’.

These steps are illustrated diagrammatically in Figure 1.

Figure 1. Likelihood components of risk assessment



Evaluating and reporting likelihood

This policy review uses a qualitative approach. The likelihood (or probability) that an event will occur was evaluated and reported qualitatively, using qualitative likelihood descriptors (Table 1).

Critical to the release, exposure and consequence assessments was an evaluation of the various pathways through which a disease agent entering Australia in a shipment of preserved duck eggs could enter Australia, be exposed to susceptible Australian animals, become established, and spread to a wider population of susceptible animals.

Table 1. Nomenclature for qualitative likelihoods

Likelihood	Descriptive definition
High	The event would be very likely to occur
Moderate	The event would occur with an even probability
Low	The event would be unlikely to occur
Very low	The event would be very unlikely to occur
Extremely low	The event would be extremely unlikely to occur
Negligible	The event would almost certainly not occur

In performing such an evaluation, likelihoods as defined above were assigned to release, exposure and outbreak scenarios as a whole. Likelihoods were not ascribed to individual pathway steps that make up each scenario. For example, in the exposure assessment, there was a general examination of the pathways whereby a susceptible host in an exposure group may become exposed to an agent before assigning an overall likelihood to this outcome.

Volume of trade

This policy review examines the likelihood of entry and exposure of a hazard over a period of a year. Therefore, the release and exposure assessments for each hazard are based on estimated annual volume of trade in preserved duck eggs.

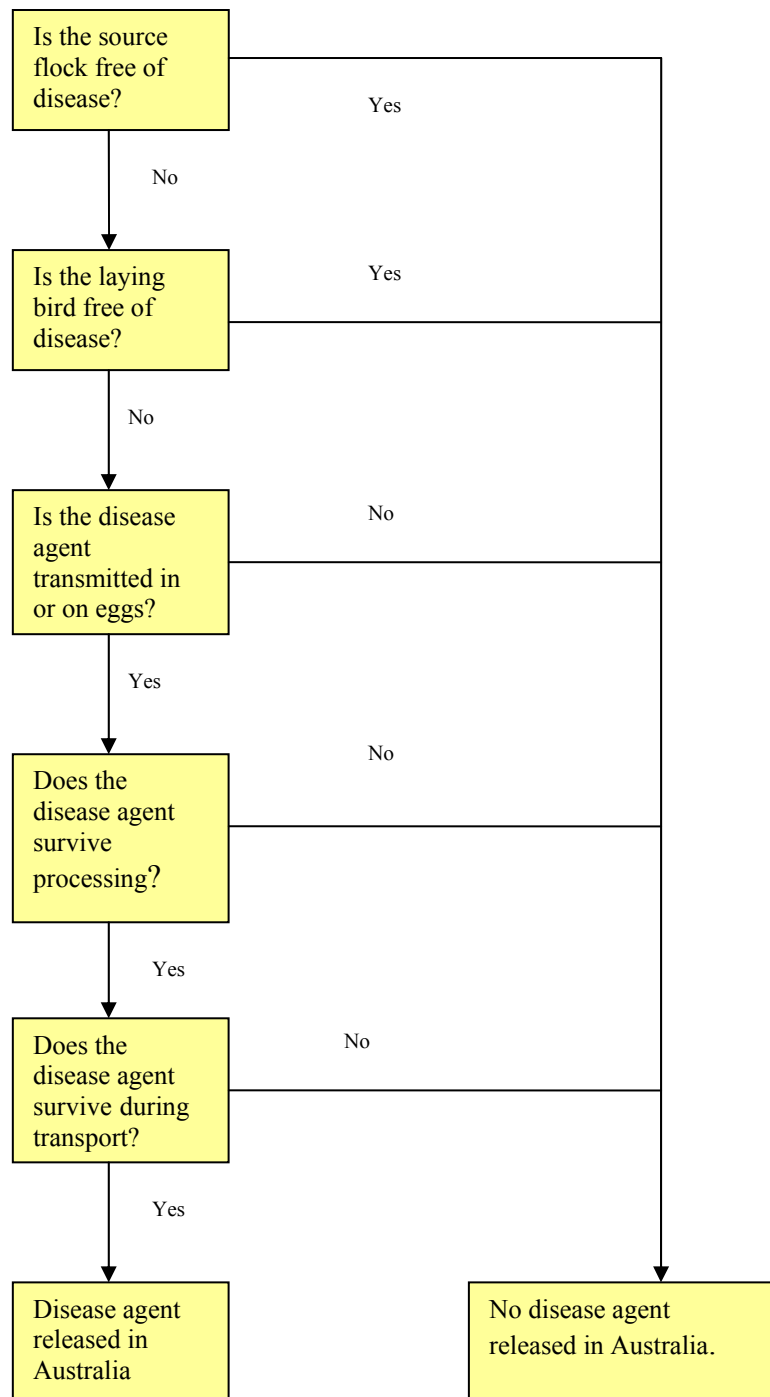
As this is a new trade, it is difficult to estimate accurately the likely annual trade volume. However, imports of retorted preserved eggs have been occurring, in accordance with existing quarantine conditions for these products. Over the period October 2004 to September 2006, a total of approximately 170,000 kilograms of retorted preserved eggs were imported from China. Based on tariff code information, approximately 130,000 kilograms of these appear to be retorted, salted and heat-treated, preserved eggs, leaving approximately 40,000 kilograms of retorted, alkalised eggs, or 65,000 and 20,000 kilograms per year of retorted salted and alkalised eggs, respectively. Assuming an individual duck egg weighs about 85 grams, this equates to an approximate trade volume of 765,000 retorted, salted and heat-treated, preserved eggs, and 235,000 retorted, alkalised, preserved eggs per year. Given the nature of the product, it is considered that there may be a small increase in the total market following the import of preserved eggs from Taiwan. It is possible that preserved eggs from Taiwan might displace some of the retorted preserved egg market from China as consumers may prefer preserved duck eggs imported from Taiwan to those imported from China, which are retorted. Therefore, the policy review estimated that the annual volume of trade in salted/heat-treated eggs and alkalised eggs from Taiwan to be approximately 380,000 and 120,000 eggs, respectively.

3.2.1 Release assessment

The release assessment considered a single release scenario – the importation from Taiwan of preserved duck eggs (both alkalised and salted and heat-treated) for human consumption. The following factors were taken into account in determining the annual likelihood of a disease agent entering Australia in a consignment of preserved eggs:

- between-flock prevalence
- within-flock prevalence
- transmission of the disease agent within or on the egg
- effect of processing on inactivation of the disease
- volume of trade.

This likelihood was termed the ‘annual likelihood of release’ (LR). A flow chart describing the release pathway is shown at Figure 2.

Figure 2. Release pathways for imported preserved eggs from Taiwan

3.2.2 Exposure assessment

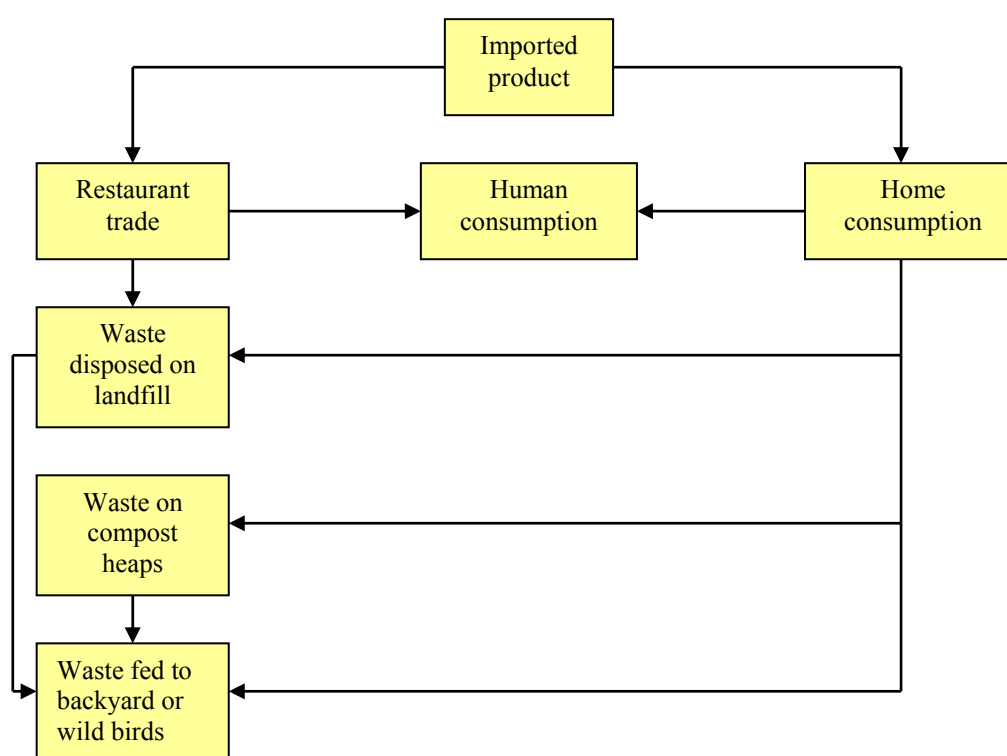
The exposure assessment considered the key distribution pathways and end-uses that could lead to each of the identified exposure groups coming into contact with each pathogenic agent of concern. The exposure groups were:

- Low biosecurity backyard poultry (bp), which are at risk of direct exposure through the feeding of kitchen scraps; and

- Wild birds (wb), which are at risk of direct exposure through scavenging waste at refuse dumps, or through exposure to wastes fed to backyard poultry or disposed of on compost heaps. It is recognised that there is a possibility of direct exposure of wild birds through inappropriate disposal of picnic waste. However, Biosecurity Australia considered that the likelihood of this is significantly less than for the exposure of wild birds to wastes either placed out as feed for backyard poultry or discarded on backyard compost heaps.

A flow chart describing the major exposure pathways is shown at Figure 3.

Figure 3. Exposure pathways for imported preserved eggs from Taiwan



Biosecurity Australia considers that the most likely waste material arising from imported preserved eggs will be fragments of shell. However, it is recognised that there may be some instances where portions of the egg itself will be discarded. Shell fragments will have been directly exposed to the effect of the processing, (either heating or alkalising) and so will represent a different, generally lower, risk level than will discarded portions of egg white or yolk.

The proportion of households that keep backyard poultry, and the proportion of those households that would feed scraps to their poultry determined the likelihood of exposure of low biosecurity poultry to kitchen scraps. At the last estimate, the proportion of households keeping backyard poultry was 6–7% (Agriculture and Resource Management Council of Australia and New Zealand 1996). This proportion was taken into account when assessing the likelihood of direct exposure of backyard birds to waste derived from imported preserved eggs.

For each pathogenic agent of concern, the final outcome of the exposure assessment was an estimation of the partial likelihood of exposure (PLE) for each exposure group (PLEbp (backyard poultry) and PLEwb (wild birds)). The PLE represents the likelihood that a domestic population of a susceptible host species is exposed to a pathogenic agent of concern through contact with imported preserved eggs (or associated wastes) that are infected or contaminated. Estimation of PLE took into consideration, inter alia, the relative volumes of potentially infected or contaminated preserved eggs (or associated wastes) likely to be directed toward each exposure group.

The next consideration following exposure of susceptible local species to any potentially contaminated material, i.e. the likelihood that an agent might establish in a local population, was dealt with in the consequence assessment (see section 3.2.3).

3.2.3 Consequence assessment

According to the OIE Code, a consequence assessment should ‘describe the potential consequences of a given exposure, and estimate the probability of them occurring’. Consequence assessment describes the process used to analyse the likelihood and impacts of establishment and spread of disease for each of the identified pathogenic agents of concern.

The plausible ‘outbreak scenario’ was considered for each identified exposure group. The likelihood of the outbreak scenario occurring was estimated, based on species and management or behaviour of each exposure group, and the characteristics of the pathogenic agent. The impact for the outbreak scenario was also estimated.

Steps in the consequence assessment process were:

- Identification of the outbreak scenario that might most likely occur as a result of host exposure to the pathogenic agent of concern.
- Determination of the likelihood of the outbreak scenario occurring — to obtain a partial likelihood of establishment and/or spread (PLES).
- Determination of the nature and magnitude of adverse effects (economic, social and environmental) for the outbreak scenario.

Identification of outbreak scenario

The outbreak scenario chosen as being most appropriate to the present review was the pathogenic agent establishing in the directly exposed population and spreading to other populations of susceptible species, where it is either eradicated due to control measures put in place, or becomes endemic in Australia. Whether or not the disease agent becomes endemic, or is eradicated, will depend on the nature of the pathogenic agent under consideration.

Likelihoods associated with the outbreak scenario

When estimating the likelihood associated with the outbreak scenario, qualitative descriptors are used as detailed previously in Table 1. For many of the diseases under consideration, Biosecurity Australia has previously assessed the likelihood of introduced disease agents spreading beyond the initially exposed group after introduction [Biosecurity Australia 2006] – the ‘*Draft Generic Import Risk Analysis Report for Chicken Meat*’. Where this has been done, the previously assessed estimates have been used in this review. For some disease agents that were not considered in the draft chicken meat IRA report, assessment of the likelihood of

spread has been done by comparison with similar, previously assessed disease agents. Where this approach has been taken, the basis for the comparison is recorded in the discussion of individual disease agents.

Scenario impacts

The establishment and spread of a disease agent may cause a number of direct and indirect impacts on biological systems. The direct and indirect impacts considered in this assessment are described below.

Direct impacts of a disease agent on host species and the environment

1. *The life or health (including production effects) of production, domestic or feral animals.*
2. *The living environment, including the life or health of native animals and plants, and any impacts on the non-living environment.*

Indirect impacts of a disease agent on host species and the environment

1. *New or modified eradication, control, surveillance or monitoring and compensation strategies or programs.*
2. *Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to, or utilising outputs from, directly affected industries.*
3. *International trade impacts, including loss of markets, meeting new technical requirements to enter or maintain markets and changes in international consumer demand.*
4. *The environment, including biodiversity, endangered species and the integrity of ecosystems.*
5. *Communities, including reduced tourism, reduced rural and regional economic viability, loss of social amenity, and any 'side effects' of control measures.*

Consideration of *the indirect impacts on the environment* includes harm arising from the impact of the pathogenic agent itself, as well as from any treatments or procedures used to control it. The extent of harm was evaluated taking into account:

- all on-site and off-site impacts
- the geographical scope and magnitude of the impact
- the frequency and duration of the action causing the harm
- the total impact which can be attributed to that action over the entire geographic area affected, and over time (i.e. cumulative impact)
- reversibility of the impact; the sensitivity of the receiving environment (recognised environmental features of high sensitivity), and
- the degree of confidence with which the impacts of the action are known and understood.

The direct and indirect consequences described above collectively cover the economic, social and environmental effects of a disease. In assessing direct and indirect impacts, it was important to ensure that particular impacts were not accounted for more than once.

In particular, the direct effects of a disease on a native or wild species were assessed under the criterion describing ‘*the living environment, including the life or health of native animals and plants, and any impacts on the non-living environment*’, whereas the indirect or ‘flow-on’ effects on the environment were assessed under ‘indirect impacts on the environment’ criterion.

Describing direct and indirect disease effects

Two groups of qualitative descriptors have been adopted by Biosecurity Australia to describe the impact of a pest or disease on each of the identified direct and indirect criteria:

- *Level of impact* — consequences accrued at a national, State or Territory, district or region, or local level.
- *Magnitude of impact* — the relative seriousness of the consequences of a pest or disease at a national, State or Territory, district or region, or local level.

Level of impact

Although the consequences of a pest or disease agent will ultimately be assessed on a national scale, it will be helpful to describe it also at the ‘State or Territory’, ‘district or region’ or ‘local’ level. These are defined as follows:

<i>National:</i>	Australia-wide
<i>State/Territory:</i>	an Australian ‘State’ (NSW, Victoria, Queensland, Tasmania, South Australia or Western Australia) or ‘Territory’ (the Australian Capital Territory and the Northern Territory)
<i>District or region:</i>	a geographically or geopolitically associated collection of aggregates — generally a recognised section of a State, such as the ‘North West Slopes and Plains’ or ‘Far North Queensland’
<i>Local:</i>	an aggregate of households or enterprises — e.g. a rural community, a town or a local government area.

Magnitude of impact

The magnitude of impact on each criterion is evaluated by Biosecurity Australia using four qualitative terms. These terms can be applied to impact on a national scale, or at the State or Territory level, the district or regional level, or local level. At each level, the frame of reference should be the impact on the community at that level, which will often differ markedly from the impact of the pest or disease on directly affected parties.

<i>Unlikely to be discernible</i>	not usually distinguishable from normal day-to-day variation in the criterion
<i>Minor significance</i>	recognisable, but minor and reversible
<i>Significant</i>	serious and substantive, but reversible and unlikely to disturb either economic viability or the intrinsic value of the

	critereon
<i>Highly significant</i>	extremely serious and irreversible and likely to disturb either economic viability or the intrinsic value of the criterion.

Assessing direct or indirect impacts on a national scale

A national scale implies the collective consequences of a pest or disease on the Australian economy or community. To estimate consequences on this scale, it is necessary to describe the outbreak scenario upon which the consequence assessment is based.

Biosecurity Australia uses a structured qualitative framework to assess the direct or indirect consequences of a pest or disease on a national scale. This framework is described schematically in Table 2 below. Under this schema, the first step is to assess the magnitude of direct or indirect impact on the national economy or the Australian community. If, for that particular criterion, there is no discernible impact at a national level (i.e. if there would be no discernible impact on the national economy, or no threat to a thing of value to the Australian community), then, in descending order, the magnitude of impact at the State or Territory level, the district level or the local level will be investigated. This results in a left-to-right movement through the columns of the table.

At each of the lower geographic levels, an impact more serious than ‘minor’ was understood to be discernible at the level above (eg a ‘significant’ impact at the State level would be considered to be equivalent to at least a ‘minor’ impact at National level). In addition, the impact of a disease at a given level in more than one State/Territory, district/region or local area was considered to represent at least the same magnitude of impact at the next highest geographic level.

Combining direct and indirect criteria

The measure of impact obtained for each direct and indirect criterion was combined to give the overall impacts of a disease agent. The following rules were used for the combination of direct and indirect impacts. The rules are mutually exclusive, and were addressed in the order that they appear in the list, i.e. *if the first set of conditions does not apply, the second set should be considered; if the second set does not apply, the third set should be considered; and so forth, until one of the rules applies.*

Where, with respect to direct or indirect criteria, the overall impact of the outbreak is

- extreme
 - when any one criterion is ‘nationally highly significant’, or
 - when more than one criterion are ‘nationally significant’, or
 - when any one criterion is ‘nationally significant’ and all others are ‘nationally minor’;
- high
 - when any one criterion is ‘nationally significant’, or
 - when all criteria are ‘nationally minor’;
- moderate
 - when any one or more criterion are ‘nationally minor’, or

- when all criteria are ‘minor at State or Territory level’ or ‘significant at a regional (or district) level’;
- low
 - when any one or more criterion are ‘minor at State or Territory level’, or
 - when all criteria are ‘minor at a regional (or district) level’ or ‘locally significant’;
- very low
 - when any one or more criterion are ‘minor at a regional (or district) level’ or ‘locally significant’, or
 - when all criteria are ‘minor at local level’;
- negligible
 - when any one or more criterion are ‘minor at local level’, or
 - when all criteria are ‘unlikely to be discernible at local level’.

Table 2. The assessment of direct or indirect impacts on a national scale¹

	Highly significant			
	Significant			
	Minor	←	<i>Greater than 'minor' at State level equals at least 'minor' at National level</i>	
National Impact	Unlikely to be discernible	Minor	←	<i>Greater than 'minor' at district/region level equals at least 'minor' at State level</i>
	-	Unlikely to be discernible	Minor	←
			Unlikely to be discernible	<i>Greater than 'minor' at Local level equals at least 'minor' at district/region level</i>
			Unlikely to be discernible	Minor
				Unlikely to be discernible
	<i>National</i>	<i>State or Territory</i>	<i>District or region</i>	<i>Local</i>
	Geographic Level			

¹ Shaded cells with bold font are those that dictate measure of national impact. Impacts greater than 'minor' at local, district or regional level are considered to represent **at least** 'minor' impacts at the next higher geographic level.

Impacts of disease outbreaks resulting from infected preserved duck eggs entering Australia

If the adverse impacts arising from a pathogenic agent entering, establishing and spreading in Australia were clearly evident without requiring a detailed analysis, or had already been determined by another related risk analysis, (in this case, the *Draft Generic Import Risk Analysis for Chicken Meat*), and were evaluated to be 'very low' or 'negligible', no further risk assessment was considered necessary for that disease agent. Where it is determined that the impact assessment could be 'low' or greater, a full risk assessment, including technical information on the disease agent, and release, exposure and consequence assessment, was completed and is set out in this document.

3.2.4 Risk estimation

In the context of this analysis, 'risk estimation' describes the integration of likelihoods and impacts, with the objective of deriving an estimate of the overall risk associated with each pathogenic agent as a result of importing preserved duck eggs.

Calculation of the partial annual likelihood of entry and exposure (PALEE)

The partial annual likelihood of entry and exposure (PALEE) is the exposure group-specific likelihood that there would be one or more host exposure events over a period of one year. This likelihood was determined for each of the exposure groups (PALEEbp and PALEEwb). The PALEE for each exposure group was calculated by determining the product of the likelihood of release (LR) and the corresponding partial likelihood of exposure (PLE) using the matrix of 'rules' for combining descriptive likelihoods (Table 3).

For example, for the low biosecurity poultry exposure group:

$$\text{PALEEbp} = \text{LR} \times \text{PLEbp}$$

Table 3. A matrix of 'rules' for combining descriptive likelihoods

	High	Moderate	Low	Very low	Extremely low	Negligible
High	High	Moderate	Low	Very low	Extremely low	Negligible
Moderate		Low	Low	Very low	Extremely low	Negligible
Low			Very low	Very low	Extremely low	Negligible
Very low				Extremely low	Extremely low	Negligible
Extremely low					Negligible	Negligible
Negligible						Negligible

Calculation of the partial annual likelihood of entry, exposure, establishment and spread (PALEES)

For each combination of exposure group and outbreak scenario, the likelihood of entry, exposure, establishment and spread (PALEESbp and PALEESwb) was obtained by combining the likelihoods of entry and exposure (PALEEbp and PALEEwb) with the likelihood of the outbreak scenario occurring (PLESbp and PLESwb), using the matrix of 'rules' for combining descriptive likelihoods (Table 3).

Calculation of the partial annual risk (PAR)

Once the likelihood of entry, exposure, establishment and spread had been determined for each combination of exposure group and outbreak scenario, this was combined with the assessment of impacts by reference to the risk estimation matrix (Table 4). The result was an estimate of the 'partial annual risk of introducing a given disease agent into Australia as a result of the decision to import preserved duck eggs from Taiwan' (PAR).

Table 4. Risk estimation matrix: estimation of the risk of entry and exposure

Annual likelihood of entry, exposure, establishment and spread	<i>High likelihood</i>	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
	<i>Moderate</i>	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
	<i>Low</i>	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk	High risk
	<i>Very low</i>	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk
	<i>Extremely low</i>	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk
	<i>Negligible likelihood</i>	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk
		<i>Negligible impact</i>	<i>Very low</i>	<i>Low</i>	<i>Moderate</i>	<i>High</i>	<i>Extreme impact</i>

Impact of entry, exposure, establishment and spread

Combination of partial annual risk estimates

Because the partial annual risks associated with each of the exposure groups were derived qualitatively, they cannot be ‘summed’ in the usual sense. Instead, a system of eleven rules has been developed to provide a conservative approximation. These rules are mutually exclusive, and should be addressed in the order that they appear in the list. For example, if the first set of conditions does not apply, the second set should be considered. If the second set does not apply, the third set should be considered, and so forth until one of the rules applies.

1. Where any one partial annual risk is extreme, the overall annual risk is also considered extreme
2. Where more than one partial annual risk is high, the overall annual risk is considered extreme
3. Where any one partial annual risk is high and each remaining partial annual risk is moderate, the overall annual risk is considered extreme
4. Where a single partial annual risk is high and the remaining partial annual risks are not unanimously moderate, the overall annual risk is considered high
5. Where all partial annual risks are moderate, the overall annual risk is considered high
6. Where one or more partial annual risks are moderate, the overall annual risk is considered moderate
7. Where all partial annual risks are low, the overall annual risk is considered moderate
8. Where one or more partial annual risks are considered low, the overall annual risk is considered low

9. Where all partial annual risks are very low, the overall annual risk is considered low
10. Where one or more partial annual risks are very low, the overall annual risk is considered very low
11. Where all partial annual risks are negligible, the overall annual risk is considered negligible.

The result of this process was an estimate of the ‘annual risk of introducing a given disease into Australia as a result of the decision to import preserved duck eggs’. This was considered the final output of the risk assessment.

3.3 Method for risk management

Risk evaluation is described in the OIE Code as the process of comparing the estimated risk with a country’s ALOP. ALOP is defined in this document and the SPS Agreement as ‘*the level of protection deemed appropriate by the WTO member country establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within its territory ...*’.

Australia has traditionally maintained a ‘very conservative’ attitude to quarantine risk. Given this, a risk that was either ‘very low’ or ‘negligible’, was considered sufficiently conservative to achieve Australia’s ALOP. Australia’s ALOP is shown in the risk estimation matrix (Table 5) as the band of cells associated with ‘very low’ risk. This provides a benchmark for evaluating risk and determining whether risk management is required.

The use of a benchmark for evaluating risk is illustrated in the process outlined below

- For each potential hazard, the level of risk, or expected loss, associated with the unrestricted importation of preserved eggs from Taiwan was estimated
- The unrestricted risk was then evaluated using the risk estimation matrix (Table 4) to determine where it fell in relation to Australia’s ALOP
- If the unrestricted risk was ‘negligible’ or ‘very low’, then it was considered acceptable and further risk management was not required
- If the unrestricted risk was ‘low’, ‘moderate’, ‘high’ or ‘extreme’, then risk management strategies were identified and, for each hazard, the risk was recalculated
- Where the subsequently restricted risk derived using a particular risk management strategy was ‘very low’ or ‘negligible’, that strategy was considered acceptable.

3.3.1 Risk management measures

If they were found necessary, risk management measures would be applied to reduce the likelihood that the importation of preserved duck eggs from Taiwan would lead to the entry, exposure, establishment and spread of exotic pathogenic agents in Australia. There are two means by which this could be achieved:

- Reducing the likelihood of pathogenic agents entering Australia in imported commodities by imposing conditions on one or more of the steps in the release scenario — i.e. ‘pre-import measures’
- Reducing the likelihood that susceptible host species in Australia would be exposed to the pathogenic agent in a contaminated imported commodity, or in other products

or waste derived from that commodity, by imposing conditions on one or more of the steps in the exposure scenario — i.e. ‘post-import measures’.

3.3.2 Pre-import measures

In accordance with accepted international standards (OIE 2007), product imported from a country or zone free of a specific disease, will be considered free of contamination of the disease agent and no import restrictions relevant to the disease will apply, subject to a satisfactory assessment of the country or zoning arrangements by the relevant Australian government authority. The principles of zoning, as they apply to this report, include, but are not limited to, the following:

- a standard of veterinary services, diagnostic capability, disease surveillance and certification arrangements deemed satisfactory by Australian Government authorities⁵;
- the disease is notifiable in the exporting market;
- appropriate government veterinary health certification is provided with each importation; and
- zoning arrangements take account of the epidemiological situation relating to the disease in the exporting market.

Biosecurity Australia recognises that there may be other equivalent risk management measures, such as flock accreditation schemes or the concept of compartmentalisation, recently introduced by the OIE. These would need to be assessed on a case-by-case basis. A rigorous assessment of any application for approval of compartmentalisation or flock accreditation schemes will be undertaken to ensure that effective risk management measures are implemented and maintained throughout the complete chain from farm to export. Correct identification of the origin of imported preserved eggs is central to the application of such risk management measures. This entails correct identification of the farm of origin, and correct identification of eggs eligible for export to Australia during all stages of production, processing, storage and transport. It also entails correct segregation to ensure that the product maintains its status and is not contaminated by disease agents. A detailed submission will need to be provided by the veterinary authority of Taiwan and Australia will conduct an on-ground assessment of the proposed compartment or flock accreditation scheme.

Risk may also be managed by requiring that the product be treated by cooking or other means that have been demonstrated, to the satisfaction of Australian Government authorities, to effectively destroy the disease agent. Such processing, if undertaken off-shore, and in combination with measures to ensure protection from post-processing contamination, would reduce the risk to negligible.

3.3.3 Post-import measures

Australia has a long history of implementing measures to reduce the likelihood of susceptible host exposure, for example farmer awareness, and biosecurity practices. Other programs help

⁵ Animal Quarantine Policy Memorandum 1999/41 provides details of the processes used to assess the effectiveness of overseas country veterinary authorities, and other matters relating to approval of countries to export to Australia.

to limit the impact of disease establishment, for example emergency control plans to limit spread and stamp out disease, and access to emergency vaccine reserves. These programs were taken into consideration in making the unrestricted risk estimate, in particular in the consequence assessment. Therefore these measures cannot be separately assessed as risk management measures, as any effect of risk reduction afforded by them has already been taken account of in the risk assessment.

Heating or other treatments, as discussed under pre-import measures, could also be applied as a post-import measure, by requiring that imported preserved eggs be held under quarantine surveillance and/or be treated to ensure destruction of disease agents, after arrival in Australia.

3.3.4 Risk management options

For the current review of risks associated with the import of traditional preserved eggs from Taiwan, risk management options considered practicable by Biosecurity Australia include:

Option 1: Allow import from a country or zone or compartment free of the pathogen of concern. As discussed above, this option reduces the risk to negligible and would achieve Australia's ALOP.

Option 2: Allow import of product which has been processed off-shore to ensure destruction of the pathogen of concern. As discussed above, this option reduces the risk to negligible and would achieve Australia's ALOP.

Option 3: Allow import of product subject to a requirement for it to be processed under quarantine control, to ensure destruction of the pathogen of concern. Any waste produced during processing is to be treated as quarantinable waste. As discussed above, this option reduces the risk to negligible and would achieve Australia's ALOP.

3.3.5 Reference List

1. World Organisation for Animal Health (OIE). 2007. "Terrestrial Animal Health Code 2007 Chapter 1.3.5 Zoning and compartmentalisation". Web page. Available at http://www.oie.int/eng/normes/mcode/en_chapitre_1.3.5.htm

4 Hazard identification

4.1 Initial hazard list

As discussed in Section 3.1 (Method for Hazard Identification) an initial hazard list was compiled by reference to the OIE disease list and scientific literature which list disease agents of relevance to ducks. These disease agents are shown in the table below (Table 5).

Table 5. Initial hazard list

OIE-listed disease agents	
Notifiable avian influenza (NAI) viruses	Newcastle disease (ND) virus
Avian infectious laryngotracheitis virus (ILT)	Avian metapneumovirus
Duck hepatitis virus	Infectious bursal disease virus (IBDV)
Japanese encephalitis (JE) virus	Marek's disease (MD) virus
<i>Mycoplasma gallisepticum</i>	<i>Mycoplasma synoviae</i>
<i>Chlamydophila psittaci</i>	<i>Pasteurella multocida</i>
<i>Salmonella</i> Gallinarum	<i>Salmonella</i> Pullorum
West Nile virus (WNV)	
Other disease agents	
Haemophilus paragallinarum	Avian encephalomyelitis virus
<i>Borrelia anserina</i>	<i>Salmonella</i> Enteritidis
Multi-drug resistant <i>Salmonella</i> Typhimurium	Avian leucosis virus
Fowl pox virus	Avian nephritis virus
Duck enteritis virus	Chicken anaemia virus
<i>Ornithobacterium rhinotracheale</i>	Enterohaemorrhagic <i>Escherichia coli</i> (EHEC)
Avian reovirus	<i>Riemerella anatipestifer</i>
<i>Mycobacterium avium-intracellulare</i>	Reticuloendotheliosis virus
Avian paramyxovirus 3	Avian paramyxovirus 2

4.2 Disease agents excluded from list

In addition, there are potential disease agents which have not been reported to occur in ducks. These agents are listed below and are not considered further. Nonetheless, should evidence of these diseases be reported in ducks at a later stage, further consideration will be given to the disease agents.

- Infectious bronchitis virus – there is no evidence of this disease agent occurring in ducks
- Infectious laryngotracheitis virus – there is no evidence of this disease agent occurring in ducks
- *Mycoplasma iowae* – there is no evidence of this disease agent occurring in ducks
- Group 1 fowl adenovirus serotype 1– there is no evidence of this disease agent occurring in ducks
- Group 1 fowl adenovirus serotype 4, Group 2 adenovirus – there is no evidence of this disease agent occurring in ducks
- Goose parvovirus – this disease agent affects only geese and Muscovy ducks. As discussed in ‘Scope’ (page 5) this policy review covers the import of preserved eggs derived from commercial flocks of Tsaiya ducks only. Therefore this disease agent is not considered further
- *Haemophilus paragallinarum* – ducks are considered refractory to experimental infection, and there is no evidence of egg transmission (Blackall and Matsumoto 2003).

4.3 Hazard refinement

The initial hazard list was refined as discussed in Section 3.1, and then further refined by considering whether or not each disease agent may be present in or on the egg, and the effects of processing on the agent. The following section summarises the available literature on the effects of heat and pH treatment on the agents included in the initial hazard list. Results of hazard refinement are presented in Table 6.

Table 6. Hazard refinement

Disease agent	Hazard identification criteria (Yes/No)								
	Agent infects ducks	May be present within egg contents	May be present on or in egg shell	Capable of adverse impact ¹	Occurrence in Australia ²	Effect of heat treatment (salted) ¹⁰	Retain for risk assessment (salted)	Effect of pH (alkalised) ¹¹	Retain for risk assessment (alkalised)
OIE-listed disease agents									
Notifiable avian influenza virus	YES	YES	YES	YES	NO	Inactivate	NO	Insufficient information	YES
Newcastle disease virus	YES	YES	YES	YES	NO ³	Inactivate	NO	Insufficient information	YES
Avian metapneumovirus	YES	NO	YES	YES	NO	Inactivate	NO	Inactivate	NO
Duck hepatitis virus	YES	NO	YES	YES	NO	Inactivate	NO	Inactivate	NO
Infectious bursal disease virus	YES	NO	YES	YES	YES ⁴	Inactivate	NO	Inactivate	NO
Japanese encephalitis virus	Serological evidence	NO	NO	YES	YES ⁵		NO		NO

Hazard identification

Disease agent	Hazard identification criteria (Yes/No)								
	Agent infects ducks	May be present within egg contents	May be present on or in egg shell	Capable of adverse impact ¹	Occurrence in Australia ²	Effect of heat treatment (salted) ¹⁰	Retain for risk assessment (salted)	Effect of pH (alkalised) ¹¹	Retain for risk assessment (alkalised)
Marek's disease virus	NO	NO	YES	YES	YES		NO		NO
<i>Mycoplasma gallisepticum</i>	YES	YES	YES	YES	YES		NO		NO
<i>Mycoplasma synoviae</i>	YES	YES	YES	YES	YES		NO		NO
<i>Chlamydophila psittaci</i>	YES	YES	YES	YES	YES		NO		NO
<i>Pasteurella multocida</i>	YES	NO	YES	YES	YES		NO		NO
<i>Salmonella Gallinarum</i>	YES	YES	YES	YES	NO	Inactivate	NO	Insufficient information	YES
<i>Salmonella Pullorum</i>	YES	YES	YES	YES	NO ⁶	Inactivate	NO	Insufficient information	YES
West Nile virus	YES	NO	NO	YES	NO		NO		NO

Disease agent	Hazard identification criteria (Yes/No)								
	Agent infects ducks	May be present within egg contents	May be present on or in egg shell	Capable of adverse impact ¹	Occurrence in Australia ²	Effect of heat treatment (salted) ¹⁰	Retain for risk assessment (salted)	Effect of pH (alkalised) ¹¹	Retain for risk assessment (alkalised)
Other disease/agents									
Avian encephalomyelitis virus	Experimental infection reported	YES	YES	YES	YES		NO		NO
<i>Borrelia anserina</i>	YES	NO	NO	YES	YES		NO		NO
<i>Salmonella</i> Enteritidis	YES	YES	YES	YES	NO ⁷	Inactivate	NO	Insufficient information	YES
Multi-drug resistant <i>Salmonella</i> Typhimurium	YES	YES	YES	YES	NO ⁸	Inactivate	NO	Insufficient information	YES
Avian leucosis virus	Experimental infection reported	YES	YES	YES	YES		NO		NO
Fowl pox virus	YES	NO	NO	YES	YES		NO		NO

Hazard identification

Disease agent	Hazard identification criteria (Yes/No)								
	Agent infects ducks	May be present within egg contents	May be present on or in egg shell	Capable of adverse impact ¹	Occurrence in Australia ²	Effect of heat treatment (salted) ¹⁰	Retain for risk assessment (salted)	Effect of pH (alkalised) ¹¹	Retain for risk assessment (alkalised)
Antibiotic resistant <i>Campylobacter jejuni</i>	YES	YES	YES	YES	YES		NO		NO
Avian nephritis virus	Not reported	YES	YES	YES	YES		NO		NO
Chicken anaemia virus		YES	YES	YES	YES		NO		NO
Duck enteritis virus	YES	YES	YES	YES	NO	Inactivate	NO	Insufficient information	YES
Enterohaemorrhagic <i>Escherichia coli</i> (EHEC)	YES	NO	YES	YES	YES		NO		NO
<i>Ornithobacterium rhinotracheale</i>	YES	YES	YES	YES	NO	Insufficient information	YES	Insufficient information	YES
<i>Riemerella anatipestifer</i>	YES	NO	YES	YES	YES		NO		NO
Avian reovirus	YES	YES	YES	YES	YES ⁹		NO		NO

Disease agent	Hazard identification criteria (Yes/No)								
	Agent infects ducks	May be present within egg contents	May be present on or in egg shell	Capable of adverse impact ¹	Occurrence in Australia ²	Effect of heat treatment (salted) ¹⁰	Retain for risk assessment (salted)	Effect of pH (alkalised) ¹¹	Retain for risk assessment (alkalised)
Reticuloendotheliosis virus	YES	YES	YES	YES	YES		NO		NO
<i>Mycobacterium avium-intracellulare</i>	YES	YES	YES	YES	YES		NO		NO
Avian paramyxovirus-2	NO	NO	YES	YES	NO	Inactivate	NO	Inactivate	NO
Avian paramyxovirus-3	NO	NO	YES	YES	NO	Inactivate	NO	Inactivate	NO

Legend

1. *Capable of adverse impact*: The pathogenic agent (or a clearly identified strain of pathogenic agent) could potentially produce adverse consequence in susceptible humans or animal/bird species in the importing country
2. *Occurrence in Australia*: The pathogenic agent (or clearly identified strain of the pathogenic agent) should not be present in the importing country. If present, the pathogenic agent is associated with a notifiable disease, or is subject to an official control or eradication program
3. Virulent Newcastle disease virus of Australian origin has occurred in Australia, but has been eradicated
4. Although the disease occurs in Australia, more pathogenic serotypes are known to exist overseas, which have not been reported in Australia
5. One human case of Japanese encephalitis acquired on the Australian mainland has been reported, and there has been serological evidence of infection in sentinel and surveyed pigs on Cape York Peninsula

6. Australian commercial poultry are considered to be free of *S. Pullorum*. There has been no isolation of the agent in Australia for more than 10 years
7. A few isolations of *S. Enteritidis* from commercial poultry have occurred, most recently in Queensland in 2005. Affected flocks are subject to control measures. Affected flocks from the 2005 outbreak are subject to ongoing control measures and intensive monitoring
8. *S. Typhimurium* occurs commonly in Australia, but multi-drug resistant strains have not been reported in Australia except in returning travellers
9. Some strains of avian reovirus are endemic in Australian poultry.
10. Refer to section 7.4.1 Effect of processing (salted eggs) for discussions of the available literature on the effect of heat treatment.
11. Refer to section 7.4.2 Effect of processing (alkalised eggs) for discussions of the available literature on the effect pH treatment.

4.4 Effect of processing on potential hazards

As part of the hazard refinement, the scientific literature regarding each disease agent was examined to determine whether the disease agent, if present in or on the egg, would be inactivated by the processing applied in the production of traditional preserved duck eggs. The results of this examination are presented in Section 4.4.1 (for salted eggs) and in Section 4.4.2 (for alkalised eggs).

4.4.1 Effect of processing (salted and heat-treated eggs)

Grijspeerdt and Herman (2003) demonstrated that when eggs are placed in boiling water, the temperature of the egg yolk reaches approximately 80 °C within 10-12 minutes. The rate of heating of eggs in steam can be assumed to be similar, indicating that the temperature of egg yolks during processing will be at or above 80 °C for approximately 30 minutes when steamed at 100 °C for 45 minutes. This is consistent with the final temperature of the egg yolk reaching 85 °C as indicated by BAPHIQ. It is also considered that the egg shell will be directly exposed to the effect of the steam and will heat much more rapidly than material deeper within the egg albumen or yolk. The external surface of the egg is considered to be at or near 100 °C for greater than 30 minutes during the steaming process.

Heat treatments required to inactivate the various disease agents are tabulated in Table 7.

Table 7. Heat susceptibility of disease agents in liquid egg and egg products

Disease agent	Product	Temperature (°C)	Time (Secs)	Processing of salted eggs effective?
Notifiable Avian Influenza virus	Whole egg	60 °C	201 secs	
	Liquid egg white	55.6 °C	372 secs	Yes
Newcastle disease virus	Liquid egg white	56.7 °C	210 secs	
	Whole egg	60 °C	210 secs	Yes
Avian metapneumovirus	Unstated	56 °C	1800 secs	Yes
Duck hepatitis viruses (DHV)	Unstated	62°C	1800 secs	Yes
<i>Salmonella Pullorum</i> / <i>S. Gallinarum</i>	Unstated	60 °C	600 sec	Yes

<i>Salmonella</i> Enteritidis / <i>S. Typhimurium</i>	Unstated	80 °C	Instantaneous	Yes
Duck enteritis virus	Unstated	60 °C	600 secs	Yes
<i>Ornithobacterium rhinotracheale</i>				Insufficient data available
APMV2		Inactivation similar to ND		Yes
APMV3		Inactivation similar to ND		Yes

Notifiable avian influenza (NAI) viruses

NAI viruses are inactivated at temperatures reached during conventional cooking, if a core temperature of 70 °C is achieved (World Health Organisation 2006). The *Draft Generic Import Risk Analysis Report for Chicken Meat* (BAPM 2006/18)⁶ recommends cooking at 70 °C for one minute as an appropriate risk management measure for avian influenza viruses.

Pasteurisation protocols used by industry for liquid egg products have been stated to be effective at inactivating the virus (e.g. whole egg at 60 °C for 201 seconds; liquid egg white, 55.6 °C for 372 seconds; 10% salted yolk, 63.3 °C for 201 seconds (World Health Organization 2005).

Infectivity of most strains was lost after heating to 60 °C for five minutes and 56 °C for 15-30 minutes; with some strains requiring up to six hours at 56 °C for inactivation (Moses et al. 1948; Lang et al. 1968b; Lang et al. 1968a; Homme and Easterday 1970; Lu et al. 2003).

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present in, or on, the egg.

Newcastle disease virus (NDV) (Avian paramyxovirus-1)

Infectivity of Newcastle disease virus (NDV) is destroyed at 56 °C in 5 minutes to 6 hours (Beard and Hanson, 1984), and 70 °C for 50 seconds (Foster and Thompson 1957). All of the NDV strains tested were inactivated at 100 °C within 60 seconds (Beard and Hanson 1984). NDV could not be recovered from liquid egg heated at 64.4 °C for 200 seconds (Gough 1973).

One report states that standard pasteurisation is not reliable to inactivate NDV (King 1991). Nonetheless, another study reported that NDV is inactivated in egg products using standard industry pasteurisation protocols (Swayne and Beck 2004). Various minimum standards have been published for pasteurisation of egg products including liquid albumen (56.7 °C for 3.5 minutes), whole egg (60 °C for 3.5 minutes), 10% salted egg yolk (63.3 °C for 3.5 minutes), and dried egg white (54.4 °C for 7 to 10 days) (Swayne and Beck 2004).

Heat treatment of liquid whole egg in a water bath maintained at 64.4 °C for 200 seconds resulted in an 8 log₁₀ reduction in the titre of NDV (Gough 1973).

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present in, or on, the egg.

⁶ Available at: <http://www.biosecurityaustralia.gov.au/>

Avian metapneumovirus (turkey rhinotracheitis)

Avian metapneumovirus, has been reported to replicate in ducks (Gough et al. 1988) and it has been suggested that ducks may act as non-clinical carriers (Shin et al. 2001) However, field evidence suggests that transmission through the egg either transovarially or by egg contamination is unlikely to occur (Cook 2000). Therefore, it was considered that avian metapneumovirus, if present on raw material for processing of preserved eggs, will be a surface contaminant only. The virus is inactivated by exposure to 56 °C for 30 minutes (Collins et al. 1986).

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present in, or on, the egg.

Duck hepatitis viruses

Duck hepatitis viruses (DHV) types 1, 2 and 3, if present, are likely to be surface contaminants only. DHV type 1 is inactivated by exposure to 62 °C for 30 minutes (Woolcock 2003). DHV type 3 is sensitive to heating at 50 °C (Haider and Calnek 1979).

Biosecurity Australia considered that it is likely that DHV type 2 would be similarly inactivated.

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present in, or on, the egg.

Infectious bursal disease virus (IBDV)

IBDV serotype 1 only causes clinical disease in chickens. A serotype 1 IBDV has been isolated from the faeces of clinically healthy adult ducks, but the significance of the isolate is uncertain (McFerran et al. 1980). It is considered relatively unlikely that IBDV will be found in duck flocks.

Egg transmission does not occur (van den Berg 2006; Saif 2006). Any contamination would be limited to surface contamination of the shell only. Organisms present on the surface of the egg shell will be exposed directly to the effects of steam during the cooking process, and will be exposed to a temperature of 100 °C for the 45 minute cooking period. Existing Australian policy for the importation of meat flavours and or pet foods containing poultry derived material require that the product be heated to 100 °C for 30 minutes. This is considered adequate to address risks posed by IBDV.

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present on the egg shell.

Salmonella Pullorum* and *S. Gallinarum

S. Gallinarum is inactivated by heating within 10 minutes at 60 °C (Shivaprasad 2003). A minimum internal temperature of 74 °C will reduce viable *Salmonella* in chicken meat by at least 7 logs (National Advisory Committee on Microbiological Criteria for Foods 2006).

As yolk temperature reaches 85 °C during processing, Biosecurity Australia considered that both of these disease agents will be inactivated by processing as described above.

***Salmonella* Enteritidis and S. Typhimurium**

Inactivation of *S. Enteritidis* occurs at a yolk temperature of 80 °C (Grijpspeerdt and Herman 2003). *S. Typhimurium* demonstrates a similar susceptibility to heat as *S. Enteritidis* (Humphrey et al. 1989)). As yolk temperature reaches 85 °C during processing, it is considered that both of these disease agents will be inactivated by processing as described above.

Duck enteritis virus (DEV)

Taiwan claims country freedom from DEV although surveys were conducted some time ago. Australia considers that further, more recent data would be necessary to provide confidence of complete freedom from this disease agent. However, it is accepted that the prevalence of disease is extremely low, since population densities in concentrated duck-producing areas result in rapid spread of DEV, and it would be expected to spread rapidly once introduced to a laying flock. This would be expected to result in detection of the outbreak, even by passive surveillance methods.

DEV is inactivated after heating at 60 °C for 10 minutes (Hess and Dardiri 1968). Salted and heat-treated eggs are exposed to steam at 100 °C for 45 minutes, and yolk temperature reaches 85 °C during processing. As discussed earlier (page 6), the core of the egg reaches 80 °C after 10–12 minutes in boiling water (Grijpspeerdt and Herman 2003), and can be expected to do the same in steam.

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present in or on the egg.

Ornithobacterium rhinotracheale

There is little information on the susceptibility of *O. rhinotracheale* to heat or chemical treatment. *O. rhinotracheale* did not survive for 24 hours at 42 °C in poultry litter (Lopes et al. 2002). When sprayed on egg shells, *O. rhinotracheale* survived less than 24 hours at 37 °C, and less than three days at room temperature. At 4 °C the bacterium remained viable for 11 days (Varga, Fodor, and Makrai 2001). The susceptibility of the organism to higher temperatures appears not to have been studied.

Further consideration of the risk due to this disease agent is required.

Avian paramyxovirus 2 (APMV 2)

APMV 2, if present, is likely to be a surface contaminant only. Some strains of APMV 2 are heat stable at 56 °C for 120 minutes (Bankowski and Corstvet 1961). However, heat stability at higher temperatures appears not to have been studied. Sensitivity of APMV 2 to heat is considered to be similar to NDV (Alexander 2003). All strains of NDV tested were inactivated at 100 °C within 60 seconds (Beard and Hanson 1984).

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present in or on the egg.

Avian paramyxovirus 3 (APMV 3)

APMV 3, if present, is likely to be a surface contaminant only. APMV 3 is reported to be more heat sensitive than NDV (Tumova, Robinson, and Easterday 1979). When an APMV 3 isolate from a parakeet was exposed to 56 °C for 30 minutes, haemagglutinating activity was lost (Smit and Rondhuis 1976).

Biosecurity Australia therefore considered that processing of salted and heat-treated duck eggs, as described, will inactivate this disease agent if it is present in or on the egg.

4.4.2 Effect of processing (alkalised eggs)

As discussed in Section 2.2.2, the processing of alkalised duck eggs involves soaking the eggs within the shell for 35 days in alkali solution with a pH of 13, to achieve an internal pH of 9.5 or higher. This is the only step in the alkalising process which is likely to have any effect on the survival of pathogenic agents of quarantine concern.

Notifiable avian influenza (NAI) viruses

NAI viruses are inactivated by acid pH (OIE 2002). NAI viruses are generally stable at pH 11 and may require a pH of 12 or more to achieve inactivation (Moses, Brandly, and Jones 1947). NAI virus on the external surface of an egg shell will be directly exposed to the alkalising solution at pH 13 for 35 days, and it is considered likely that it would be inactivated. However, as the pH of the egg yolk may only reach 9.5 following the alkalisation process, it is not possible to conclude that virus within the egg yolk or albumen would be inactivated by the process.

Further risk assessment will be required for NAI viruses.

Newcastle disease virus (avian paramyxovirus 1)

NDV is inactivated by acid pH but the effect of alkaline pH is not described (OIE 2002). Moses, Brandly and Jones (1947) stated that NDV is generally stable at pH 11 and may require a pH of 12 or more to achieve a rapid and complete inactivation. After exposure for a week, NDV is inactivated by pH 9. However, no mention was made of strain variation or possible protective effects of egg yolk or albumen. NDV on the external surface of an egg shell will be directly exposed to the alkalising solution at pH 13 for 35 days, and it is considered likely that it would be inactivated as a result. However, it is not possible to conclude that virus within the egg yolk or albumen would be inactivated by the process.

Further risk assessment will be required for ND virus.

Avian metapneumovirus (turkey rhinotracheitis)

No evidence of egg transmission has been reported. Therefore any contamination would be limited to surface contamination of the shell only. The virus is reported to be inactivated by extremes of pH >10 (Townsend et al. 2000). Therefore it is reasonable to conclude that any external contamination would be inactivated during the alkalising process following exposure to pH 13. No further risk assessment is considered necessary for this disease agent.

Duck hepatitis virus

No evidence of egg transmission has been reported. Therefore any contamination would be limited to surface contamination of the shell only. The virus is reported to be inactivated by extremes of pH >12 (Davis 1987). Therefore it is reasonable to conclude that any external contamination would be inactivated during the alkalising process following exposure to pH 13. No further risk assessment is considered necessary for this disease agent.

Infectious bursal disease virus (IBDV)

IBDV serotype 1 only causes clinical disease in chickens. A serotype 1 IBDV has been isolated from the faeces of clinically healthy adult ducks, but the significance of the isolate is uncertain (McFerran et al. 1980). It is considered relatively unlikely that IBDV will be found in duck flocks.

Egg transmission does not occur (van den Berg 2006; Saif 2006). Therefore any contamination would be limited to surface contamination of the shell only. The virus has been reported to be inactivated after exposure to pH 12 at 30 °C for 1 hr (Benton et al. 1967). Therefore it is reasonable to conclude that any external contamination would be inactivated during the alkalising process following exposure to pH 13. No further risk assessment is considered necessary for this disease agent.

Salmonella Pullorum* and *S. Gallinarum*; *Salmonella* Enteritidis and *S. Typhimurium

Salmonellae can grow in a pH range 4.0 to 9.0 (Gast 2003). However, when 50–100 colony forming units (CFU) of *Salmonella* Enteritidis were inoculated into the yolk of eggs, which were subsequently subjected to the alkalising process, viable cell counts in the egg initially increased to approx 10^7 – 10^8 CFU/g. During the alkalising process, the pH of the yolk increased from 6.5 to 10.3. Towards the end of the process, due to the alkaline pH, cell counts fell to about 10^6 CFU/g. Following processing, the eggs were stored at 25 °C. Viable *Salmonellae* were still able to be found in at least some of the eggs after 2–3 months storage (Fu and Su 1997)

Therefore, it is not possible to conclude that Salmonellae within the egg would be inactivated by processing. Further risk assessment will be required for *Salmonella* Pullorum and *S. Gallinarum*, *S. Enteritidis* and *S. Typhimurium* (multi-drug resistant).

Duck enteritis virus

Taiwan claims country freedom from DEV although surveys were conducted some time ago. Australia considers that further, more recent data would be necessary to provide confidence of complete freedom from this disease agent. However, it is accepted that the prevalence of disease is extremely low, since population densities in concentrated duck-producing areas result in rapid spread of DEV, and it would be expected to spread rapidly once introduced to a laying flock. This would be expected to result in detection of the outbreak, even by passive surveillance methods.

The virus is inactivated virtually instantaneously at pH 11 (Hess and Dardiri 1968). However, the pH value of the yolk and albumen is not guaranteed to exceed 9.5, so DEV is retained for further risk assessment.

Ornithobacterium rhinotracheale

There is little information on the susceptibility of *O. rhinotracheale* to physical or chemical action. Therefore it is not possible to conclude that *O. rhinotracheale* either within or on the egg would be inactivated by the process. The disease agent is retained for further risk assessment.

Avian paramyxovirus 2 (APMV 2)

In the absence of any specific reports on the inactivation of APMV 2, it is assumed that APMV 2 has a similar spectrum of sensitivity to APMV 1. However, unlike APMV 1, there is no evidence of egg transmission of APMV 2. Therefore any contamination would be limited to surface contamination of the shell only. APMV 1 is generally stable at pH 11 and may require a pH of 12 or more to achieve rapid and complete inactivation. Contaminants on the surface of the egg shell will be exposed to pH 13, and so Biosecurity Australia considered that the virus would be inactivated by the process. No further risk analysis is considered necessary.

Avian paramyxovirus 3 (APMV 3)

In the absence of any specific reports on the inactivation of APMV 3, it is assumed that APMV 3 has a similar spectrum of sensitivity to APMV 1. However, unlike APMV 1, there is no evidence of egg transmission of APMV 3. Therefore any contamination would be limited to surface contamination of the shell only. APMV 1 is generally stable at pH 11 and may require a pH of 12 or more to achieve rapid and complete inactivation. Contaminants on the surface of the egg shell will be exposed to pH 13 during the alkalisising process, and Biosecurity Australia considered that the virus would be inactivated by the process. No further risk analysis is considered necessary.

4.5 Conclusions: hazard identification

4.5.1 Salted and heat-treated eggs

On the basis of the hazard refinement process, only *Ornithobacterium rhinotracheale* is retained for further disease risk assessment.

4.5.2 Alkalised eggs

On the basis of the hazard refinement process, the following disease agents of ducks are retained for further disease risk assessment:

- Notifiable avian influenza viruses
- Newcastle disease virus
- *Salmonella Pullorum/Salmonella Gallinarum*
- *Salmonella Enteritidis* /Multidrug resistant *Salmonella Typhimurium*
- Duck enteritis virus
- *Ornithobacterium rhinotracheale*

4.5.3 Reference list

1. Alexander, D. J. 2003. Newcastle disease, other avian Paramyxoviruses, and Pneumovirus infections. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 63-99. Ames, Iowa, USA: Iowa State Press.
2. Bankowski, R. A., and R. Corstvet. 1961. Isolation of a hemagglutinating agent distinct from Newcastle disease from the respiratory tract of chickens. *Avian Diseases* 5: 253-69.
3. Beard, C. W., and R. P. Hanson. 1984. Newcastle disease. In *Diseases of Poultry*. 8th ed., Editors M. S. Hofstad, H. J. Barnes, B. W. Calnek, W. M. Reid, and H. W. Yoder, 452-70. Ames, Iowa: Iowa State University Press.
4. Benton, W. J., M. S. Cover, J. K. Rosenberger, and R. S. Lake. 1967. Physicochemical properties of the infectious bursal agent (IBA). *Avian Diseases* 11, no. 1: 438-45.
5. Collins, M. S., R. E. Gough, S. A. Lister, N. Chettle, and R. Eddy. 1986. Further characterisation of a virus associated with turkey rhinotracheitis. *The Veterinary Record* 119: 606-7.
6. Cook, J. K. A. 2000. Avian rhinotracheitis. *Revue Scientifique Et Technique Office International Des Epizooties* 19, no. 2: 602-13.
7. Davis, D. 1987. Temperature and pH stability of duck hepatitis virus. *Avian Pathology* 16, no. 1: 21-30.
8. Fu, Y.-M., and T. Su. 1997. Survival of Salmonella enteritidis during the processing and storage of processed duck egg. *Journal of Food and Drug Analysis*. 5, no. 2: 171-78.
9. Gast, R. K. 2003. Paratyphoid infections. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 583-613. Ames, Iowa, USA: Iowa State Press.
10. Gough, R., M. S. Collins, W. J. Cox, and N. J. Chettle. 1988. Experimental infection of turkeys, chickens, ducks, geese, guinea fowl, pheasants and pigeons with turkey rhinotracheitis virus. *The Veterinary Record* 123: 58-59.
11. Gough, R. E. 1973. Thermostability of Newcastle disease virus in liquid whole egg. *The Veterinary Record* 93: 632-33.
12. Grijspeerdt, K., and L. Herman. 2003. Inactivation of Salmonella enteritidis during boiling of eggs. *International Journal of Food Microbiology* 82, no. 1: 13-24.
13. Haider, S. A., and B. W. Calnek. 1979. In vitro isolation, propagation, and characterization of duck hepatitis virus type III. *Avian Diseases* 23, no. 3: 715-29.
14. Hess, W. R., and A. H. Dardiri. 1968. Some properties of the virus of duck plague. *Archiv Für Die Gesamte Virusforschung* 24: 148-53.
15. Humphrey, T. J., M. Greenwood, R. J. Gilbert, B. Rowe, and P. A. Chapman. 1989. The survival of salmonellas in shell eggs cooked under simulated domestic conditions. *Epidemiol Infect* 103, no. 1: 35-45.
16. King, D. J. 1991. Evaluation of different methods of inactivation of Newcastle disease virus and avian influenza virus in egg fluids and serum. *Avian Diseases* 35: 505-14.
17. Lopes, V. C., B. Velayudhan, D. A. Halvorson, and K. V. Nagaraja. 2002. Survival of

- Ornithobacterium rhinotracheale* in sterilized poultry litter. *Avian Diseases* 46: 1011-14.
18. Moses, H. E., C. A. Brandly, and E. E. Jones. 1947. The pH stability of viruses of Newcastle disease and fowl plague. *Science* 105: 477-79.
 19. National Advisory Committee on Microbiological Criteria for Foods. 2006. "Response to the questions posed by FSIS regarding consumer guidelines for the safe cooking of poultry products. Draft adopted March 24, 2006." Web page, [accessed April 2006]. Available at http://www.fsis.usda.gov/PDF/NACMCF_Report_Safe_Cooking_Poultry_032406.pdf.
 20. Saif, Y M (saif.1@osu.edu). 6 January 2006. "IBDV in Ducks." E-mail to David Kim (David.Kim@affa.gov.au).
 21. Shin, H. J., M. K. Njenga, D. A. Halvorson, D. P. Shaw, and K. V. Nagaraja. 2001. Susceptibility of ducks to avian pneumovirus of turkey origin. *American Journal of Veterinary Research* 62: 991-94.
 22. Shivaprasad, H. L. 2003. Pullorum disease and fowl typhoid. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 568-82. Iowa, USA: Iowa State Press.
 23. Smit, T., and P. R. Rondhuis. 1976. Studies on a virus isolated from the brain of a parakeet (*Neophema* sp). *Avian Pathology* 5: 21-30.
 24. Swayne, D. E., and J. R. Beck. 2004. Heat inactivation of avian influenza and Newcastle disease viruses in egg products. *Avian Pathology* 33, no. 5: 512-18.
 25. Swayne David E., and Beck Joan R. 2004. Heat Inactivation of Avian Influenza and Newcastle Disease Viruses in Egg Products. *Avian Pathology* 33, no. 5: 512-18.
 26. Townsend, E. , D. A. Halvorson, K. V. Nagaraja, and D. P. Shaw. 2000. Susceptibility of an avian pneumovirus isolated from Minnesota turkeys to physical and chemical agents. *Avian Diseases* 44: 336-42.
 27. Tumova, B., J. H. Robinson, and B. C. Easterday. 1979. A hitherto unreported paramyxovirus of turkeys. *Research in Veterinary Science* 27: 135-40.
 28. van den Berg, Thierry (Thierry.vandenBerg@var.fgov.be; thvan@var.fgov.be). 27 June 2006. "IBDV in Duck Eggs." E-mail to Dr. David Kim (David.Kim@affa.gov.au).
 29. Woolcock, P. R. 2003. Duck hepatitis. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 343-54. Iowa, USA: Iowa State Press .
 30. World Health Organisation. 2006. "Avian influenza: food safety issues - microbiological risks in food." Web page, [accessed July 2006]. Available at <http://www.who.int/foodsafety/micro/avian/en/index1.html>.

5 Risk assessments – salted and heat-treated duck eggs

As discussed above (Section 4), only one disease agent (*Ornithobacterium rhinotracheale*) was retained for further risk assessment for salted and heat-treated preserved eggs.

5.1 *Ornithobacterium rhinotracheale*

Based on an examination of the scientific literature, it was not possible to definitively conclude that *Ornithobacterium rhinotracheale* would be inactivated by processing of salted and heat-treated duck eggs, and it was therefore necessary to examine the risk posed by the introduction of this disease agent.

5.1.1 Technical information

Background

O. rhinotracheale is a respiratory pathogen of avian species, and has been implicated in both primary and secondary infections in chickens and turkeys. While the bacteria has been isolated from ducks there are no published reports of it causing disease, and it is likely that infection remains subclinical. The bacterium was isolated for the first time in chickens and pigeons in Taiwan in 2006 (Tsai and Huang 2006). Infection with *O. rhinotracheale* has not been documented in avian species in Australia. It is of no known public health significance, and is not OIE-listed.

Agent characteristics

There is little information on the susceptibility of *O. rhinotracheale* to physical or chemical action. *O. rhinotracheale* in poultry litter survived for one day at 37 °C, 6 days at 22 °C, 40 days at 4 °C and at least 150 days at –12 °C (Lopes et al. 2002). When sprayed on egg shells, *O. rhinotracheale* survived less than 24 hours at 37 °C, and less than three days at room temperature. However, at 4 °C the bacterium remained viable for 11 days (Varga, Fodor, and Makrai 2001).

Epidemiology

O. rhinotracheale infection in commercial poultry has been reported in the US, Germany, South Africa, Netherlands, France, Belgium, Hungary, Japan, Canada, the United Kingdom (Turan and Ak 2002), Brazil (Canal et al. 2003), and Pakistan (Naeem, Malik, and Ullah 2003), but not in Australia. Natural infections have been reported in chickens and turkeys, and the organism has been isolated from wild birds (gulls, rooks, partridge, chukar, pheasant, pigeon, quail, duck, ostrich, geese and Guinea fowl) (van Empel and Hafez 1999; van den Bosch 2001). It has been postulated that infection in the poultry population may have resulted from the relatively recent transmission of *O. rhinotracheale* to domestic poultry from wild birds (Amonsin et al. 1997). Experimental studies have shown that all strains tested seem able to infect turkeys and meat chickens with comparable severity (van den Bosch 2001).

Transmission in eggs

O. rhinotracheale spreads both vertically and horizontally (van Veen, Vrijenhoek, and van Empel 2004). Vertical and horizontal transmissions have been demonstrated in turkeys within the hatchery (van Veen, Vrijenhoek, and van Empel 2004). Egg transmission (either transovarially or by cloacal contamination) is supported by the isolation of *O. rhinotracheale* from the oviducts and ovaries of experimentally infected 56 week old turkey breeder hens (Back et al. 1998). Horizontal transmission is by direct or indirect contact through aerosols or drinking water (Chin, van Empel, and Hafez 2003).

Quarantine significance

O. rhinotracheale is not an OIE-listed disease agent.

O. rhinotracheale is not notifiable in any State or Territory of Australia, and is not subject to official controls within Australia. *O. rhinotracheale* is not included in Australia's Emergency Animal Disease Response Agreement. Therefore, it is considered to be of relatively minor concern, and is unlikely to have consequences for the poultry industry in Australia that are discernible beyond the district/region level.

5.1.2 Risk assessment

Biosecurity Australia has previously conducted an IRA on this disease agent, and the results of that analysis have been published in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the impact of *O. rhinotracheale* becoming endemic in Australia as **very low**. As can be seen from the risk estimation matrix (Table 4) an organism which has impacts which are very low or lower cannot present a risk greater than very low. As this unrestricted risk estimate achieves Australia's ALOP¹, no risk management is considered necessary for this disease agent, beyond ensuring that the processing of the salted and heat-treated eggs is carried out as advised by BAPHIQ, and that post-processing contamination is prevented by good handling practices.

5.1.3 Reference list

1. Amonsin, A., J. F. Wellehan, L. L. Li, P. Vandamme, C. Lindeman, M. Edman, R. A. Robinson, and V. Kapur. 1997. Molecular epidemiology of *Ornithobacterium rhinotracheale*. *Journal of Clinical Microbiology* 35, no. 11: 2894-98.
2. Back, A., G. Rajashekara, R. B. Jeremiah, D. A. Halvorson, and K. V. Nagaraja. 1998. Tissue distribution of *Ornithobacterium rhinotracheale* in experimentally infected turkeys. *The Veterinary Record* 143: 52-53.
3. Canal, C. W., J. A. Leao, D. J. Ferreira, M. Macagnan, C. T. P. Salle, and A. Back. 2003. Prevalence of antibodies against *Ornithobacterium rhinotracheale* in broilers and breeders in southern Brazil. *Avian Diseases* 47: 731-37.
4. Chin, R. P., P. C. van Empel, and H. M. Hafez. 2003. *Ornithobacterium rhinotracheale* infection. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 683-90. Ames, Iowa, USA: Iowa State Press.
5. Lopes, V. C., B. Velayudhan, D. A. Halvorson, and K. V. Nagaraja. 2002. Survival of *Ornithobacterium rhinotracheale* in sterilized poultry litter. *Avian Diseases* 46: 1011-14.
6. Naeem, K., A. Malik, and A. Ullah. 2003. Seroprevalence of *Ornithobacterium rhinotracheale* in chickens in Pakistan. *The Veterinary Record* 153: 533-34.
7. Tsai, HJ Hsiang PH. The prevalence and microbial susceptibilities of Salmonella and Campylobacter in ducks in Taiwan. *J Vet Med Sci*. 2005; 67(1):7-12.
8. Turan, N., and S. Ak. 2002. Investigation of the presence of *Ornithobacterium rhinotracheale* in chickens in Turkey and determination of the seroprevalence of the infection using the Enzyme-Linked Immunosorbent Assay. *Avian Diseases* 46: 442-46.
9. van den Bosch, G. 2001. "*Ornithobacterium rhinotracheale*: the current status." Web page. Available at <http://www.poultry-health.com/fora/turkhelth/turtec24/vdbosch.htm>.
10. van Empel, P. C. M., and H. M. Hafez. 1999. *Ornithobacterium rhinotracheale*: a review. *Avian Pathology*. 28: 217-27.
11. van Veen, L., M. Vrijenhoek, and P. van Empel. 2004. Studies of the transmission routes of *Ornithobacterium rhinotracheale* and immunoprophylaxis to prevent infection in young meat turkeys. *Avian Diseases* 48: 233-37.
12. Varga, J., L. Fodor, and L. Makrai. 2001. Characterisation of some *Ornithobacterium rhinotracheale* strains and examination of their transmission via eggs. *Acta Veterinaria Hungarica* 49, no. 2: 125-30.

6 Risk assessments – alkalised eggs

6.1 Notifiable avian influenza viruses

6.1.1 Technical information

Background

Avian influenza viruses (AIV) circulate in wild and domestic birds. Most AIV are of low pathogenicity, producing either subclinical disease or mild respiratory or reproductive disease in domestic birds. Notifiable forms of AI viruses do, however, exist, which are either highly pathogenic, or of low pathogenicity but with the potential to mutate to highly pathogenic forms.

Highly pathogenic notifiable avian influenza (HPNAI), formerly known as fowl plague, is a highly contagious systemic disease of poultry that causes high mortality in domestic chickens. HPNAI viruses have been documented to arise from mutations in low pathogenicity notifiable AI viruses (LPNAI) viruses, with mutations probably occurring within domestic poultry populations (Swayne and Suarez 2000; Garcia et al. 1996).

Although outbreaks in Australia have been recorded, Australia is currently free of HPNAI. The definition of notifiable avian influenza (NAI) viruses was recently reviewed by the OIE and is described below (World Organisation for Animal Health (OIE) 2005).

Agent characteristics

AIV are single-stranded, enveloped RNA viruses of the *Orthomyxoviridae* family. The influenza viruses that constitute this family are classified into types A, B or C based on differences between their nucleoproteins and matrix protein antigens. All AIV belong to type A (Easterday, Hinshaw, and Halvorson 1997).

Influenza A viruses are divided into subgroups according to serologic reactions to their haemagglutinin (HA) and neuraminidase (NA) surface antigens. Sixteen haemagglutinins (H1–H16) and nine neuraminidases (N1–N9) have been identified to date (Swayne and Halvorson 2003; Fouchier et al. 2005). The HA gene is the primary determinant of pathogenicity in chickens.

All reported outbreaks of HPNAI in poultry have been of the H5 or H7 subtypes, with the exception of two H10 isolates that fulfilled some criteria for classification as highly pathogenic (Wood et al. 1996). Many H5 and H7 subtypes isolated from poultry have been of low or mild pathogenicity (Swayne and Suarez 2000; Swayne and Halvorson 2003). However, because of the risk of an H5 or H7 virus becoming virulent by mutation, all H5 and H7 viruses have been identified as NAI viruses (World Organisation for Animal Health (OIE) 2005; Alexander 2004).

For the purposes of the OIE Code (World Organisation for Animal Health (OIE) 2007), 'avian influenza in its notifiable form (NAI) is defined as an infection of poultry caused by any influenza virus of the H5 or H7 subtypes, or by any AI virus with an intravenous

pathogenicity index (IVPI) greater than 1.2 (or as an alternative at least 75% mortality) as described below. NAI viruses can be divided into HPNAI and LPNAI:

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

Table 8. Resistance of agent to physical and chemical action

Temperature	Most strains inactivated by 56 °C for 3 hours; 60 °C for 30 minutes
pH:	Inactivated by acid pH
Chemicals:	Inactivated by oxidising agents, sodium dodecyl sulphate, lipid solvents, β -propiolactone
Disinfectants	Inactivated by formalin and iodine compounds
Survival:	Remains viable for long periods in tissues, faeces, and also in water

(World Organisation for Animal Health (OIE) 2002)

NAI viruses are generally stable at pH 11 and may require a pH of 12 or more to achieve inactivation (Moses, Brandly, and Jones 1947). NAI virus on the external surface of an egg shell will be directly exposed to the alkalisising solution at pH 13 for 35 days, and it is considered likely that it would be inactivated. However, as the pH of the egg yolk may only reach 9.5 following the alkalisisation process, it is not possible to conclude that virus within the egg yolk or albumen would be inactivated by the process.

Epidemiology

The epidemiology of AIV has been previously reviewed in the *Draft Generic Import Risk Analysis Report for Chicken Meat* (Biosecurity Australia 2006). AIV are distributed worldwide in many species of domestic and wild birds, including chickens, turkeys, domestic and wild waterfowl and game birds, passerines, psittacines, raptors and ratites (Easterday, Hinshaw, and Halvorson 1997; Swayne and Halvorson 2003). Wild birds, particularly wild aquatic birds such as ducks, gulls and shorebirds, are believed to provide a reservoir of AIV, with asymptomatic enteric infections leading to faecal shedding of virus. H5 and H7 subtypes are found sporadically in ducks, shorebirds and gulls, although other HA types are more

commonly isolated from these groups of birds (Sharp et al. 1993; Fouchier et al. 2003; Munster et al. 2005).

In late 2002, some H5N1 strains in Asia became highly pathogenic in ducks, killing many ducks as well as other wild waterfowls (Sturm-Ramirez et al. 2004). In an experimental study, some ducks exhibited clinical signs of infection, including neurological signs and mortality, when challenged with H5N1 viruses isolated from poultry and humans in Vietnam in 2003/2004 (Hulse-Post et al. 2005). Those ducks that survived the infection shed virus for up to 17 days.

Virus is circulated in waterfowl by the faecal-oral route, with the virus persisting in bodies of water for variable periods of time from 9 to 100 days (Stallknecht et al. 1990). Domestic birds, including poultry, are infected through direct contact with wild birds, or through faecal contamination of water or feed supplies (Swayne and Suarez 2000), and spread of infection between farms can occur mechanically by people or fomites, or by aerosol.

In the absence of good biosecurity, non-migratory wild bird species may also represent a risk of transmitting H5N1 locally between poultry farms (FAO Technical Task Force on Avian Influenza 2005). The likelihood of transmission of HPNAI virus by wild passerine birds was examined using an isolate from an Australian outbreak. In an experimental study using a Victorian strain of HPNAI virus (A/Chicken/Vic/1/85 (H7N7)), inoculated sparrows had a 30% mortality rate, with surviving birds shedding virus in the faeces, while inoculated starlings had 100% mortality (Nestorowicz et al. 1987). The authors concluded that infection in starlings would have been self-limiting, but sparrows may have been effective at transmitting the virus.

Transmission in eggs

AIV can be present within, or on the surface of, eggs laid by naturally-infected hens. H5N2 virus was isolated from the albumen, the yolk and the shell surface of infertile eggs laid by infected hens during the 1983-84 outbreak of HPNAI in Pennsylvania, US (Cappucci et al. 1985). Data reported in the same study indicated that the virus can survive for at least several days in the albumen and yolk of eggs stored at 10-18 °C. There is generally a large drop in egg production in flocks experiencing an outbreak of HPNAI; however, influenza virus has been isolated from clinically unaffected birds during an outbreak (Cappucci et al. 1985). Most eggs laid during an outbreak of HPNAI were of market quality; however, approximately 10% were thin or soft shelled or abnormally small (Cappucci et al. 1985).

In an experimental study using HPNAI H5N2 to infect mature white leghorn hens, the virus was also recovered from 12 of 14 eggs laid on the third day post-inoculation (Beard, Brugh, and Johnson 1984).

There is no available data on egg transmission of HPNAI or LPNAI in duck eggs. However, given that HPNAI causes a systemic disease in both chickens and ducks with egg transmission in hen eggs, it is highly probable that HPNAI and LPNAI would be present in duck eggs.

Quarantine significance

NAI is notifiable to the OIE, and trade in potentially affected animals or animal products is subject to international controls.

Avian influenza is notifiable in all Australian States and Territories. Australia's policy in the event of an outbreak is eradication as detailed in AUSVETPLAN (Animal Health Australia 2005). Australia's Emergency Animal Disease Response Agreement currently includes HPNAI in category 3. Diseases in this category are considered to be of moderate public impact, having the potential to cause significant (but generally moderate) national socio-economic consequences through international trade losses, market disruptions involving two or more States, and severe production losses to affected industries. Category 3 diseases have minimal or no effects on human health or the environment and costs are funded 50% by government and 50% by industry (Animal Health Australia 2002). The categorisation of HPNAI and LPNAI under the Emergency Animal Disease Response Agreement is currently under review, particularly in the light of the human health consequences of H5N1 AI.

There have been five outbreaks of HPNAI reported in Australia, the most recent of which occurred in 1997. This outbreak cost A 4.445 million and involved the destruction of 310,565 chickens, 1.23 million fertile chicken eggs, 261 emu chicks and 147 emu eggs. The flow-on effects to industry were considerable (Selleck et al. 2003). Australia is currently free of HPNAI, and there is no evidence of LPNAI in the Australian commercial poultry flock.

Eradication of HPNAI in Hong Kong in 1997 and the US in 1983–85 cost \$US 12 million and \$US 63 million in government funds, and involved the depopulation of 1.4 million and 17 million birds respectively (Swayne and Suarez 2000). The outbreak in the US is estimated to have cost an additional \$US 350 million in increased consumer costs (Galyon and Roth 2003), and resulted in an increase in retail egg prices of more than 30% (Animal and Plant Health Inspection Service 1995).

In 1999–2000 HPNAI in Italy resulted in the destruction of more than 13 million birds, and interrupted activities of establishments such as hatcheries, feed mills, abattoirs, and processing plants. Disruptions to the marketing system caused unemployment and heavy economic loss both to the poultry industry and the community (Capua et al. 1999). Recent outbreaks of HPNAI in the Netherlands, Belgium and Germany resulted in the culling of over 25 million commercial birds, disruptions to exports of breeding stock, hatching eggs and chicks, and disruptions to the supply of commercial meat and eggs for the European Union and export markets (Shane 2003). The costs to the Netherlands Government for control of the 2003 HPNAI outbreak were reported to be €270 million, and costs related to industry and trade disruption were estimated at €500 million (Weijtens 2006). HPNAI outbreaks in Asia and Europe in 2003–07 continue to have significant effects on domestic and international trade at the time of writing, with domestic sales of poultry meat declining sharply in many affected countries. Over 150 million birds have been destroyed/culled in Asia alone. It is estimated that total poultry farm losses in Asia in 2004 were in excess of \$10 billion (FAO Newsroom 2005). An outbreak of H5N1 avian influenza in the UK in February 2007 led to the slaughter of approximately 160,000 turkeys [ProMed email "Avian influenza (32): UK (England), China (Hong Kong), Turkey"].

Outbreaks of LPNAI in poultry can also be costly in terms of production losses, costs of eradication and loss of export markets. An outbreak of LPNAI (H7N2) in Virginia in 2002 is estimated to have cost the poultry industry approximately US\$130 million, and resulted in the depopulation of 4.7 million birds (Akey 2003). The United States has experienced trade embargos associated with the presence of LPNAI viruses in the north-eastern United States (Myers, Rhorer, and Clifford 2003).

In some circumstances, some strains of NAI may be transmitted from infected poultry to humans, causing illness and death (World Health Organization 2003; Koopmans et al. 2004; World Health Organization 2004b; Perdue and Swayne 2005). The risk of transmission from poultry to humans is greatest when infected birds have close contact with family members (e.g. entering the family home), multiple species of animals are farmed in the same location, untreated chicken faeces are used as fertilizer, sick or dead birds are inappropriately disposed of, and birds or their meat are marketed in unregulated live bird markets ((FAO/OIE/WHO 2005). The husbandry and marketing systems that have contributed to human infections in the outbreaks in Asia do not occur in Australia, which has a lower likelihood of widespread human contact with poultry.

It is expected that personnel working with HPNAI-infected flocks under conditions such as occurred in the Netherlands would use appropriate protective equipment and take recommended precautions against infection (World Health Organization 2004a). Nevertheless, it is acknowledged that compliance with preventive measures amongst poultry workers during culling operations is highly variable, and that transmission of virus to humans in such circumstances may occur (Bosman, Meijer, and Koopmans 2005).

6.1.2 Risk assessment – Highly pathogenic avian influenza (HPNAI)

Taiwan has never reported infection with HPNAI to the OIE (OIE 2006). While this remains the case, the likelihood of introduction of HPNAI in alkalised duck eggs from Taiwan is negligible. Given the high levels of infection with HPNAI which are currently being experienced in many parts of Asia, it is possible that HPNAI will be reported in Taiwan in the future. For this reason, Biosecurity Australia has conducted a risk assessment for this disease, to guide decision making should an outbreak occur. The report of the risk assessment is at Appendix 1.

6.1.3 Risk assessment – Low pathogenicity notifiable avian influenza (LPNAI)

Release assessment

In a letter of May 2005, Taiwan stated that it is free from HPNAI but has had some cases of LPNAI (January to 9 March 2004).

Ducks can be infected with LPNAI and not exhibit clinical signs, or only display mild respiratory and reproductive disease. Biosecurity Australia considers that LPNAI could exist in a population for some time and could spread widely before being recognised, especially where disease recognition relies on passive rather than active surveillance; therefore in a country where LPNAI is endemic in ducks the likelihood that a source flock will be infected with LPNAI was considered to be **moderate**.

AI virus can be present in or on the surface of eggs laid by naturally infected hens, and the virus can survive for at least several days in the albumen and yolk of eggs stored at 10-18 °C. While there is no available data on egg transmission of LPNAI in duck eggs, given that LPNAI causes reproductive disease in both chickens and ducks, it is considered probable LPNAI would be present in infected duck eggs. Therefore, LPNAI could be present in, or on, a proportion of eggs laid by ducks with or without clinical signs in an infected flock.

Based on the above information, the overall likelihood that LPNAI would enter Australia through the importation of alkalised duck eggs from Taiwan was assessed as **moderate**.

Exposure assessment

As explained in the Chapter on Methods, low biosecurity backyard poultry and wild birds are the only exposure groups at significant risk.

Preserved eggs are a delicacy item and it is assumed that minimal waste would result from the purchase and consumption of these products. It is likely that only a small proportion of those households that keep backyard poultry and feed them kitchen scraps would also purchase preserved duck eggs from Taiwan. The fraction of the egg that does become available will consist mostly of shell, and would have been directly exposed to the alkalising agent at pH 13. The smaller quantity of waste that consists of egg fragments however, may not have reached a pH of greater than 9.5, and cannot be confidently expected to be completely free of viable virus. Waste generated from restaurant use will generally be disposed of in municipal refuse dumps, while waste from domestic consumption could be fed to backyard poultry or added to household composts. It is considered that only a small percentage of imported preserved eggs will eventually become available to susceptible exposure groups.

LPNAI, within egg waste, is likely to survive in the environment under ambient temperatures for several days, giving ample time for wild birds to locate and scavenge accessible material. The time between feeding of scraps and consumption by low biosecurity backyard poultry is likely to be very short, so environmental degradation of the disease agent will be minimal.

Overall, it is assessed that the likelihoods that backyard poultry (PLEbp) and wild birds (PLEwb) would be exposed to an infectious dose of LPNAI virus are **low**.

Consequence assessment

Likelihood of establishment and spread

The likelihood of establishment and spread of LPNAI in Australian poultry has been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the likelihood that LPNAI would spread from the primary exposure groups to cause disease in commercial poultry in Australia following exposure of susceptible Australian species to food scraps containing the organism as **moderate** and **very low** respectively, for backyard poultry and wild birds.

Impacts of establishment and spread

The impacts of establishment and spread of LPNAI in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the impact of LPNAI spreading to commercial poultry in Australia as **moderate**.

Risk estimation

After combining the likelihoods of entry (LR) and exposure (PLEbp and PLEwb), as detailed on page 27, the partial annual likelihoods of entry and exposure for backyard poultry and wild birds (PALEEbp and PALEEwb respectively) were estimated as **low**.

Combining these with the likelihoods of establishment and spread (PLESbp and PLESwb) as discussed on page 27, resulted in estimates of **low** and **very low** (respectively) for the partial annual likelihoods of entry, exposure, establishment and spread (PALEEESbp and PALEEESwb).

Combining this with the impact of the establishment of the disease, as detailed on page 27, provided partial annual risk estimates of **low** for backyard poultry, and **very low** for wild birds.

Finally, combining the partial annual risks for backyard poultry and wild birds according to the rules detailed on page 28 yielded a final risk for LPNAI of **low**.

As this unrestricted risk estimate does not achieve Australia's (ALOP)¹, risk management was considered necessary for this disease agent.

6.1.4 Reference List

1. Akey, B. L. 2003. Low-pathogenicity H7N2 avian influenza outbreak in Virginia during 2002. *Avian Diseases* 47: 1099-103.
2. Alexander, D. J. 2004. Avian Influenza (version adopted May 2005). In *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. 5th ed., OIE, <http://www.oie.int/eng/normes/mmanual/A_00037.htm>. Office International des Epizooties.
3. Animal and Plant Health Inspection Service. 1995. "Highly pathogenic avian influenza: a threat to U.S. poultry." Web page, [accessed June 2003]. Available at <http://www.aphis.usda.gov/oa/pubs/broai.pdf>.
4. Animal Health Australia. 1995. "Animal Health in Australia 1994." Web page. Available at <http://www.aahc.com.au/status/ahiareport>.
5. ———. 1998. *Animal Health in Australia 1997*, Editors L. Lehane, M. J. Nunn, and P. M. Thornber. Department of Primary Industries and Energy, Canberra, Australia.
6. Animal Health Australia. 2002. "The Emergency Animal Disease Response Agreement." Web page, [accessed June 2003]. Available at <http://www.aahc.com.au/eadp/deed.pdf>.
7. Animal Health Australia. 2005. "AUSVETPLAN 2005 Disease Strategy: Avian influenza (Edition 3.1)." Web page, [accessed November 2005]. Available at http://www.animalhealthaustralia.com.au/programs/eadp/ausvetplan_home.cfm.
8. Arzey, G. 2004. The role of wild aquatic birds in the epidemiology of avian influenza in Australia. *Australian Veterinary Journal* 82, no. 6: 377-78.
9. Beard, C. W., M. Brugh, and D. C. Johnson. 1984. Laboratory studies with the Pennsylvania avian influenza viruses (H5N2). In *Proceedings of the 88th Annual Meeting of the United States Animal Health Association* United States Animal Health Association, 462-73USA: United States Animal Health Association.
10. Bosman, A., Meijer, A., and Koopmans, M. 2005. "Final analysis of Netherlands avian influenza outbreaks reveals much higher levels of transmission to humans than previously thought." Web page, [accessed 28 January 2005]. Available at <http://www.eurosurveillance.org/ew/2005/050106.asp>.
11. Cappucci, D. T. Jr., D. C. Johnson, M. Brugh, T. M. Smith, C. F. Jackson, J. E. Pearson, and D. A. Senne. 1985. Isolation of avian influenza virus (subtype H5N2) from chicken eggs during a natural outbreak. *Avian Diseases* 29, no. 4: 1195-200.
12. Capua, I., S. Marangon, L. Selli, D. J. Alexander, D. E. Swayne, M. D. Pozza., E. Parenti, and F. M. Cancellotti. 1999. Outbreaks of highly pathogenic avian influenza (H5N2) in Italy during October 1997-January 1998. *Avian Pathology* 28: 455-60.
13. Easterday, B. C., V. S. Hinshaw, and D. A. Halvorson. 1997. Influenza. In *Diseases of Poultry*. 10th ed., Editors B. W. Calnek, H. J. Banes, C. W. Beard, L. R. McDougald, and Y. M Saif, 583-605. London, UK: Mosby-Wolfe.
14. FAO Newsroom. 2005. "Fighting bird flu at its origin to prevent a human flu pandemic." Web page, [accessed February 2005]. Available at <http://www.fao.org/newsroom/en/news/2005/89912/index.html>.
15. FAO/OIE/WHO. 2005. "FAO/OIE/WHO consultation on avian influenza and human health: risk

- reduction measures in producing, marketing and living with animals in Asia." Web page, [accessed August 2005]. Available at <http://www.fao.org/ag/againfo/subjects/documents/ai/concmalaysia.pdf>.
16. FAO Technical Task Force on Avian Influenza. 2005. "FAOAIDEnews: Update on the avian influenza situation (as of 15/02/2005) - Issue no. 28." Web page, [accessed February 2005]. Available at <http://www.fao.org/docs/eims/upload/174499/AVIbull028.pdf>.
 17. Fouchier, R. A., V. Munster, A. Wallensten, T. M. Bestebroer, S. Herfst, D. Smith, G. F. Rimmelzwaan, B. Olsen, and A. D. Osterhaus. 2005. Characterisation of a novel Influenza A virus haemagglutinin subtype (H16) obtained from black headed gulls. *Journal of Virology* 79, no. 5: 2814-22.
 18. Fouchier, R. A., B. Olsen, T. M. Bestebroer, S. Herfst, L. van der Kemp, G. F. Rimmelzwaan, and A. D. Osterhaus. 2003. Influenza A virus surveillance in wild birds in Northern Europe in 1999 and 2000. *Avian Diseases* 47, no. 3s: 857-60.
 19. Galyon, J. and Roth, J. "Highly pathogenic avian influenza in Hong Kong, 2001." Web page, [accessed 14 August 2003]. Available at <http://www.vetmed.iastate.edu/services/institutes/iicab/fad/overview3/avianflu.htm>.
 20. Garcia, M., J. M. Crawford, J. W. Latimer, E. Rivera-Cruz, and M. L. Perdue. 1996. Heterogeneity in the haemagglutinin gene and emergence of the highly pathogenic phenotype among recent H5N2 avian influenza viruses in Mexico. *Journal of General Virology* 77: 1493-504.
 21. Homme, P. J. , and B. C. Easterday. 1970. Avian influenza virus infections. I. Characteristics of Influenza A/Turkey/Wisconsin/1966 virus. *Avian Diseases* 14: 66-74.
 22. Hulse-Post, D. J., K. M. Sturm-Ramirez, J. Humberd, P. Seiler, E. A. Govorkova, S. Krauss, and et. al. 2005. Role of domestic ducks in the propagation and biological evaluation of highly pathogenic H5N1 viruses in Asia. *Proceedings of the National Academy of Sciences of the United States of America* 102, no. 30: 10682-87.
 23. Keawcharoen, J., K. Oraveerakul, T. Kuiken, R. A. Fouchier, A. Amonsin, and S. Payungporn. 2004. Avian influenza H5N1 in tigers and leopards. *Emerging Infectious Diseases* 10, no. 12: 2189-91.
 24. Koopmans, M., B. Wilbrink, M. Conyn, G. Natrop, H. van der Nat, H. Vennema, A. Meijer, J. van Steenberg, R. Fouchier, A. Osterhaus, and A. Bosman. 2004. Transmission of H7N7 avian influenza A virus to human beings during a large outbreak in commercial poultry farms in the Netherlands. *The Lancet* 363: 587-93.
 25. Kuiken, T., G. Rimmelzwaan, D. van Riel, G. van Amerongen, M. Baars, R. Fouchier, and A. Osterhaus. 2004. Avian H5N1 influenza in cats. *Scienceexpress* 10.1126/science.1102287: 3 pages.
Notes: available at www.sciencexpress.org
 26. Lang, G., O. Narayan, B. T. Rouse, A. E. Ferguson, and M. C. Connell. 1968a. A new influenza A virus infection in turkeys II. A highly pathogenic variant, A/Turkey/Ontario 7732/66. *Canadian Veterinary Journal* 9, no. 7: 151-60.
 27. Lang, G., B. T. Rouse, O. Narayan, A. E. Ferguson, and M. C. Connell. 1968b. A new influenza virus infection in turkeys I. Isolation and characterization of virus 6213. *Canadian Veterinary Journal* 9, no. 1: 22-29.
 28. Lu, H., A. E. Castro, K. Pennick, J. Liu, P. Dunn, D. Weinstock, and D. Henzler. 2003. Survival of avian influenza virus H7N2 in SPF chickens and their environments. *Avian Diseases* 47: 1015-21.

29. Morgan, I. R., and A. P. Kelly. 1990. Epidemiology of an avian influenza outbreak in Victoria in 1985. *Australian Veterinary Journal* 67, no. 4: 125-28.
30. Moses, H. E. , C. A. Brandly, and E. E. Jones. 1947. The pH stability of viruses of Newcastle disease and fowl plague. *Science* 105: 477-479.
31. Munster, V. J., A. Wallensten, C. Baas, G. F. Rimmelzwaan, M. Schutten, B. Olsen, A. D. Osterhaus, and R. A. Fouchier. 2005. Mallards and highly pathogenic avian influenza ancestral viruses, Northern Europe. *Emerging Infectious Diseases* 11, no. 10: in press.
32. Myers, T. J., M. D. A. Rhorer, and J. Clifford. 2003. USDA options for regulatory changes to enhance the prevention and control of avian influenza. *Avian Diseases* 47: 982-87.
33. Nestorowicz, A., Y. Kawaoka, W. J. Bean, and R. G. Webster. 1987. Molecular analysis of the hemagglutinin genes of Australian H7N7 influenza viruses: role of passerine birds in maintenance or transmission? *Virology* 160: 411-18.
34. OIE. "Disease information published during the past eighteen months. Index by disease." Web page, [accessed 15 November 2006]. Available at http://www.oie.int/eng/info/hebdo/a_dsum.htm.
35. Perdue, M. L., and D. E. Swayne. 2005. Public health risk from avian influenza viruses. *Avian Diseases* 49: 317-27.
36. ProMED-mail. 2004. Avian influenza, human - East Asia (50): Thailand. *ProMED-Mail* 2004 20041001.2708: <<http://www.promedmail.org>>.
37. ———. 2005. Avian Influenza - Asia (12): Vietnam, civets, H5N1. *ProMED-Mail* 2005 20050826.2527: <<http://www.promedmail.org>>.
38. ———. 2006. Avian Influenza - worldwide(47): Europe. *ProMED-Mail* 2006 20060309.0752: <<http://www.promedmail.org>>.
39. Rimmelzwaan, G. F., D. van Riel, M. Baars, T. M. Bestebroer, G. van Amerongen, R. A. M. Fouchier, A. D. Osterhaus, and T. Kuiken. 2006. Influenza A virus (H5N1) infection in cats causes systemic disease with potential novel routes of virus spread within and between hosts. *American Journal of Pathology* 168, no. 1: 176-83.
40. Selleck, P. W., G. Arzey, P. D. Kirkland, R. L. Reece, A. R. Gould, P. W. Daniels, and H. A. Westbury. 2003. An outbreak of highly pathogenic avian influenza in Australia in 1997 caused by an H7N4 virus. *Avian Diseases* 47: 806-11.
41. Selleck, P. W., L. J. Gleeson, P. T. Hooper, H. A. Westbury, and E. Hansson. 1997. Identification and characterisation of an H7N3 influenza A virus from an outbreak of virulent avian influenza in Victoria. *Australian Veterinary Journal* 75, no. 4: 289-92.
42. Shane, S. M. 2003. Disease continues to impact the world's poultry industries. *World Poultry* 19, no. 7: 22-23.
43. Sharp, G. B., Y. Kawaoka, S. M. Wright, B. Turner, V. Hinshaw, and R. G. Webster. 1993. Wild ducks are the reservoir for only a limited number of influenza A subtypes. *Epidemiology and Infection* 110: 161-76.
44. Stallknecht, D. E., M. T. Kearney, S. M. Shane, and P. J. Zwank. 1990. Effects of pH, temperature, and salinity on persistence of avian influenza viruses in water. *Avian Diseases* 334: 412-18.
45. Sturm-Ramirez, K. M., T. Ellis, B. Bousfield, L. Bissett, K. Dyrting, J. E. Rehg, L. Poon, Y. Guan, M. Peiris, and R. G. Webster. 2004. Reemerging H5N1 influenza viruses in

- Hong Kong in 2002 are highly pathogenic to ducks. *Journal of Virology* 78, no. 9: 4892-901.
46. Swayne, D. E., and D. A. Halvorson. 2003. Influenza. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 135-60. Iowa, USA: Iowa State Press.
 47. Swayne, D. E., and D. L. Suarez. 2000. Highly pathogenic avian influenza. *Revue Scientifique Et Technique Office International Des Epizooties* 19, no. 2: 463-82.
 48. Tracey, J. P., R. Woods, D. Roshier, P. West, and G. R. Saunders. 2004. The role of wild birds in the transmission of avian influenza for Australia: an ecological perspective. *Emu* 104: 109-24.
 49. Weijtens, M. 2006. "Avian influenza in the Netherlands - Outbreak 2003 - Regionalisation." Web page, [accessed February 2006]. Available at http://www.wto.org/english/tratop_e/sps_e/meet_jan06_e/meet_jan06_e.htm.
 50. Wood, G. W., J. Banks, I. Strong, G. Parsons, and D. J. Alexander. 1996. An avian influenza virus of H10 subtype that is highly pathogenic for chickens, but lacks multiple basic amino acids at the haemagglutination cleavage site. *Avian Pathology* 25: 799-806.
 51. World Health Organization. 2003. "Influenza A (H5N1) in Hong Kong Special Administrative Region of China – Update 2." Web page, [accessed 19 June 2003]. Available at http://www.who.int/csr/don/2003_02_27a/en/.
 52. World Health Organization. 2004a. "WHO interim recommendations for the protection of persons involved in the mass slaughter of animals potentially infected with highly pathogenic avian influenza viruses." Web page, [accessed 21 July 2004a]. Available at http://www.who.int/csr/disease/avian_influenza/guidelines/en/Avian%20Influenza.pdf.
 53. World Health Organization. 2004b. "Avian influenza and human health. Report by the WHO Secretariat for 114th Session of the WHO Executive Board, April 2004." Web page, [accessed January 2005b]. Available at http://www.who.int/gb/ebwha/pdf_files/EB114/B114_6-en.pdf.
 54. World Organisation for Animal Health (OIE). 2002. "Technical Disease Cards - A150 - Highly Pathogenic Avian Influenza." Web page, [accessed 1 March 2004]. Available at http://www.oie.int/eng/maladies/fiches/a_A150.htm.
 55. World Organisation for Animal Health (OIE). 2003. "Draft report of the meeting of the OIE *ad hoc* group on avian influenza." Web page, [accessed January 2005]. Available at http://www.oie.int/eng/AVIAN_INFLUENZA/AHG_AI_Nov2003.pdf.
 56. World Organisation for Animal Health (OIE). 2004. "Terrestrial Animal Health Code Chapter 2.7.12 Highly Pathogenic Avian Influenza." Web page, [accessed February 2005]. Available at http://www.oie.int/eng/normes/mcode/en_chapitre_2.7.12.htm.
 57. World Organisation for Animal Health (OIE). 2007. "Terrestrial Animal Health Code 2005 Chapter 2.7.12 Avian Influenza." Web page, [accessed August 2005]. Available at http://www.oie.int/eng/normes/mcode/en_chapitre_2.7.12.htm.

6.2 Newcastle disease virus

6.2.1 Technical information

Background

Strains of Newcastle disease virus (NDV) vary greatly in their virulence and tissue tropism and, in susceptible birds, infection induces a wide range of clinical signs and pathological lesions. Based on the severity of the disease produced in infected chickens, NDV strains are broadly classified as velogenic (highly virulent), mesogenic (moderately virulent) and lentogenic (mildly virulent) (Alexander 2003).

The natural hosts of NDV are domestic poultry, including chickens, turkeys, ducks, geese, pigeons, quail, pheasants, guinea fowl and ostriches, and many species of captive caged birds and wild birds (Alexander 2000). Susceptibility varies between species, with chickens the most likely to show clinical ND, and water birds the least likely to be affected clinically (Kaleta and Baldauf 1988). Infection with NDV has also been reported in reptiles such as crocodiles, snakes and lizards (Kouwenhoven 1993). Human infections with NDV are generally mild and conjunctivitis associated with NDV has been reported in workers in laboratories and poultry processing plants (Chang 1981).

ND is an OIE-listed disease. Although outbreaks in Australia have been recorded, Australia is currently considered free of virulent ND, with vaccination, in accordance with the OIE definition of a ND-free country.

Agent characteristics

Resistance to physical and chemical agents is as follows. NDV has been shown to be readily inactivated at pH < 2, or > 11. However, the internal pH of alkalised eggs can only be guaranteed to reach a pH of 9.5 so any NDV contained within the egg can not be considered to be reliably inactivated. However, any NDV contaminating the external surface of the shell would be inactivated by the alkalisising process. (World Organisation for Animal Health (OIE) 2002b; Beard and Hanson 1984; Lancaster 1963a; Lancaster 1963b)

Table 9. Resistance of NDV to physical and chemical action

pH:	Readily inactivated by high and low pH (<pH 2 and > pH 11)
Chemicals:	Inactivated by lipid solvents (chloroform, ether, alcohol)
Disinfectants:	Inactivated by a wide range of chemicals, including chlorine-based disinfectants, lipid solvents, formalin, propiolactone and phenol.
Survival:	Inactivated by direct sunlight within 30 minutes, but in cool weather can retain infectivity in faeces and contaminated poultry sheds for up to 21 days. Survives frozen in bone marrow and muscle > 300 days; at 5 °C for >134 days Survives in lake water for up to 19 days; in soil for up to 22 days.

Epidemiology

NDV has a worldwide distribution. However, the prevalence of ND is difficult to estimate given the widespread use of live ND vaccines, problems with the diagnosis and reporting of ND and the presence of strains of low virulence to chickens in some countries (Alexander 2000).

ND is highly contagious and transmission of virus is frequently by direct contact with diseased or carrier birds. Virus is excreted from the respiratory tract and in the faeces for at least one day before clinical signs become apparent (Sinha, Hanson, and Brandly 1954) and birds can be infected both by inhalation of aerosols or by ingestion. Infection from fomites such as chicken crates, egg flats, contaminated feed, trucks, dust, humans, other animals, feathers and clothing is important in the spread of the disease in an outbreak (Lancaster and Alexander 1975; Alexander 2000). There are conflicting reports of the susceptibility of mammals including cats, foxes, dogs, pigs and rats to infection with NDV but it is probable they act as mechanical carriers only (Lancaster 1963a; Asplin 1949; Lancaster 1963b). Windborne transmission of NDV has been postulated and virus has been recovered from the air up to 64 metres from infected premises (Hugh-Jones et al. 1973). Contaminated vaccines and contaminated water have also been implicated in spread of virus during epizootics (Alexander 2003).

Infection with NDV has been reported in 241 species of birds. Susceptibility varies between species and many may be infected with NDV without showing signs of disease (Kaleta and Baldauf 1988). Ducks can become infected but do not show clinical signs even when infected with strains that are pathogenic to chickens (Kaleta and Baldauf 1988). Ducks, both wild and domestic, can act as carriers (Gough et al. 1988); (Alexander 2000); (Gilchrist 1993); (Wobeser GA 1997). Psittacine birds may excrete virus in their faeces for more than a year after recovery from clinical disease (Kouwenhoven 1993; Erickson et al. 1977). Wild birds may serve as a significant reservoir of NDVs and may introduce the virus to poultry. They may also be responsible for transmitting infection within an area, following the infection of poultry (Alexander 2000; Gilchrist 2005). Several species of introduced and native wild birds, found in Australia, have been shown to be susceptible to infection with NDV (Gilchrist 1993) and viruses of low virulence have been isolated from wild birds in Western Australia (Alexander, Mackenzie, and Russell 1986) and Victoria (Peroulis-Kourtis et al. 2002; Peroulis and O'Riley 2004). Sampling of wild bird populations suggests that the virulent NDV strains that have been isolated from poultry are antigenically distinct from the avirulent ND strains circulating in Australian wild birds (Gould et al. 2001; Peroulis and O'Riley 2004).

Transmission in eggs

Little is known about the deposition of ND viruses in eggs. Velogenic NDV has been sporadically identified in eggs of infected hens based on recovery of viruses from hatched chicks or from cell cultures prepared from embryos of infected hens (Capua et al. 1993); (Chen Juipin and Wang Chingho 2002). NDV has been demonstrated in and on eggs (Williams and Dillard 1968; Lancaster 1963a) and in the reproductive tract of hens (Biswal and Morrill 1954). There is some evidence to suggest that egg transmission may occur (Hofstad 1949) (Bivins, Rhodes-Miller, and Beaudette 1950; Zagar and Pomeroy 1950; French, St George, and Percy 1967; Collins, Gough, and Alexander 1993) but egg

transmission of virulent strains was not considered to be of epidemiological significance in chickens as birds quickly ceased laying and infected embryos died (Beard and Hanson 1984). However, recent studies have shown immune hens challenged with virulent NDV may lay contaminated eggs. While no virus was isolated from the shell in these studies, challenge virus was isolated from the albumen of one of 187 eggs produced within two weeks of challenge (Australian Animal Health Laboratory 2002). Capua et al. (2003) reported a case of casual isolation of NDV from embryonated eggs.

Egg production may decrease in chickens infected with virulent NDV, and eggs may be malformed or have defective shells. Changes in yolk and albumen quality were reported (Monlux 1972).

While there is no data available on NDV transmission in duck eggs, it is likely that transmission would be similar to that in hen eggs. Because ducks are not usually clinically affected by strains virulent to chickens it is unlikely that egg production would be affected by infection with NDV.

Quarantine significance

ND is an OIE-listed disease, and trade in potentially affected animals or animal products is subject to international controls.

ND is notifiable in all Australian States and Territories. Australia's policy in the event of an outbreak is eradication and is detailed in AUSVETPLAN (Animal Health Australia 2004). Australia's Emergency Animal Disease Response Agreement includes ND in category 3. Diseases in this category are considered to be of moderate public impact, having the potential to cause significant (but generally moderate) national socio-economic consequences through international trade losses, market disruptions involving two or more States, and severe production losses to affected industries. Category 3 diseases cause minimal or no effect on human health or the environment and costs are funded 50% by government and 50% by industry (Animal Health Australia 2002).

A series of outbreaks of ND of endemic origin occurred in Australia between 1998 and 2002 (World Organisation for Animal Health (OIE) 2002a). Following the outbreaks in 2002, a National Management Plan for ND was developed and instigated (Animal Health Australia 2005). The basis for the Plan is compulsory national vaccination of all commercial poultry flocks with structured surveillance and standard operating procedures and programs for virulent NDV. Under the Standard Operating Procedures, meat chickens are vaccinated against ND at between one and 14 days of age. The Plan aims to maintain Australia's virulent ND-free status.

Direct costs of the outbreak at Mangrove Mountain, NSW, in 1999 to government and industry were \$26.4 million and involved destruction of 1.9 million birds on commercial poultry farms and backyard properties, as well as pet birds. The estimated indirect cost of the outbreak to the poultry industry was \$200 million (NSW Agriculture 2002). Other outbreaks at Blacktown, NSW (1998), and Tamworth, NSW (1999–2000), cost \$2.8 million and \$0.45 million respectively. Limited outbreaks in Meredith, Victoria, and Horsley Park, NSW in 2002, cost industry and government \$1.9 million and \$0.4 million respectively in shared costs under the Emergency Animal Disease Response Agreement (M. Willoughby, Animal Health Australia, pers. comm. January 2006). Overseas outbreaks of ND have also been costly to

eradicate. For example, control of an outbreak of ND in Southern California in 2003 cost over \$US100 million and resulted in the slaughter of more than 3 million birds (Shane 2003).

6.2.2 Risk assessment

Release assessment

ND is endemic in many countries. In most countries with commercial poultry industries, vaccination is used to control the disease. According to OIE, NDV is present in Taiwan and vaccination is practised (OIE 2006b), although no outbreaks have been reported for the past eighteen months (OIE 2006a).

Vaccinated chickens are protected from clinical disease, but can become infected and shed virulent virus. Therefore, in a country where regular vaccination is practised, virulent viruses may be circulating without causing clinical disease. It is accepted that the level of infection would be lower than in non-vaccinated, infected flocks. Passive surveillance systems would not be expected to identify these sub-clinical infections in vaccinated poultry.

Ducks can become infected with NDV but do not usually show clinical signs, even when infected with strains that are pathogenic to chickens, although outbreaks of severe disease have been recorded (Alexander 2003). Therefore, duck flocks could be infected with virulent NDV contracted from vaccinated poultry. Further, duck flocks would not be expected to be vaccinated against ND, even in a country in which vaccination of commercial chickens is practised.

NDV is readily transmissible, especially among birds kept in large groups. Therefore, it can be assumed that, if NDV is present within an unvaccinated flock of ducks, a large proportion of the flock will be infected.

NDV has been demonstrated in, and on, eggs and in the reproductive tract of hens (Biswal and Morrill 1954). As ducks do not show signs of clinical ND, it is considered that they would be unlikely to suffer a drop in egg production as a result of infection with the virus. Therefore, NDV could be present in, or on, a proportion of eggs laid by ducks in an infected flock.

Overall, considering the effect of vaccination on limiting, but not eliminating infection with ND, and the possibility of sub-clinical infection in vaccinated chickens and in unvaccinated ducks, the likelihood of NDV being present in eggs, and the lack of proven efficacy of pH levels attained within eggs during the alkalising process as an inactivation method for NDV, the overall likelihood that NDV would enter Australia through the importation of alkalised duck eggs from Taiwan was assessed as **moderate**.

Exposure assessment

As explained in the Chapter on Methods, low biosecurity backyard poultry and wild birds are the only exposure groups at significant risk.

Preserved eggs are a delicacy item and it is assumed that minimal waste would result from the purchase and consumption of these products. It is likely that only a small proportion of households that keeps backyard poultry and feed them kitchen scraps would also purchase alkalised duck eggs from Taiwan. The fraction of the egg that does become available will consist mostly of shell, and would have been directly exposed to the alkalising agent at pH 13.

The smaller quantity of waste that consists of egg fragments however, may not have reached a pH of greater than 9.5, and cannot be confidently expected to be completely free of viable virus.

Waste generated from restaurant use will generally be disposed of in municipal refuse dumps, while waste from domestic consumption could be fed to backyard poultry or added to household composts. It is considered that only a small percentage of imported alkalised eggs will eventually become available to susceptible exposure groups.

NDV, within egg waste (excluding shell), is likely to survive in the environment under ambient temperatures for several days. The time between feeding of scraps and consumption by low biosecurity backyard poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. Overall, the likelihood that a backyard bird or a wild bird would be exposed to an amount of scrap from an infected alkalised duck egg, containing a sufficient quantity of virus to infect a susceptible bird, was assessed as **low**.

Consequence assessment

Likelihood of establishment and spread

The likelihood of establishment and spread of ND in Australia has been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the likelihood that NDV would spread beyond an initially exposed group of backyard poultry or wild birds as **very low** and **low** respectively.

Impacts of establishment and spread

The impacts of establishment and spread of NDV in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the impact of NDV spreading beyond the primary exposure group, including effects of eradication by stamping out, as **moderate**.

Risk estimation

After combining the likelihoods of entry (LR) and exposure (PLEbp and PLEwb), as detailed on page 27, the partial annual likelihoods of entry and exposure for backyard poultry and wild birds (PALEEbp and PALEEwb) were both estimated as **low**.

Combining these with the likelihoods of establishment and spread (PLESbp and PLESwb) as discussed on page 27, resulted in estimates of **very low** for the partial annual likelihoods of entry, exposure, establishment and spread (PALEEESbp and PALEEESwb).

Combining this with the impact of the establishment of the disease, as detailed on page 27, provided partial annual risk estimates of **very low** for both backyard poultry and wild birds.

Finally, combining the partial annual risks for backyard poultry and wild birds according to the rules detailed on page 28 yielded a final risk for ND of **low**.

As this unrestricted risk estimate exceeds Australia's ALOP¹, risk management was considered necessary for this disease agent.

6.2.3 Reference List

1. Alexander, D. J. 1997. *Heat inactivation of Newcastle disease virus [NDV] in homogenised chicken meat*, Contract No. 0513 between the Commonwealth of Australia and the Veterinary Laboratories Agency.
2. Alexander, D. J. 2000. Newcastle disease and other avian paramyxoviruses. *Revue Scientifique Et Technique Office International Des Epizooties* 19, no. 2: 443-62.
3. ———. 2003. Newcastle disease, other avian Paramyxoviruses, and Pneumovirus infections. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 63-99. Ames, Iowa, USA: Iowa State Press.
4. Alexander, D. J., J. S. Mackenzie, and P. H. Russell. 1986. Two types of Newcastle disease virus isolated from feral birds in Western Australia detected by monoclonal antibodies. *Australian Veterinary Journal* 63, no. 11: 365-67.
5. Alexander, D. J., and R. J. Manvell. 2004. Heat inactivation of Newcastle disease virus (strain Herts 33/56) in artificially infected chicken meat homogenate. *Avian Pathology* 33, no. 2: 222-25.
6. Animal Health Australia. 2002. "The Emergency Animal Disease Response Agreement." Web page, [accessed June 2003]. Available at <http://www.aahc.com.au/eadp/deed.pdf>.
7. Animal Health Australia. 2004. "AUSVETPLAN Disease Strategy: Newcastle disease (Version 3.0, 2004)." Web page, [accessed September 2005]. Available at http://www.animalhealthaustralia.com.au/shadomx/apps/fms/fmsdownload.cfm?file_uid=2B277B94-D677-87C4-E607-D2DF8E1A3440&siteName=aahc.
8. Animal Health Australia. 2005. "Newcastle Disease Management Plan." Web page, [accessed January 2006]. Available at <http://www.animalhealthaustralia.com.au/programs/special/ndv.cfm>.
9. Asplin, F. D. 1949. Observations on the viability of Newcastle disease virus. *The Veterinary Record* 61: 159-60.
10. Australian Animal Health Laboratory, CSIRO Livestock Industries. 2002. An investigation of shedding of Newcastle disease virus (NDV) on or in the eggs of hens vaccinated against NDV and then challenged with NDV. Unpublished report. P. Selleck, and S. Lowther.
11. Beard, C. W. , and R. P. Hanson. 1984. Newcastle disease. In *Diseases of Poultry*. 8th ed., Editors M. S. Hofstad, H. J. Barnes, B. W. Calnek, W. M. Reid, and H. W. Yoder, 452-70. Ames, Iowa: Iowa State University Press.
12. Biswal, G., and C. C. Morrill. 1954. The pathology of the reproductive tract of laying pullets affected with Newcastle disease. *Poultry Science* 33: 880-897.
13. Bivins, J. A., B. Rhodes-Miller, and F. R. Beaudette. 1950. Search for virus in eggs laid during recovery postinoculation with Newcastle disease virus. *American Journal of Veterinary Research* 11: 426-27.
14. Capua, I., M. Scacchia, T. Toscani, and V. Caporale. 1993. Unexpected isolation of virulent Newcastle disease from commercial embryonated fowls eggs. *Journal of Veterinary Medicine B* 40: 609-12 .

15. Chang, P. W. 1981. Newcastle disease. *CRC Handbook Series in Zoonoses. Section B: Viral Zoonoses.* editor G. W. Beran, 261-74. Vol. II. Boca Raton, Florida: CRC Press.
16. Chen Juipin, and Wang Chingho. 2002. Clinical Epidemiologic and Experimental Evidence for the Transmission of Newcastle Disease Virus Through Eggs. *Avian Diseases* 46, no. 2.
17. Collins, M. S., R. E. Gough, and D. J. Alexander. 1993. Antigenic differentiation of avian pneumovirus isolated using polyclonal antisera and mouse monoclonal antibodies. *Avian Pathology* 22: 469-79.
18. Erickson, G. A., C. J. Mare, G. A. Gustafson, L. D. Miller, S. J. Protor, and E. A. Carbrey. 1977. Interactions between viscerotropic velogenic Newcastle disease virus and pet birds of six species. I. Clinical and serological responses, and viral excretion. *Avian Diseases* 21, no. 4: 642-54.
19. Foster, N. M., and C. H. Thompson. 1957. The comparative thermostability of four strains of Newcastle disease virus of widely varying virulence. *Veterinary Medicine* 52: 119-21.
20. French, E. L., T. D. St George, and J. J. Percy. 1967. Infection of chicks with recently isolated Newcastle disease viruses of low virulence. *Australian Veterinary Journal* 43: 404-9.
21. Gilchrist, P. 1993. Newcastle disease as a threat to native birds. *Proceedings of the Ninth Australian Poultry and Feed Convention*, 120-125.
22. ———. 2005. Involvement of free-flying wild birds in the spread of the viruses of avian influenza, Newcastle Disease and infectious bursal disease from poultry products to commercial poultry. *World's Poultry Science Journal* 61: 198-214.
23. Gough, R. E. 1973. Thermostability of Newcastle disease virus in liquid whole egg. *The Veterinary Record* 93: 632-33.
24. Gough, R. E. , D. J. Alexander, M. S. Collins, S. A. Lister , and W. J. Cox. 1988. Routine virus isolation or detection in the diagnosis of diseases in birds. *Avian Pathology* 17, no. 4: 893-907.
25. Gould, A. R., J. A. Kattenbelt, P. Selleck, E. Hansson, A. Della-Porta, and H. A. Westbury. 2001. Virulent Newcastle disease in Australia: Molecular epidemiological analysis of viruses isolated prior to and during the outbreaks of 1998-2000. *Virus Research* 77: 51-60.
26. Hofstad, M. S. 1949. A study on the epizootiology of Newcastle disease (pneumoencephalitis). *Poultry Science* 28: 530-533.
27. Hugh-Jones, M., W. H. Allan, F. A. Dark, and G. J. Harper. 1973. The evidence for the airborne spread of Newcastle disease. *Journal of Hygiene (Cambridge)* 71: 325-39.
28. Kaleta, K. F., and C. Baldauf. 1988. Newcastle disease in free living and pet birds. *Developments in Veterinary Virology* 8: 197-246.
29. King, D. J. 1991. Evaluation of different methods of inactivation of Newcastle disease virus and avian influenza virus in egg fluids and serum. *Avian Diseases* 35: 505-14.
30. Kouwenhoven, B. 1993. Newcastle disease. In *Virus Infections of Birds*. Editors J. B. McFerran, and M. S. McNulty, 341-61. Amsterdam: Elsevier Science Publishers B.V.
31. Lancaster, J. E. 1963a. Newcastle disease - modes of spread. Part I. *The Veterinary Bulletin* 33: 221-28.
32. Lancaster, J. E. 1963b. Newcastle disease - modes of spread. Part II. *The Veterinary Bulletin* 33:

279-85.

33. Lancaster, J. E., and D. J. Alexander. 1975. *Newcastle disease virus and spread. A review of some of the literature*. Canadian Department of Agriculture.
Notes: 79 pages, 2 maps
34. Monlux, W. S. 1972. Signs and lesions of viscerotropic velogenic Newcastle disease in chickens. *Proceedings of the 76th Annual Meeting of the US Animal Health Association*, 288-90.
35. NSW Agriculture. 2002. "Performance audit report." *Managing animal disease emergencies*, The Audit Office of New South Wales, Australia.
36. OIE. "Disease information published during the past eighteen months. Index by disease." Web page, [accessed 15 November 2006a]. Available at http://www.oie.int/eng/info/hebdo/a_dsum.htm.
37. OIE. "Handistatus II." Web page, [accessed 15 November 2006b].
38. Peroulis, I., and K. O'Riley. 2004. Detection of avian paramyxoviruses and influenza viruses amongst wild bird populations in Victoria. *Australian Veterinary Journal* 82, no. 1,2: 79-82.
39. Peroulis-Kouritis, I., K. O'Riley, D. Grix, R. J. Condron, and C. Ainsworth. 2002. Molecular characteristics of Victorian Newcastle disease virus isolates from 1976 to 1999. *Australian Veterinary Journal* 80, no. 7: 422-24.
40. Shane, S. M. 2003. Disease continues to impact the world's poultry industries. *World Poultry* 19, no. 7: 22-23.
41. Sinha, S. K. , M. S. Hanson, and C. A. Brandly. 1954. Aerosol transmission of Newcastle disease in chickens. *American Journal of Veterinary Research* 15: 287-92.
42. Swayne David E., and Beck Joan R. 2004. Heat Inactivation of Avian Influenza and Newcastle Disease Viruses in Egg Products. *Avian Pathology* 33, no. 5: 512-18.
43. Williams, J. E., and L. H. Dillard. 1968. Penetration patterns of *Mycoplasma gallisepticum* and Newcastle disease virus through the outer structures of chicken eggs. *Avian Diseases* 12: 650-657.
44. Wobeser GA. 1997. Diseases of wild waterfowl, 2nd Ed.
45. World Organisation for Animal Health (OIE). 2002a. "Handistatus II: Annual health information." Web page, [accessed 2004a]. Available at <http://www.oie.int/hs2/report.asp>.
46. World Organisation for Animal Health (OIE). 2002b. "Technical Disease Cards - A160 - Newcastle Disease." Web page, [accessed 1 March 2004b]. Available at http://www.oie.int/eng/maladies/fiches/a_A160.htm.
47. Zagar, S. L. and B. S. Pomeroy. 1950. The effects of commercial living Newcastle disease virus vaccines. *American Journal of Veterinary Research* 11: 272-77.

6.3 *Salmonella Gallinarum/Salmonella Pullorum*

6.3.1 Technical information

Background

Pullorum disease and fowl typhoid are septicaemic bacterial diseases of chickens, turkeys and pheasants. Pullorum disease is caused by *Salmonella Pullorum*, and fowl typhoid is caused by *Salmonella Gallinarum* (Shivaprasad 2000). Although listed as separate species, these diseases are commonly discussed together in standard texts, because of their similarity in terms of epidemiology and management (Shivaprasad 2000; Davies 2004). These two *Salmonella* species are distinguished from the remainder of the salmonellae, in that they are host-adapted and highly pathogenic for avian species, but are considered to pose little zoonotic risk (Davies 2004).

Both pullorum disease and fowl typhoid are OIE-listed diseases, and it is generally recommended that export of poultry and poultry products be made only from flocks known to be serologically negative for these diseases (Shivaprasad 2000). The OIE records that these two diseases have been eradicated from Australian commercial flocks (World Organisation for Animal Health (OIE) 2005), although there has been serological or other evidence of the diseases being present in Australia in the past. The Australian Salmonella Reference Laboratory has not recorded isolation of *S. Pullorum* from any Australian source since 1992 (Davos 2003). Fowl typhoid was last reported in Australia in 1952 (Animal Health Australia 2001).

Epidemiology

Chickens are the natural hosts for both *S. Pullorum* and *S. Gallinarum*. Naturally-occurring outbreaks have occurred in turkeys, pheasants, guinea fowl, quail, sparrows and parrots (Shivaprasad 2003). Pullorum disease has been described in canaries and bullfinches, and fowl typhoid in ring doves, ostriches and peafowl (Shivaprasad 2003).

Natural infections with *S. Pullorum* and *S. Gallinarum* have been reported in ducks (Buxton 1957; Chute and Gershman 1963; Snoeyenbos GH 1991; Barrow et al. 1999); however, ducks appear to be quite resistant to the disease agents (Chute and Gershman 1963; Barrow et al. 1999; Buchholz and Fairbrother 1992).

An experimental study has reported that when 10 days-old Mallard ducklings were inoculated with *S. Pullorum* either orally (10^5 to 10^{10} colony forming units (cfu)/ml) or intravenously (10^3 to 10^8 cfu/ml), neither mortality nor morbidity was observed. In addition, viable *S. Pullorum* was isolated from Mallard livers at two weeks post-inoculation but not at three weeks post-inoculation, and all histopathological examinations of lungs, heart, liver, kidneys, pancreas and spleen were normal. In contrast, in the same experiment the mortality rate of 10 days-old bobwhite quails was higher than 60% when they were inoculated orally or intravenously with 10^3 to 10^8 cfu/ml of *S. Pullorum*, and viable organisms were isolated from all tissues of sick bobwhite quails, which either died during the experimental period or were euthanized at the end of the experiment. There was also slight to moderate diffuse or multifocal necrotising inflammation in tissues examined (Buchholz and Fairbrother 1992).

The researchers concluded that Mallards experience a short, subclinical infection that is resolved without lasting tissue damage.

Ducks were also shown to be very resistant to experimental infection with *S. Gallinarum* (Barrow et al. 1999). A group of day-old ducks inoculated orally with 3×10^8 cfu in 0.3 ml of *S. Gallinarum* exhibited neither mortality nor morbidity during the three week experimental period. Post-mortem examination of livers and spleens three weeks after inoculation revealed that the organs appeared healthy. In addition, no growth of *S. Gallinarum* was observed by culturing liver swabs on MacConkey agar, indicating an inability of the bacteria to multiply in the reticulo-endothelial system (Barrow et al. 1999).

Transmission via eggs

S. Pullorum is vertically transmitted, with organisms localising in the ovule or contaminating the ovum following ovulation. *S. Pullorum* was detected in eggs laid by commercial layer hens that had been experimentally infected at 4-5 days of age (Berchieri et al. 2001) and in another study at one week of age (Wigley et al. 2001; Pinheiro, de Oliveira, and Berchieri 2001). The organism has also been isolated from spray-dried whole egg (McCullough and Eisele 1951).

There is conflicting evidence regarding egg transmission of *S. Gallinarum*. Although vertical transmission of *S. Gallinarum* is reported (Shivaprasad 1997; Wray and Davies 2001), recent attempts to isolate the organism from the eggs of experimentally-infected hens were unsuccessful (Berchieri et al. 2001). Furthermore, in an *in vitro* experiment, *S. Gallinarum* was isolated from eggs immediately after inoculation with the organism, but could not be isolated from the eggs 24 and 48 hours later (Berchieri et al. 2001). Therefore, the role of vertical transmission in the spread of *S. Gallinarum* is unclear.

Transmission via the egg can also occur through shell penetration but has been reported to be of minor importance (Williams, Dillard, and Hall 1968). A carrier state exists, with infected birds capable of infecting the next generation through vertical transmission, and other birds through faecal shedding of organisms (Shivaprasad 2003).

6.3.2 Risk assessment

Release assessment

S. Pullorum and *S. Gallinarum* have been reported to occur in ducks, however, ducks appear to be quite resistant to the organisms. On balance, Biosecurity Australia considers that *S. Pullorum* and *S. Gallinarum* may be present in ducks, but at a much lower prevalence than may be expected in chicken flocks.

The major route of transmission of *S. Pullorum* in chickens is vertically via transovarial transmission, with the organism being present in up to 33% of eggs laid by an infected hen. It can be assumed that *Salmonellae* can be transmitted in eggs laid by infected ducks in a similar way.

It has been reported that *Salmonellae* can grow in a range of pH, from pH 4.0 – 9.0. This would suggest that they will be inactivated by the process of producing alkalised eggs, during which the internal contents of the egg reaches a pH of 9.5 or higher. However, another *Salmonella*, *S. Enteritidis*, has been shown to survive at high levels (10^5 cfu/g) in alkalised

eggs for 2-3 months after processing (Fu and Su 1997). Therefore the effect of the alkalising cannot be assumed to be completely effective in reducing the infection to safe levels.

Both *S. Gallinarum* and *S. Pullorum* have been reported in Taiwan in the past, and it is not known whether they are currently notifiable and subject to an official control program. In countries where the disease is endemic, the prevalence of infection is difficult to estimate.

Based on this information, the overall annual likelihood that *Salmonella Pullorum* and *Gallinarum* would enter Australia through the importation of alkalised eggs was assessed as **low**.

Exposure assessment

As explained in the Chapter on Methods, low biosecurity backyard poultry and wild birds are the only exposure groups at significant risk.

Preserved eggs are a delicacy item and it is assumed that minimal waste would result from the purchase and consumption of these products. It is likely that only a small proportion of those households that keep backyard poultry and feed them kitchen scraps would also purchase preserved duck eggs from Taiwan. The fraction of the egg that does become available will consist mostly of shell, and would have been directly exposed to the alkalising agent at pH 13. The smaller quantity of waste that consists of egg fragments however, may not have reached a pH of greater than 9.5. Although unable to conclude that there will be a complete inactivation of any contaminating bacteria, it is likely that some degree of inactivation of surface bacteria will occur. Waste generated from restaurant use will generally be disposed of in municipal refuse dumps, while waste from domestic consumption could be fed to backyard poultry or added to household composts. It is considered that only a small percentage of imported preserved eggs will eventually become available to susceptible exposure groups.

The load of bacteria present in eggs laid by naturally infected ducks has not been recorded. Therefore, it is difficult to determine if a sufficient dose of bacteria would be present in or on an infected or contaminated duck egg. *Salmonellae*, within egg waste, are likely to survive and grow in the environment under ambient temperatures for several days, giving ample time for wild birds to locate and scavenge accessible material. The time between feeding of scraps and consumption by low biosecurity backyard poultry is likely to be very short, so environmental degradation of the disease agent will be minimal.

Overall, it was assessed that the annual likelihoods that backyard poultry (PLEbp) and wild birds (PLEwb) would be exposed to an infectious dose of *Salmonella Pullorum* or *S. Gallinarum* are **low**.

Consequence assessment

Establishment and spread was considered in the context of an outbreak scenario occurring in low biosecurity backyard poultry and wild birds, in which the pathogenic agent establishes in the exposed populations and spreads to other populations of susceptible species in Australia.

Likelihood of establishment and spread

The likelihood of establishment and spread of *S. Pullorum* and *S. Gallinarum* in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the likelihood

that these organisms would spread from backyard poultry or wild birds to other susceptible species in Australia as **very low** and **extremely low** respectively.

Impacts of establishment and spread

The impacts of establishment and spread of *S. Pullorum* and *S. Gallinarum* in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia considers that the impact of *S. Pullorum* and *S. Gallinarum* spreading to commercial poultry in Australia will be due mainly to costs associated with control and eradication of the disease. Biosecurity Australia has assessed the impacts as **low**.

Risk estimation

After combining the likelihoods of entry (LR) and exposure (PLEbp and PLEwb), as detailed on page 27, the partial annual likelihoods of entry and exposure for both backyard poultry and wild birds (PALEEbp and PALEEwb respectively) were estimated as **very low**.

Combining these with the likelihoods of establishment and spread (PLESbp and PLESwb) as discussed on page 27, resulted in an estimate of **extremely low** for the partial annual likelihoods of entry, exposure, establishment and spread (PALEEESbp and PALEEESwb).

Combining this with the impact of the establishment of the disease, as detailed on page 27, provided partial annual risk estimates of **negligible** for both wild birds and backyard poultry.

Finally, combining the partial annual risks for backyard poultry and wild birds according to the rules detailed on page 28 yielded a final risk for *S. Pullorum* and *S. Gallinarum* of **negligible**.

As this unrestricted risk estimate achieves Australia's ALOP, risk management was not considered necessary for these disease agents.

6.3.3 Reference List

1. Animal Health Australia. 2001. *Animal Health in Australia 2000*, Australian Animal Health Council, Canberra.
2. Barrow, P. A., M. A. Lovell, C. K. Murphy, and K. Page. 1999. Salmonella infection in a commercial line of ducks; experimental studies on virulence, intestinal colonization and immune protection. *Epidemiol Infect* 123, no. 1: 121-32.
3. Berchieri, A., C. K. Murphy, K. Marston, and P. A. Barrow. 2001. Observations on the persistence and vertical transmission of *Salmonella enterica* serovars Pullorum and Gallinarum in chickens: effect of bacterial and host genetic background. *Avian Pathology* 30: 221-31.
4. Biosecurity Australia. 2006. *Draft Generic Import Risk Analysis Report for Chicken Meat*, Australian Government Department of Agriculture, Fisheries and Forestry, Canberra, Australia.
5. Buchholz, P. S., and A. Fairbrother. 1992. Pathogenicity of *Salmonella pullorum* in northern bobwhite quail and mallard ducks. 36, no. 2: 304-12.
6. Buxton, A. 1957. *Salmonellosis in Animals*. 52. Farnham Royal, Bucks, England: Commonwealth Agricultural Bureaux.
7. Chute, H. L., and M. Gershman. 1963. Case Report-Salmonella Pullorum in a Muscovy (Cairina Moschata) Duck. *Avian Diseases* 7, no. 2: 168-&.
8. Davies, R. 2004. Fowl typhoid and Pullorum disease. In *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. 5th ed., OIE, <http://www.oie.int/eng/normes/mmanual/A_00106.htm>. Office International des Epizooties.
9. Davos, Dianne, Editor. 2003. *Australian Salmonella Reference Centre 2003 Annual Report*. Adelaide, South Australia: Institute of Medical and Veterinary Science.
10. Fu, Y.-M., and T. Su. 1997. Survival of *Salmonella enteritidis* during the processing and storage of processed duck egg. *Journal of Food and Drug Analysis*. 5, no. 2: 171-78.
11. McCullough, N. B., and C. W. Eisele. 1951. Experimental human salmonellosis IV. Pathogenicity of strains of *Salmonella Pullorum* obtained from spray-dried whole egg. *Journal of Infectious Diseases* 89: 259-65.
12. Pinheiro, L. A. S., G. H. de Oliveira, and A. Berchieri. 2001. Experimental *Salmonella enterica* serovar Pullorum infection in two commercial varieties of laying hens. *Avian Pathology* 30: 129-33.
13. Shivaprasad, H. L. 1997. Pullorum disease and fowl typhoid. In *Diseases of Poultry*. 10th ed., Editors B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDougald, and Y. M. Saif, 82-96. London, UK: Mosby-Wolfe.
14. ———. 2000. Fowl typhoid and pullorum disease. *Revue Scientifique Et Technique Office International Des Epizooties* 19, no. 2: 405-24.
15. ———. 2003. Pullorum disease and fowl typhoid. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 568-82. Iowa, USA: Iowa State Press.

16. Snoeyenbos GH. 1991. Pullorum disease. In *Diseases of Poultry*. 9 ed., Editors Calnek B W, Barnes HJ, Beard CW, Reid WM, and Yoder HW Jr, 87-99. Ames Iowa USA: Iowa State University Press.
17. Wigley, P., A. Berchieri, K. L. Page, A. L. Smith, and P. A. Barrow. 2001. *Salmonella enterica* serovar Pullorum persists in splenic macrophages and in the reproductive tract during persistent, disease-free carriage in chickens. *Infection and Immunity* 69, no. 12: 7873-79.
18. Williams, J. E., L. H. Dillard, and G. O. Hall. 1968. The penetration patterns of *Salmonella* Typhimurium through the outer structures of chicken eggs. *Avian Diseases* 12: 445-66.
19. World Organisation for Animal Health (OIE). 2005. "Handistatus II: Annual health information." Web page, [accessed 2006]. Available at <http://www.oie.int/hs2/report.asp>.
20. Wray, C., and R. H. Davies. 2001. *Enterobacteriaceae*. In *Poultry Diseases*. 5th ed., Editors F. Jordan, M. Pattison, D. Alexander, and T. Faragher, 95-130. London, UK: WB Saunders.

6.4 *Salmonella* Enteritidis/Multi-drug resistant *Salmonella* Typhimurium

6.4.1 Technical information

Background

S. Enteritidis and *S. Typhimurium* are typically non-host-specific bacterial pathogens, principally of concern as a major cause of food-borne salmonellosis in humans. In poultry, strains of these two *Salmonella* serovars cause systemic infection, leading to contamination of meat and eggs. *S. Enteritidis* and *S. Typhimurium* seldom cause clinical disease, except in susceptible young birds (Gast 2003). The serovars are distributed virtually worldwide in a range of species (Barrow 2000) including ducks, but significant subtypes and particular strains of concern are not endemic in Australia.

S. Enteritidis phage type 4 (PT 4), phage type 8 (PT 8) and phage type 13a (PT 13a) are generally recognised as the most important of the 50 or so phage types of *S. Enteritidis*. In some countries, invasive strains of PT4 have replaced all other phage types in the poultry population (Wilks, Parkinson, and Young 2000). In 1994, *S. Enteritidis* PT 4 was isolated from samples taken from a commercial layer flock in Australia. However, the isolates were thought to be the result of laboratory contamination since isolation could not be repeated on re-sampling of the same shed (Davos 2002). A *S. Enteritidis* outbreak involving a commercial meat-chicken company was investigated in Queensland in 2005. Control measures, including culling, stringent disinfection procedures and on-going monitoring, were put in place by Queensland State Government authorities (K Bell, Safe Food Queensland, Australia, pers. comm. May 2006).

More than 270 phage types of *S. Typhimurium* are recognised, of which definitive phage type 104 (DT104) is one of the most important. Multi-drug resistant strains of *S. Typhimurium* DT 104 have not been isolated from poultry flocks in Australia (Joint Expert Technical Advisory Committee on Antibiotic Resistance 1999).

S. Enteritidis PT 4 and *S. Typhimurium* DT 104 have been isolated from cases of human Salmonellosis in Australia, but are usually associated with overseas travel (Davos 2002; O'Grady et al. 2001). However, the source of a recent outbreak of multi-drug resistant *S. Typhimurium* DT 104 was imported contaminated food (O'Grady et al. 2001). Introduction of these pathogens would have a significant impact on the Australian poultry industry through their effect on public health, animal health and trade (Crerar, Nicholls, and Barton 1999).

Salmonellosis, due to *S. Enteritidis* or *S. Typhimurium* infection, is no longer a disease notifiable to the OIE.

Transmission via eggs

Intact shell eggs have been implicated as the major vehicle of transmission of *S. Enteritidis* (Cox 1995). Although experimental studies have shown that both *S. Enteritidis* and *S. Typhimurium* are able to colonise the tissues of the reproductive tract and forming eggs at equivalent rates (Keller et al. 1997), transovarian contamination of commercially produced eggs with *S. Typhimurium* is rare (Keller et al. 1997).

The numbers of *Salmonella* in the contents of freshly laid eggs are usually low (<10 cfu/mL) but may be as high as 100 cfu/mL (Humphrey et al. 1989; Humphrey et al. 1991). The principle sites of contamination within eggs are the albumen (80% positive eggs) or outside of the yolk membrane (13%) (Gast and Beard 1990); (Humphrey et al. 1991).

Salmonella spp. can die rapidly on egg shells but their survival is enhanced by high relative humidity and low temperatures during storage (Humphrey 1994). Temperature and location of the organism within the egg (whether in the albumen or yolk) have a significant effect on growth and survival of the organisms. At temperatures less than 10 °C growth is slow, or does not occur at all, but above 12 °C growth is rapid (Cox 1995). Yolk is nutrient-rich and supports growth of *Salmonella* spp. whereas the albumen contains lysozymes, proteins and polypeptides that inhibit bacterial growth. A study on the survival of *S. Enteritidis* during the processing and storage of processed duck eggs found that *Salmonella* survived in most eggs even after 2 months of storage, although the pH achieved during the alkalising process did lead to some reduction in titre when compared to non-alkalised eggs (Fu and Su 1997). Another study on the development of thermal inactivation models for *S. Enteritidis* with temperature, pH and NaCl as controlling factors found that in general the heat resistance of bacteria increased with decreasing water activity, with pH also effecting thermal inactivation with low and high pH values generally decreasing heat resistance (Blackburn et al. 1997). *S. Typhimurium* has been isolated from both duck egg shells and egg contents (Saitanu K and Jerngklinchan J 1994)].

6.4.2 Risk assessment

Release assessment

There are no data available on the prevalence of *S. Enteritidis* and multi-drug resistant *S. Typhimurium* in duck flocks.

The prevalence of *S. Enteritidis* contaminated eggs from naturally infected commercial layer flocks has generally been low. One study examined 1,119 hen eggs from two small flocks to investigate the prevalence of *S. Enteritidis* in eggs, and reported that 11 (1%) were positive for *S. Enteritidis*. The positive eggs were all found to contain fewer than 10 organisms (Humphrey et al. 1989). In another study, 5,790 hen eggs from 15 flocks were examined for the presence of *S. Enteritidis* in the egg contents. The prevalence of *S. Enteritidis* positive eggs was found to be 0.6% as 32 eggs were contaminated with the organism (Humphrey et al. 1991).

The Food Safety and Inspection Service, US Department of Agriculture, in a simulation study has estimated that the prevalence of *S. Enteritidis* positive hen eggs in the US is between 6 positive eggs per 100,000 eggs and 14 positive eggs per 10,000 eggs (Food Safety and Inspection Service 1998).

Based on this information, and in light of the fact that the alkalising process cannot be relied upon to completely destroy all *Salmonellae*, the overall annual likelihood that *Salmonella* Enteritidis and multi-drug resistant *Salmonella* Typhimurium would enter Australia through the importation of alkalised eggs was assessed as **low**.

Exposure assessment

As explained in the Chapter on Methods, low biosecurity backyard poultry and wild birds are the only exposure groups at significant risk.

Preserved eggs are a delicacy item and it is assumed that minimal waste would result from the purchase and consumption of these products. It is likely that only a small proportion of those households that keep backyard poultry and feed them kitchen scraps would also purchase preserved duck eggs from Taiwan. The fraction of the egg that does become available will consist mostly of shell, and would have been directly exposed to the alkalising agent at pH 13. The smaller quantity of waste that consists of egg fragments however, may not have reached a pH of greater than 9.5. Although unable to conclude that there will be a complete inactivation of any contaminating bacteria, it is likely that some degree of inactivation of surface bacteria will occur. Waste generated from restaurant use will generally be disposed of in municipal refuse dumps, while waste from domestic consumption could be fed to backyard poultry or added to household composts. It is considered that only a small percentage of imported preserved eggs will eventually become available to susceptible exposure groups.

The load of bacteria present in eggs laid by naturally infected ducks has not been recorded. Therefore, it is difficult to determine if a sufficient dose of bacteria would be present in, or on, an infected or contaminated duck egg. *Salmonellae*, within egg waste, are likely to survive and grow in the environment under ambient temperatures for several days, giving ample time for wild birds to locate and scavenge accessible material. The time between feeding of scraps and consumption by low biosecurity backyard poultry is likely to be very short, so environmental degradation of the disease agent will be minimal.

Overall, it was assessed that the annual likelihoods that backyard poultry (PLEbp) and wild birds (PLEwb) would be exposed to an infectious dose of *Salmonella* Enteritidis or *S. Typhimurium* are **low**.

Consequence assessment

Likelihood of establishment and spread

The likelihood of establishment and spread of *S. Enteritidis* and *S. Typhimurium* in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the likelihood that these organisms would spread from backyard poultry or wild birds to other susceptible species in Australia as **low**.

Impacts of establishment and spread

The impacts of establishment and spread of *S. Enteritidis* or *S. Typhimurium* in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the impact of *S. Enteritidis* or *S. Typhimurium* spreading in commercial poultry in Australia as **moderate**, based on the costs of effective control and eradication of the infection.

Risk estimation

After combining the likelihoods of entry (LR) and exposure (PLEbp and PLEwb), as detailed on page 27, the partial annual likelihoods of entry and exposure for both backyard poultry and wild birds (PALEEbp and PALEEwb respectively) were estimated as **very low**.

Combining these with the likelihoods of establishment and spread (PLESbp and PLESwb) as discussed on page 27, resulted in an estimate of **very low** for the partial annual likelihoods of entry, exposure, establishment and spread (PALEEEsbp and PALEEEswb).

Combining this with the impact of the establishment of the disease, as detailed on page 27, provided partial annual risk estimates of **very low** for both backyard poultry and wild birds.

Finally, combining the partial annual risks for backyard poultry and wild birds according to the rules detailed on page 28 yielded a final risk for *S. Enteritidis* and *S. Typhimurium* of **low**.

As this unrestricted risk estimate exceeds Australia's ALOP, risk management was considered necessary for this organism.

6.4.3 Reference List

1. Barrow, P. A. 2000. The paratyphoid salmonellae. *Review Scientifique Et Technique Office International Des Epizooties* 19, no. 2: 351-75.
2. Biosecurity Australia. 2006. *Draft Generic Import Risk Analysis Report for Chicken Meat*, Australian Government Department of Agriculture, Fisheries and Forestry, Canberra, Australia.
3. Blackburn, C. W., L. M. Curtis, L. Humpheson, C. Billon, and P. J. McClure. 1997. Development of thermal inactivation models for *Salmonella enteritidis* and *Escherichia coli* O157:H7 with temperature, pH and NaCl as controlling factors. *Int J Food Microbiol* 38, no. 1: 31-44.
4. Cox, J. M. 1995. *Salmonella Enteritidis: the egg and I*. *Australian Veterinary Journal* 72, no. 3: 108-15.
5. Crerar, S. K., T. J. Nicholls, and M. D. Barton. 1999. Multi-resistant *Salmonella* Typhimurium DT104 - implications for animal industries and the veterinary profession. *Australian Veterinary Journal* 77: 170-171.
6. Davos, D. 24 January 2002. Letter to Ashley Hall.
7. Food Safety and Inspection Service. 1998. "*Salmonella* Enteritidis Risk Assessment. *Shell Eggs and Egg Products*. Production Module." Web page, [accessed 27 October 2006]. Available at <http://www.fsis.usda.gov/OPHS/risk/pdfrisk2.pdf>.
8. Fu, Y.-M., and T. Su. 1997. Survival of *Salmonella enteritidis* during the processing and storage of processed duck egg. *Journal of Food and Drug Analysis*. 5, no. 2: 171-78.
9. Gast, R. K. 2003. Paratyphoid infections. In *Diseases of Poultry*. 11th ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 583-613. Ames, Iowa, USA: Iowa State Press.
10. Gast, R. K., and C. W. Beard. 1990. Production of *Salmonella enteritidis*-contaminated eggs by experimentally infected hens. *Avian Diseases* 34: 438-46.
11. Humphrey, T. J. 1994. Contamination of egg shell and contents with *Salmonella enteritidis*: a review. *International Journal of Food Microbiology* 21: 31-40.
12. Humphrey, T. J., A. Baskerville, S. L. Mawer, B. Rowe, and S. Hopper. 1989. *Salmonella enteritidis* PT4 from the contents of intact eggs: a study involving naturally infected hens. *Epidemiology Infection* 103: 415-23.
13. Humphrey, T. J., A. Whitehead, A. H. L. Gawler, A. Henley, and B. Rowe. 1991. Numbers of *Salmonella enteritidis* in the contents of naturally contaminated hens' eggs. *Epidemiology and Infection* 106: 489-96.
14. Joint Expert Technical Advisory Committee on Antibiotic Resistance. 1999. *The use of antibiotics in food-producing animals: antibiotic-resistant bacteria in animals and humans.*, Commonwealth Department of Health and Aged Care and Commonwealth Department of Agriculture, Fisheries and Forestry-Australia.
15. Keller, L. H., D. M. Schifferli, C. E. Benson, S. Aslam, and R. J. Eckroade. 1997. Invasion of chicken reproductive tissues and forming eggs is not unique to *Salmonella enteritidis*. *Avian Diseases* 41: 535-39.

16. O'Grady, K., Powling, J., Tan, A., Valcanis, M., Lightfoot, D., Gregory, J., Lalor, K., Guy, R., Ingle, B., Andrews, R., Crerar, S., and Stafford, R. 2001. "*Salmonella* Typhimurium PT 104." Web page, [accessed 4 February 2003]. Available at <http://www.health.gov.au/pubhlth/cdi/ausbreak.htm#pt104a>.
17. Saitanu K, and Koowatananukul C Jerngklinchan J. 1994. Incidence of *Salmonella* in duck eggs in Thailand. *Southeast Asian J Trop Med Public Health* 25, no. 2: 328-31.
18. Wilks, C., G. Parkinson, and P. Young. 2000. *International review of Salmonella Enteritidis (SE) epidemiology and control policies*, RIRDC Publication No 00/145. Rural Industries Research and Development Corporation, Canberra, Australia.

6.5 Duck enteritis virus

6.5.1 Technical information

Background

Duck virus enteritis (DVE), also known as duck plague, is an acute, haemorrhagic disease of ducks, geese and swans, all members of the family Anatidae of the order Anseriformes. It is a significant disease of ducks, and produces economic losses in flocks of ducks similar to those caused by ND in flocks of chickens. Infection has not been reported in other avian species, mammals or humans, and has not been reported in Australia.

Agent taxonomy

The aetiological agent of DVE is duck enteritis virus (DEV), a herpesvirus with a viral genome of linear, double stranded DNA. DEV is an unassigned member of the family Herpesviridae (Fauquet et al. 2005).

Agent characteristics

The characteristics of DEV have been described (Hess and Dardiri 1968). The virus is sensitive to ether and chloroform. Exposure of the virus to chymotrypsin, trypsin and pancreatic lipase at 37 °C for 18 hours markedly reduced or destroyed virus infectivity. Duck enteritis virus was inactivated at 56 °C and 60 °C within 10 minutes or at 50 °C for two hours. At room temperature (22 °C), DEV was not detectable after 30 days. Drying (over calcium chloride in a desiccator) at 22 °C resulted in inactivation of DEV in 9 days. Duck enteritis virus is fairly stable to pH range of 5 to 10, but is inactivated at pH 11 or higher, and pH 3 or lower (Hess and Dardiri 1968).

Epidemiology

DEV has been reported in the US, Canada, Europe (Netherlands, France, Germany, Belgium, the UK, Hungary, Denmark and Austria) and Asia (India, Malaysia, Thailand, Vietnam and China).

An outbreak of disease causing up to 20% mortality occurred in Pekin ducklings in Victoria in 1984. Histopathological lesions were similar to those of DEV but all attempts at isolating or identifying the cause of the outbreak failed (Scott et al. 1984). In 1989, an outbreak of disease clinically and pathologically similar to DEV with a high mortality rate (97 %) was reported in a flock of geese in south-eastern Queensland. A herpesvirus was isolated from tissues of affected geese; however, the virus was different from DEV, as the virus was not neutralised by a DEV reference antiserum (Ketterer et al. 1990). The virus was further characterised using PCR and found to be antigenically and genomically distinct from the herpesvirus that causes duck virus enteritis (Gough and Hansen 2000).

Taiwan claims country freedom from DEV. A written report of May 2006 from the BAPHIQ has informed Biosecurity Australia that Taiwan conducted a two-year serological survey between 2000 and 2001. In the survey, a total of 3,200 serum samples from ducks and 2,003

serum samples from geese were collected. The result of the survey was that no antibody against DEV was detected in any of the samples. Since then, surveillance for DEV has depended on ruling out suspect cases. No cases have been detected. While this provides strong evidence that there is an extremely low incidence of DEV in ducks in Taiwan, further surveys would be necessary to provide confidence of freedom.

Natural susceptibility appears to be limited to members of the family Anatidae of the order Anseriformes (ducks, geese and swans) (Sandhu and Shawky 2003). There are no reports of natural infection in other avian species or mammals (Sandhu and Shawky 2003).

DEV is shed in the faeces and in oral-respiratory secretions (Friend 1999), and acutely infected birds with DEV have been shown to shed over $3.7 \log_{10}$ plaque forming units (PFU) of the virus per ml of faeces (Spieker 1979). The virus can be transmitted by direct contact between infected and susceptible birds, or indirectly by contact with a contaminated environment (Sandhu and Shawky 2003). Water appears to be the natural means of virus transmission since outbreaks in domestic ducks have been limited to birds having access to open bodies of water co-inhabited by free-flying waterfowls (Sandhu and Shawky 2003).

Experimentally, DEV has been transmitted by oral, intranasal, intramuscular and conjunctival route of inoculation. The intramuscular route of inoculation required the least amount of virus (titre of $0.4 \log_{10}$ PFU in Muscovy embryo fibroblast cell) to produce mortality. Conjunctival and intranasal exposure required titres of 2.2 and $3.5 \log_{10}$ PFU, respectively, and the oral route required the most amount of virus (titre of $3.7 \log_{10}$ PFU) to produce mortality (Spieker, Yuill, and Burgess 1996).

Although the oral route of inoculation required the most amount of DEV to produce mortality, acutely infected birds were reported to shed in excess of $3.7 \log_{10}$ PFU per ml of faeces (Spieker 1970), which is a sufficient quantity of the virus to produce mortality following oral exposure.

DEV establishes and maintains a latent infection in the trigeminal ganglia in recovered birds typical of herpesvirus infections (Shawky and Schat 2002). Cloacal and oral swabs obtained from experimentally induced carrier birds have shown that the virus is shed intermittently (Burgess, Ossa, and Yuill 1979; Burgess and Yuill 1983). DEV latency and reactivation have been blamed for subsequent outbreaks of disease in domestic and migrating waterfowl populations.

Disease transmission in eggs

The virus was isolated from an egg found in the cloaca of a duck that died of duck virus enteritis (Jansen 1961). The course of DVE is so acute that birds may die with a fully-formed egg in the cloaca.

Experimental vertical transmission has been reported in DEV carrier Muscovy, Pekin and Mallard ducks, with the effects of vertical transmission varying with the strain of DEV and the species of ducks (Burgess and Yuill 1981). The fertility and hatchability of eggs laid by the DEV carrier ducks were significantly decreased compared with those of the uninfected control birds. Hatchlings surviving longer than two weeks were apparently healthy but virus was detected in cloacal swabs. Some ducklings showed typical oral lesions but none developed neutralising antibody. Hatchlings persistently infected with DEV were shown to excrete the virus in much the same way as adult DEV carriers do (Burgess and Yuill 1981).

Quarantine significance

DEV is not an OIE listed disease but is notifiable in all Australian States and Territories. Duck virus enteritis is not included in Australia's Emergency Animal Disease Response Agreement, and is of no public health significance (Sandhu and Shawky 2003). Outbreaks may cause serious economic losses due to decreased egg production in breeder ducks, decreased weight gain and increased susceptibility to other infections (Shawky, Sandhu, and Shivaprasad 2000) and increased mortality and carcass condemnation in ducklings and goslings. Vaccination programs would also be required. Outbreaks in wild waterfowl could result in significant mortality events.

6.5.2 Risk assessment

Release assessment

Taiwan claims country freedom from DEV although surveys were conducted some time ago. Australia considers that further, more recent data would be necessary to provide confidence of complete freedom from this disease agent. However, it is accepted that the prevalence of disease is extremely low, since population densities in concentrated duck-producing areas result in rapid spread of DEV, and it would be expected to spread rapidly once introduced to a laying flock. This would be expected to result in detection of the outbreak, even by passive surveillance methods.

Experimentally induced DEV carrier ducks have been shown to shed the virus in eggs, leading to infection of ducklings. Acute infection leads to a significant drop in egg production, which decreases the likelihood of infected eggs being produced. However, recovered birds may be carriers. Therefore, DEV could be present in a proportion of eggs laid by ducks in an infected flock.

DEV is unlikely to be inactivated by the processes involved in producing alkalised eggs.

Based on this information, the overall likelihood that DEV would enter Australia through the importation of alkalised duck eggs from Taiwan was assessed as **extremely low**.

Exposure assessment

Preserved eggs are a delicacy item and it is assumed that minimal waste would result from the purchase and consumption of these products. It is likely that only a small proportion of households that keeps backyard ducks and feed them kitchen scraps would also purchase preserved duck eggs from Taiwan. The fraction of the egg that does become available will consist mostly of shell, and would have been directly exposed to the alkalising agent at pH 13. The smaller quantity of waste that consists of egg fragments however, may not have reached a pH of greater than 9.5, and cannot be confidently expected to be completely free of viable virus. DEV, protected within egg waste, is likely to survive in the environment under ambient temperatures for several days. The time between feeding of scraps and consumption by low biosecurity ducks is likely to be very short, so environmental degradation of DEV if present will be minimal. However, the majority of backyard birds in Australia are hens, and only ducks are susceptible to DEV.

Waste generated from restaurant use will generally be disposed of in municipal refuse dumps, while waste from domestic consumption could be fed to backyard ducks. It is considered unlikely that wild Anatidae would be present in refuse dumps. Therefore, it was considered relatively unlikely that restaurant wastes from imported Taiwanese preserved duck eggs would be exposed to susceptible birds in Australia.

Overall, it was assessed that the likelihood that susceptible backyard or wild ducks would be exposed to an infectious dose of DEV would be **extremely low**.

Consequence Assessment

Likelihood of establishment and spread.

In backyard poultry flocks, which generally have relatively few birds, there is less opportunity for the generation of high levels of environmental contamination, than might occur with an outbreak of infectious disease in a large commercial flock. As a result, there is less chance of disease spread to other flocks or other exposure groups. DEV could multiply extensively in backyard ducks, but transmission to other populations of such a relatively uncommonly kept species was considered unlikely. It was also considered unlikely that populations of domestic ducks, which were likely to be exposed to waste derived from imported Taiwanese preserved eggs, would be in close contact with wild ducks. Spread to wild ducks was therefore considered to be an unlikely event.

Biosecurity Australia considered that the most likely outcome of exposure of low biosecurity poultry would be sudden high mortality in a small backyard flock, which might go undiagnosed. However, if the disease was recognised, eradication would be relatively quick due to the small number of birds involved.

The major commercial duck producers in this country have enterprise level biosecurity plans. While these will not necessarily guarantee that there will be no contact between commercial ducks and domestic or wild birds, it should help to decrease the likelihood of this occurrence. In addition to this decreased likelihood of infection, the rapid response to any outbreak of DEV by the farm management, and the wide geographical distribution of the six commercial duck farms across the eastern States of Australia would significantly reduce the likelihood that an outbreak of DEV in one commercial flock of ducks would spread to the other flocks.

Therefore, Biosecurity Australia considered that the likelihood of DEV spreading beyond the initial exposure group in Australia is **very low**.

Impacts of establishment and spread.

DEV is a serious disease of ducks, and produces economic losses in flocks of ducks similar to those caused by ND in flocks of chickens. The impacts of establishment and spread of NDV in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the impact of NDV spreading and becoming endemic in Australia as moderate. However, there are significant differences between the two diseases.

Infection with DEV has not been reported in avian species other than ducks. NDV affects a wide variety of birds of all species. However, as DEV affects only ducks, and the duck industry is much less widespread than the poultry industry generally, it is reasonable to expect

that the impact of DEV on the commercial duck industry in Australia would be much lower than those of NDV on the poultry industry generally.

NDV has been shown to cause significant losses in wild bird populations. DEV also could cause deaths in wild populations, but would once again be limited only to ducks, so the potential effects on the environment are also less than would be expected from an outbreak of ND.

Given the differences between the two diseases in the species potentially at risk, it is considered that the impact of DEV in Australia would be **low**.

Risk estimation

After combining the likelihoods of entry (LR) and exposure (PLEbp and PLEwb), as detailed on page 27, the partial annual likelihoods of entry and exposure for backyard poultry and wild birds (PALEEbp and PALEEwb respectively) were estimated as **negligible**.

Combining these with the likelihoods of establishment and spread (PLESbp and PLESwb) as discussed on page 27, resulted in estimates of **negligible** for the partial annual likelihoods of entry, exposure, establishment and spread (PALEESbp and PALEESwb).

Combining this with the impact of the establishment of the disease, as detailed on page 27, provided partial annual risk estimates of **negligible** for both backyard poultry and wild birds.

Finally, combining the partial annual risks for backyard poultry and wild birds according to the rules detailed on page 28 yielded a final risk for DVE of **negligible**.

As this unrestricted risk estimate achieves Australia's ALOP, risk management was not considered necessary for this organism.

6.5.3 Reference List

1. Burgess, E. C., J. Ossa, and T. M. Yuill. 1979. Duck plague: a carrier state in waterfowl. *Avian Diseases* 24, no. 4: 940-949.
2. Burgess, E. C., and T. M. Yuill. 1981. Vertical transmission of duck plague virus (DPV) by apparently healthy DPV carrier waterfowl. *Avian Diseases* 25, no. 4: 795-800.
3. ———. 1982. Superinfection in ducks persistently infected with duck plague virus. *Avian Dis* 26, no. 1: 40-46.
4. ———. 1983. The influence of seven environmental and physiological factors on duck plague virus shedding by carrier mallards. *Journal of Wildlife Diseases* 19, no. 2: 77-81.
5. Butterfield, W. K., and A. H. Dardiri. 1969. Serologic and immunologic response of ducks to inactivated and attenuated duck plague virus. *Avian Diseases* 13: 876-87.
6. Fauquet, C. M., M. A. Mayo, J. Manniloff, U. Desselberger, and L. A. Ball, Editors. 2005. *Virus Taxonomy: classification and nomenclature of viruses*. Eighth Report of the International Committee on the Taxonomy of Viruses ed. San Diego, California, U.S.A.: Elsevier Academic Press.
7. Friend, M. 1999. Duck Plague. In *Field Manual of Wildlife Diseases: general field procedures and diseases of birds*. Editors M. Friend, and J. C. Franson, 141-51. Washington DC, USA: USGS.
8. Gough, R. E., E. D. Borland, I. F. Keymer, and J. C. Stuart. 1987. An outbreak of duck virus enteritis in commercial ducks and geese in East Anglia. *The Veterinary Record* 121, no. 4: 85.
9. Gough, R. E., and W. R. Hansen. 2000. Characterization of a herpesvirus isolated from domestic geese in Australia. *Avian Pathology* 29, no. 5: 417-22.
10. Hansen, W. R., S. W. Nashold, D. E. Docherty, S. E. Brown, and D. L. Knudson. 2000. Diagnosis of duck plague in waterfowl by polymerase chain reaction. *Avian Diseases* 44, no. 2: 266-74.
11. Hess, W. R., and A. H. Dardiri. 1968. Some properties of the virus of duck plague. *Archiv Für Die Gesamte Virusforschung* 24: 148-53.
12. Islam, M. R., and M. A. H. N. A. Khan. 1995. An immunocytochemical study on the sequential tissue distribution of duck plague virus. *Avian Pathology* 24: 189-94.
13. Jansen, J. 1961. Duck plague. *British Veterinary Journal* 117: 349-56.
14. ———. 1964. Duck plague (a concise survey). *The Indian Veterinary Journal* 41: 309-16.
15. Jansen, J., and R. Wemmenhove. 1965. Duck plague in domesticated geese (*Anser anser*). *Tijdschr Diergeneeskd* 90: 811-15.
16. Ketterer, P. J., B. J. Rodwell, H. A. Westbury, P. T. Hooper, A. R. Mackenzie, and J. G. Prior H. C. Dingle. 1990. Disease of geese caused by a new herpesvirus. *Australian Veterinary Journal* 67, no. 12: 446-48.
17. Keymer, I. F., and R. E. Gough. 1986. Duck virus enteritis (anatid herpesvirus infection) in mute swans (*Cygnus olor*). *Avian Pathology* 15, no. 1: 161-70.
18. Kulkarni, D. D., P. C. James, and S. Sulochana. 1998. Assessment of the immune response to

- duck plague vaccinations. *Research in Veterinary Science* 64: 199-204.
19. Leibovitz, L. 1968. Progress report: duck plague surveillance of American anseriformes. *Bulletin of the Wildlife Disease Association* 4, no. 87-91.
 20. ———. 1971. Gross and histopathologic changes of duck plague (duck virus enteritis). *Am J Vet Res* 32, no. 2: 275-90.
 21. Leibovitz, L., and J. Hwang. 1968. Duck plague on the American continent. *Avian Diseases* 12: 361-78.
 22. Lin, W., K. M. Lam, and W. E. Clark. 1984a. Active and passive immunization of ducks against duck viral enteritis. *Avian Diseases* 28, no. 4: 968-77.
 23. Lin, W., K. M. Lam, and W. E. Clark. 1984b. Isolation of an apathogenic immunogenic strain of duck enteritis virus from waterfowl in California. *Avian Diseases* 28, no. 3: 641-50.
 24. Montali, R. J., M. Bush, and G. A. Greenwell. 1976. An epornitic of duck viral enteritis in a zoological park. *Journal of the American Veterinary Medical Association* 169, no. 9: 954-58.
 25. Montgomery, R. D., G. Stein, M. N. Novilla, S. S. Hurley, and R. J. Fink. 1981. An outbreak of duck virus enteritis (duck plague) in a captive flock of mixed waterfowl. *Avian Diseases* 25, no. 1: 207-13.
 26. Proctor, S. J. 1975. Pathogenesis of digestive tract lesions in duck plague. *Vet Pathol* 12, no. 5-6: 349-61.
 27. Sandhu, T. S., and S. A. Shawky. 2003. Duck Virus Enteritis (Duck Plague). In *Diseases of Poultry*. 11 ed., Editors Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, and D. E. Swayne, 354-63. Iowa, USA: Iowa State Press.
 28. Scott, P. C., D. A. Barr, I. D. Connaughton, J. Gould, and A. Brightling. 1984. Mortality in Pekin ducklings associated with eosinophilic intranuclear inclusions in hepatocytes. *Australian Veterinary Journal* 61, no. 10: 328-29.
 29. Shawky, S. 2000. Target cells for duck enteritis virus in lymphoid organs. *Avian Pathology* 29, no. 6: 609-16.
 30. Shawky, S., T. Sandhu, and H. L. Shivaprasad. 2000. Pathogenicity of a low-virulence duck virus enteritis isolate with apparent immunosuppressive ability. *Avian Diseases* 44, no. 3: 590-599.
 31. Shawky, S., and K. A. Schat. 2002. Latency sites and reactivation of duck enteritis virus. *Avian Diseases* 46, no. 2: 308-13.
 32. Spieker, J. O. 1979. "Virulence assay and other studies of six North American strains of duck plague virus tested in wild and domestic waterfowl." University of Wisconsin.
 33. Spieker, J. O., T. M. Yuill, and E. C. Burgess. 1996. Virulence of six strains of duck plague virus in eight waterfowl species. *J Wildl Dis* 32, no. 3: 453-60.
 34. Toth, T. E. 1970. Two aspects of duck virus enteritis parental immunity and persistence/excretion of virulent virus. *Proceedings 74th Annual Meeting of the United States Animal Health Association and 13th Annual Conference of American Association of Veterinary Laboratory Diagnosticians*, 304-14.
 35. Toth, T. E. 1971. Active immunization of White Pekin ducks against duck virus enteritis (duck plague) with modified-live-virus vaccine: serologic and immunologic response of

breeder ducks. *American Journal of Veterinary Research* 32, no. 1: 75-81.

36. Van Dorssen, C. A., and H. Kunst. 1955. Susceptibility of ducks and various other water fowl to duck plague virus. *Tijdschr Diergeneesk* 80: 1286-95.
37. World Organisation for Animal Health. 2004. "Chapter 2.7.10 Duck virus enteritis in Manual of Standards for Diagnostic Tests and Vaccines for Terrestrial Animals." Web page. Available at http://www.oie.int/eng/normes/mmanual/A_00111.htm.
38. World Organisation for Animal Health. "Terrestrial Animal Health Code 2005 Chapter 2.7.10 Duck Virus Enteritis." Web page, [accessed 2006].

6.6 *Ornithobacterium rhinotracheale*

Biosecurity Australia has previously conducted an IRA on this disease agent, and the results have been published in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the impact of *O. rhinotracheale* becoming endemic in Australia as **very low**. As can be seen from the risk estimation matrix (Table 4) an organism which has impacts which are very low or lower cannot present a risk greater than very low. As this unrestricted risk estimate achieves Australia's ALOP¹, no risk management is considered necessary for this organism, beyond ensuring that the processing of alkalised eggs was carried out as advised by BAPHIQ, and that post-processing contamination was prevented by good handling practices.

7 Risk management

In the risk assessment chapters, Biosecurity Australia assessed the unrestricted risk estimate for each disease agent, to ascertain whether it exceeded Australia's ALOP. In cases where the unrestricted risk was found to be 'very low' or 'negligible' it was concluded that no risk management measures were required for that disease agent with the importation of salted and heat-treated or alkalised preserved eggs from Taiwan. In situations where the unrestricted risk estimation has confirmed that the biosecurity risk associated with importation exceeds Australia's ALOP, consideration is given to measures that could be used to reduce the biosecurity risks associated with the importation of the commodity to acceptable levels (i.e. very low to negligible).

7.1 Salted and heat-treated eggs

As discussed above (Section 5), no specific risk management measures were required for salted and heat-treated eggs, beyond ensuring that the processing of the eggs is carried out as advised by BAPHIQ, and that post-processing contamination is prevented by good handling practices. To ensure that these pre-requisites are met, an import permit will be required, and the product must be accompanied by an official health certificate as described in section 8.1.

7.2 Alkalised eggs

For the following disease agents, the unrestricted risk estimate exceeded Australia's ALOP, and risk management measures are deemed necessary (Table 10).

Table 10. Disease agents requiring risk management

<i>Disease Agent</i>	<i>Unrestricted annual risk</i>
<i>Low pathogenic notifiable avian influenza viruses</i>	<i>Low</i>
<i>Newcastle disease virus</i>	<i>Low</i>
<i>Salmonella Enteritidis and multi-drug resistant S. Typhimurium</i>	<i>Low</i>

Additional risk management measures would be required to address the risks of alkalised eggs for the diseases listed in Table 10 above. This could include treatment to ensure destruction of the pathogen (such as heating) or measures to decrease the likelihood of entry of the disease, such as country or zone freedom, or flock accreditation. Taiwan would also be required to certify to country freedom from HPNAI (in poultry).

In order for Australian Government authorities to be satisfied that Taiwan, or a zone within Taiwan, is free of a given disease, they must have knowledge of the veterinary services of Taiwan and be satisfied that those veterinary services have the capacity for disease control, monitoring and surveillance as appropriate for the disease. In some cases, it might be necessary for the disease to be subject to compulsory notification. Biosecurity Australia recognises that Taiwan may wish to make a submission for access based on equivalent risk management measures, such as flock accreditation schemes or the concept of

compartmentalisation, recently introduced by the OIE. These would need to be assessed on a case-by-case basis. A rigorous assessment of any application for compartmentalisation or flock accreditation schemes will be undertaken to ensure that effective biosecurity measures are implemented and maintained throughout the complete chain from farm to processing for export. A detailed submission will need to be provided by the veterinary authority of Taiwan and Australia will conduct an on-ground assessment of the proposed compartment or flock accreditation scheme.

8 Quarantine measures

8.1 Salted and heat-treated eggs ¹

- 1 Each consignment must be accompanied by a Government Veterinary Certificate in accordance with the Office International des Epizooties (OIE) International Terrestrial Animal Health Code 'Model international veterinary certificate for meat of domestic animals' (Appendix 4.2.1. of the Code) signed by an Official Veterinarian of Taiwan. The certificate must provide details of:
 - a. the packaging of the salted preserved eggs including details of the labelling,
 - b. the addresses and veterinary approval numbers of establishments at which the salted and heat-treated preserved eggs were prepared and the establishment at which they were stored prior to export,
 - c. the names and addresses of the exporter and the consignee.
- 2 The Official Veterinarian of Taiwan must certify in English, that:
 - a. The ducks from which the eggs were derived have been continuously resident in Taiwan since hatching;
 - b. The establishment where the salted and heat-treated preserved eggs were processed and any establishment where the salted and heat-treated preserved eggs were stored prior to shipment to Australia, have current Australian Quarantine and Inspection Service (AQIS) approval for facilities and hygienic operation;
 - c. Officials of the Competent Authority of Taiwan supervised the operation of the plants in which salted and heat-treated preserved eggs were processed for export to Australia.
 - d. During processing, the duck eggs were cooked in steam for 45 minutes at 100°C, to reach a core temperature of 85 °C.
 - e. A quality assurance program is in place to ensure that product destined for export to Australia is kept separate from product not eligible for export to Australia, and is handled in such a way as to ensure that there is no cross-contamination.
 - f. The salted and heat-treated preserved eggs were prepared for export and packed on (dates) in bags, wrappers or packing containers which were clean and new, and in a manner which prevented contamination.
 - g. The identification number of the establishment where the salted and heat-treated preserved eggs were prepared is readily visible on the package or wrapping containing the eggs, in such a way that the numbers cannot readily be removed without damaging the package or wrapping.

- h. The salted and heat-treated preserved eggs were not exposed to contamination prior to export.
- i. The eggs are being transported to Australia in a clean container sealed with an Official Government seal; the container contains only eggs eligible for entry into Australia.

¹ IMPORTERS SHOULD NOTE: Imported food must comply with the *Imported Food Control Act 1992* and the Australia New Zealand Standards Code (FSC) in its entirety. Under the *Imported Food Control Act 1992*, AQIS may inspect, or inspect and analyse imported food to determine compliance with the FSC. These food safety and labelling requirements are separate from, and additional to, Australian quarantine requirements. Information on the FSC may be obtained from Food Standards Australia New Zealand (FSANZ) (www.foodstandards.gov.au).

9 Review

Quarantine measures for importation may be reviewed if there are any changes in the source country's import policy or animal disease status, or at any time at the discretion of Australia's Director of Animal and Plant Quarantine.

Highly pathogenic notifiable avian influenza (HPNAI)

10.1 Risk assessment – alkalised eggs

Release assessment

Taiwan has never reported infection with HPNAI to the OIE (OIE 2006). While this remains the case, the likelihood of introduction of HPNAI in alkalised duck eggs from Taiwan is negligible. Given the high levels of infection with HPNAI which are currently being experienced in many parts of Asia, it is possible that HPNAI will be reported in Taiwan in the future. Should Taiwan report a case of HPNAI, the following factors would need to be considered.

Ducks and other waterfowls are a natural reservoir of influenza A viruses. HPNAI is readily transmissible, especially among birds kept in large groups. Therefore, it can be assumed that, if HPNAI is present within a flock of ducks, a large proportion of the flock will be infected. Depending on the strain of HPNAI which is present, the infection may not be noticed in duck flocks as many AI viruses which are highly pathogenic for chickens do not cause clinical disease in ducks.

AI virus can be present in, or on the surface of, eggs laid by naturally-infected hens. H5N2 virus was isolated from the albumen, the yolk and the shell surface of infertile eggs laid by infected hens during the 1983-84 outbreak of HPNAI in Pennsylvania (Cappucci et al. 1985). Data reported in the same study indicated that the virus can survive for at least several days in the albumen and yolk of eggs stored at 10-18 °C. There is generally a large drop in egg production in flocks experiencing an outbreak of HPNAI; however, AI virus has been isolated from clinically unaffected birds during an outbreak (Cappucci et al. 1985). Therefore, HPNAI could be present in, or on, a proportion of eggs laid by ducks with or without clinical signs in an infected flock.

Based on this information, the overall likelihood that HPNAI would enter Australia through the importation of alkalised duck eggs from Taiwan should HPNAI occur there in the future, was assessed as **moderate**.

Exposure assessment

As explained in the Chapter on Methods, low biosecurity backyard poultry and wild birds are the only exposure groups at significant risk.

Preserved eggs are a delicacy item and it is assumed that minimal waste would result from the purchase and consumption of these products. It is likely that only a small proportion of those households that keep backyard poultry and feed them kitchen scraps would also purchase preserved duck eggs from Taiwan. The waste fraction of the egg that does become available to wild birds or to backyard poultry will consist mostly of egg shell, which would have been

directly exposed to the alkalising treatment. Although unable to conclude that there will be a complete inactivation of any contaminating virus, it is likely that some degree of inactivation of surface virus will occur. Waste generated from restaurant use will generally be disposed of in municipal refuse dumps, while waste from domestic consumption could be fed to backyard poultry or added to household composts. It is considered that only a small percentage of imported preserved eggs will eventually become available to susceptible exposure groups.

HPNAI, within egg waste, is likely to survive in the environment under ambient temperatures for several days, giving ample time for wild birds to locate and scavenge accessible material. The time between feeding of scraps and consumption by low biosecurity backyard poultry is likely to be very short, so environmental degradation of the disease agent will be minimal.

Overall, it was assessed that the likelihoods that backyard poultry (PLEbp) and wild birds (PLEwb) would be exposed to an infectious dose of HPNAI virus are **low**.

Consequence assessment

Likelihood of establishment and spread.

The likelihood of establishment and spread of HPNAI in Australian poultry has been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. In that report, Biosecurity Australia assessed the likelihood that HPNAI would spread, (before being identified and eradicated), beyond an initially exposed group of backyard poultry or wild birds as **very low**, and **low**, respectively.

Impacts of establishment and spread.

The impacts of establishment and spread of HPNAI in Australian poultry have been examined previously in the *Draft Generic Import Risk Analysis Report for Chicken Meat*. As detailed in that report, Biosecurity Australia has assessed the impact of HPNAI spreading to commercial poultry, and an eradication program being implemented in Australia, as **moderate**.

Risk estimation

While Taiwan maintains its HPNAI free status, the risk of introduction of HPNAI in imported alkalised eggs is negligible, and this achieves Australia's ALOP, so no risk management measures are required for HPNAI. However risk assessment was performed, to determine what risk management measures, if any, would be required, should HPNAI be reported in Taiwan.

After combining the likelihoods of entry (LR) and exposure (PLEbp and PLEwb), as detailed on page 27, the partial annual likelihoods of entry and exposure for backyard poultry and wild birds (PALEEbp and PALEEwb respectively) were both estimated as **low**.

Combining these with the likelihoods of establishment and spread (PLESbp and PLESwb) as discussed on page 27, resulted in estimates of **very low** for the partial annual likelihoods of entry, exposure, establishment and spread (PALEEESbp and PALEEESwb).

Combining this with the impact of the establishment of the disease, as detailed on page 27, provided partial annual risk estimates of **very low** for both backyard poultry and wild birds.

Finally, combining the partial annual risks for backyard poultry and wild birds according to the rules detailed on page 28 yielded a final risk for HPNAI of **low**.

As this unrestricted risk estimate exceeds Australia's ALOP, risk management would be necessary for this organism with the importation of alkalised eggs.