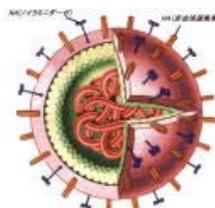


**QUALITATIVE RISK ASSESSMENT:  
LOW PATHOGENIC NOTIFIABLE AVIAN  
INFLUENZA (H5 AND H7) IN POULTRY MEAT**



**Version 1**

**Prepared by:**

**Dr Mirzet Sabirovic  
Simon Hall  
Dr Andrew Paterson**

**Approved by:**

**Nigel Gibbens  
(29 December 2004)**

## **Table of Contents**

Executive Summary .....	3
1. Introduction .....	4
2. Risk question .....	4
3. Scope.....	4
4. Hazard identification.....	5
5. Risk assessment.....	6
5.1. Release assessment.....	7
5.2. Risk estimation and conclusion.....	22
6. References.....	22

## Executive Summary

The risk of introducing low pathogenic notifiable avian influenza (LPNAI) virus of the H5 or H7 subtype infection to a country which imports fresh poultry meat for human consumption from a country not known to be free from LPNAI is considered to be negligible (Table 1).

**Table 1: Risk pathway and summary of release assessment**

There is an indeterminable likelihood that LPNAI will be present in migrating waterfowl and other wild birds entering the territory of the exporting country
There is a low likelihood that LPNAI infection will be present in wild birds in the exporting country
There is a low likelihood that LPNAI will be introduced to a commercial poultry flock and remain active and undetected when birds are selected for slaughter
There is a low likelihood that commercial poultry for slaughter will be infected and that infection will remain undetected during loading, transport and lairage
There is a low likelihood that infected commercial poultry for slaughter will pass ante-mortem or pre-slaughter health inspection
There is a low likelihood that stunning, killing or bleeding will result in cross-contamination of slaughtered poultry with LPNAI virus
There is a low likelihood that scalding, plucking and 'whole bird inspection' will result in contamination of poultry carcasses with LPNAI virus
There is a negligible likelihood that number of LPNAI virus particles sufficient to transmit infection will remain on the internal or external surfaces of the carcass following evisceration, post-evisceration inspection and final washing

The release assessment pathway contains several steps where the likelihood of LPNAI virus presence is successively reduced. Considered together, these successive risk reduction steps lead to an overall negligible likelihood that exported poultry carcasses or poultry meat produced for human consumption in accordance with international guidelines will have sufficient quantities of virus to initiate LPNAI infection in susceptible species.

This assessment is also supported by practical experience. That is, no introduction of LPNAI to poultry operations in any country has ever been attributed to imports of infected poultry meat.

## 1. Introduction

This qualitative risk assessment was undertaken to assist the process of identifying appropriate Sanitary and Phytosanitary (SPS) measures to manage the risk of importing LPNAI infection in fresh poultry meat. According to the SPS Agreement, these measures must not be restrictive to trade while maintaining appropriate levels of protection (ALOP).

The ALOP for a country that considers itself free from LPNAI infection is that imported poultry or poultry products must present a negligible risk that LPNAI will become established in domestic and wild birds.

## 2. Risk question

*“What is the risk of introducing low pathogenic notifiable avian influenza (LPNAI) virus of H5 or H7 subtypes to a country that imports fresh poultry meat for human consumption from a country not known to be free from LPNAI”?*

## 3. Scope

This qualitative risk assessment deals with:

- LPNAI which includes all avian influenza viruses of H5 and H7 subtypes that are not HPNAI viruses, as defined in Article 2.7.12.5 of the OIE Code (OIE, 2004a);
- Poultry, defined by the OIE Code as ‘*all birds reared or kept for the production of meat or eggs for consumption...*’ (OIE, 2004a, p.299);
- Poultry meat which is fresh, frozen or chilled poultry meat which has not been heat treated or subjected to any other processing capable of destroying virus;
- Poultry meat which is produced for human consumption in accordance with the Codex Alimentarius Recommended Code of Hygienic Practice

for Poultry Processing (CAC/RCP 14-1976) (Codex Alimentarius Commission, 1976);

- Poultry meat that is imported from a country believed to be free from highly pathogenic notifiable avian influenza (HPNAI) but not known to be free from LPNAI.

#### **4. Hazard identification**

The hazard of interest is LPNAI virus, as defined by the scope of this risk analysis. These viruses may undergo genetic mutation and become HPNAI viruses capable of causing severe outbreaks of disease in poultry

##### **Supporting evidence**

Avian influenza (AI) virus is a single-stranded RNA virus. It is a member of the *Orthomyxoviridae* family (Swayne and Beck, 2004). The virus has two types of glycoprotein antigens (H – haemagglutinin; N – neuraminidase) located at the outer surface of the virus. To date, 15 haemagglutinin (HA) and 9 neuraminidase (NA) subtypes of the virus have been isolated from birds and may occur in any possible combinations (Capua and Alexander, 2002).

The OIE divides AI viruses into highly pathogenic notifiable avian influenza (HPNAI) viruses and low pathogenic notifiable avian influenza viruses (LPNAI) (OIE, 2004a) according to their ability to cause the disease in poultry.

However, H5 and H7 viruses with multiple basic amino acids at the HA0 cleavage site can be HPNAI even if they fail to induce any disease signs in infected chicken (Alexander, 2004).

Historically, the HPNAI viruses of H5 and H7 subtypes have been associated with most severe outbreaks of avian influenza in poultry (Easterday and others, 1997) and are therefore of importance for international trade.

The AI viruses have a high mutation rate due to their single-stranded genomes “*which lack an error correction mechanism*” (Ferguson and Bush, 2004, p.12). On occasions, it has been demonstrated that LPNAI viruses have been introduced into commercial poultry and subsequently emerged as HPNAI viruses due to mutation (Tollis and Di Trani, 2002; Capua and others, 2002). The primary route of infection with AI in poultry is faecal-oral transmission.

## 5. Risk assessment

For the purpose of this qualitative risk assessment, the following terminology will apply:

Term	Definition
AI	Avian Influenza
LPNAI	Low Pathogenic Notifiable Avian Influenza of H5 and H7 subtypes
LPAI	Low Pathogenic Avian Influenza of other H subtypes, but not LPNAI
HPNAI	Avian influenza viruses that “ <i>have an intravenous pathogenicity index (IVPI) in 6-weeks 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 or greater than 1.2 or cause less than 75% mortality in an intravenous lethality test should be sequenced to determine whether multiple basic amino acid motif is similar to that observed with other HPNAI viruses, the isolate being tested should be considered as HPNAI</i> ” (OIE, 2004a, p.299)
Poultry meat	Fresh, frozen or chilled poultry meat which has not been heat treated or subjected to any other processing capable of destroying virus; produced for human consumption in accordance with the Codex Alimentarius Recommended Code of Hygienic Practice for Poultry Processing (CAC/RCP 14-1976) (Codex Alimentarius Commission, 1976)
Exporting country	A country believed to be free from HPNAI but not known to be free from LPNAI through surveillance

For the purpose of the release assessment section (Section 5.1) of this qualitative risk assessment, the following terminology will apply (OIE, 2004):

<b>Term</b>	<b>Definition</b>
<b>Likelihood</b>	Probability; the state or fact of being likely
<b>Likely</b>	Probable; such as well might happen or be true; to be reasonably expected
<b>High</b>	Extending above the normal or average level
<b>Highly</b>	In a higher degree
<b>Low</b>	Less than average; coming below the normal level
<b>Negligible</b>	Not worth considering; insignificant
<b>Remote</b>	Slight, faint
<b>Would</b>	To express probability; past of Will: expressing a wish, ability, capacity, probability or expectation

### **5.1. Release assessment**

This section considers the release assessment pathway. It assesses the likelihood of LPNAI virus presence at each successive step within the pathway that leads to production of fresh poultry meat for human consumption that is destined for export from a country not known to be free from LPNAI.

**5.1.1. There is an indeterminable likelihood that LPNAI infection will be present in migrating waterfowl and other wild birds entering the territory of the exporting country**

**Assumptions:**

- *LPNAI viruses are detected on a regular basis in migratory waterfowl and other wild birds;*
- *LPNAI viruses are detected on an irregular and unpredictable basis in migratory waterfowl and other wild birds;*
- *The proportion of LPNAI virus isolates is low compared to other LPNAI viruses isolates from migratory waterfowl.*

**Supporting evidence**

Wild aquatic birds, shorebirds and gulls are considered to be the natural host of AI virus without showing clinical signs of the disease (Suarez, 2000). In wild aquatic birds, the AI virus replicates in the cells lining the intestinal tract and is excreted in high concentrations in the faeces (up to  $10^{8.7}$  50% egg infectious doses/gram)(Webster, 1998). “*The complete host ecology of influenza is unlikely ever to be fully understood*” (Tollis and Di Trani, 2002, p.204) because of the complex interactions between the virus and the aquatic birds. The following examples indicate that the distribution of AI viruses varies to a great extent and their detection depends on the year and season.

Systematic surveillance of the presence of AI viruses, carried out from 1973 to 1986 and involving over 20,000 birds, resulted in AI virus isolation from 15% of duck and geese samples and from 2% of samples from other birds. These findings indicate that the AI virus is primarily present in migratory waterfowl (various authors cited in Capua and Alexander, 2002). The relative proportion of H5 and H7 subtypes among the AI virus isolates was not indicated.

Another study carried out from 1975 to 1999 involved 3299 migratory birds in the North Caspian region. In total, 344 subtypes of the AI virus were obtained. The H5 subtype was isolated on two occasions (0,3%) (Lvov and others, 2001). The isolation of H5 subtype was only made in one year during the total observation period of 24 years.

AI virus was isolated from 12% of 3200 samples collected from 1900 migratory ducks in Sweden during 2002. Five different types of AI virus were identified, among them a low pathogenic H5N2 virus (Wallensten and others, 2004). The relative proportion of H5 and H7 subtype among the AI virus isolates was not indicated.

Twenty isolates (6,7%) of AI virus of eight different subtypes were obtained from 311 samples collected from free-living ducks trapped between 1999 and 2001 in Italy. Two samples (0,6%) yielded an H7N3 subtype. This was the first identification of this subtype of the AI virus in the region under study (Campitelli and others, 2004). The following year, this subtype was implicated in outbreaks of HPNAI in commercial poultry.

Twenty-two isolates (2,7%) of the AI virus of different H subtypes were obtained from 802 cloacal samples from ducks and coots collected from 1993 to 1998 in Italy. Overall seroprevalence to various AI viruses was higher in ducks (52,2%) compared to coots (7.2%). This study indicated a continuous circulation of LPAI virus of the H5 subtype in ducks during all sampling seasons. This study also suggested a significant antigenic diversity within at least some subtypes and raised the issue of deciding which reference subtype strains should be used as reference viruses for AI surveillance (De Marco and others, 2004).

10,945 samples collected from feral waterfowl from 1998 to 2002 in Taiwan yielded 232 (2,1%) AI virus isolates of seventeen different H subtypes. The

H7 subtype was detected in samples collected from three consecutive years (Cheng and others, 2004). The relative proportion of H7 subtype among the AI isolates was not indicated.

One hundred and eight isolates of AI virus of different H subtypes were isolated from waterfowl in Alaska from 1991 to 1994 (Ito and others, 1995). The H7 subtype was isolated on only one occasion (0,9%). In a study in Canada, AI virus was isolated from 60% of juvenile ducks before migration (Hinshaw and others, 1980).

There are numerous examples in the literature of different AI viruses being detected in wild aquatic birds. Historic surveillance data of wild aquatic birds indicate that the detection of LPNAI viruses is infrequent, unpredictable and proportionally low compared to other detected AI viruses. There is also no information regarding the extent that the antibody response against other types of AI viruses will have on the establishment and regular circulation LPNAI virus infection in wild aquatic birds.

### ***5.1.2. There is a low likelihood that LPNAI infection will be present in wild birds in the exporting country***

***Assumptions:***

- *The frequency of transmission of LPNAI infection from migratory waterfowl to local birds that share their habitat is irregular;*
- *There is a low likelihood that LPNAI infection will be established in wild birds in the exporting country.*

### **Supporting evidence**

Data presented in section 5.1.1 indicate that AI tends to be detected in birds that use the major waterfowl flyways around the world. Infected waterfowl are more likely to be detected in late summer, particularly when young birds assemble for migration.

AI virus may be transmitted from migratory waterfowl to non-migratory lake and wetland birds and wild ducks due to shared habitat. One study suggested that AI circulation in non-migratory bird species occurs at low levels and is limited to a few subtypes compared to migratory waterfowl (De Marco and others, 2004). Another study (quoted in De Marco and others, 2004) also demonstrates that “*wild and domestic ducks differ with regard to the Influenza HA subtypes most frequently circulating in both groups*” (De Marco and others, 2004, p.206). However, this may not apply to all geographical areas (Alexander, 2004). This would mean that under certain circumstances non-migratory bird species may be infected with the same AI virus as migratory birds.

There is a low likelihood that LPNAI will be introduced to a commercial poultry flock and remain active and undetected when poultry are selected for slaughter

***Assumptions:***

- *Introductions of LPNAI virus into commercial poultry are irregular events that usually occur following a failure of farm biosecurity;*
- *It is likely that such events mainly occur in those operations that are located on major migratory flyways or have close contact with local wild birds that share habitat with migratory waterfowl;*
- *It is likely that such introductions will result in at least mild clinical signs, affecting production parameters that are monitored at commercial poultry farms supplying export markets;*
- *It is unlikely that LPNAI would be introduced to a commercial poultry flock and remain active and undetected in poultry selected for slaughter.*
- *It is likely that LPNAI virus replication in affected domestic poultry will be reduced or eliminated after seroconversion.*

### Supporting evidence

Most LPAI virus introductions into domestic poultry have been recorded in poultry operations located in areas that are on main migratory flyways for waterfowl (Banks and others, 2001; Halvorson, 2002). One hundred and eight introductions of the LPAI virus into domestic poultry have been recorded during the period of 25 years in Minnesota (USA). Twenty (18,5%) of these introductions have been LPNAI virus of H5 or H7 subtype (Halvorson, 2002). On other occasions, it is considered that LPNAI viruses have been introduced into commercial poultry from wild birds reservoir and subsequently emerged as HPNAI viruses due to mutation (Tollis and Di Trani, 2002; Capua and others, 2002). AI virus introductions into domestic poultry appear to be more frequent in countries that still pursue practices such as *“surface storage of drinking water, rearing mixed species on the same farm, failure to build bird-proof food stores, construction of artificial ponds to attract waterfowl”* (Alexander, 2000, p.11).

Domestic poultry are considered to be aberrant (new) hosts for AI viruses. When introduced, the virus will replicate and occasionally cause disease (Suarez, 2000). On most occasions the disease may not transmit well enough to cause an epidemic (Suarez, 2000); on other occasions it may lead to widespread epidemics if not controlled (Alexander, 2004). However, in most cases, the introductions *“do not continue for long because of the control efforts or failure of the virus to adapt to the new host”* (Suarez, 2000, p16).

Following introduction to a poultry farm, the virus has the ability to rapidly spread within flocks at the affected farm (Capua and others, 2002). Easterday and others (1997) consider that the incubation period may vary from a few hours to three days in individual birds and up to 14 days following the introduction of the virus into a poultry flock. Once the virus is introduced to poultry it is easily spread within a flock by direct contact with infected faeces. According to Lu and others (2004), heavy virus shedding (90%-100% infected

birds shedding) occurred between 4 and 7 days after the onset of the disease. A few birds (15%) continued to shed virus at 13 days post-disease onset. However, *“most viral shedding from infected poultry stops after seroconversion”* (Halvorson, 2002) which usually takes up to 14 days (Ritchie and Carter, 1995) after infection.

Historic data indicate that the impact on production parameters in the affected poultry farms is one of the primary reasons for the detection of the introduction of LPNAI virus into commercial poultry. There is no data in the literature to indicate that LPNAI infection may occur in domestic poultry without causing any clinical signs whatsoever. It is considered (European Commission, 2000, p.13) that the severity of the disease produced by LPAI is *“greatly influenced by the strain of the virus, the species and age of host; the immune status of the host against the virus and particularly the presence of other infectious agents such as Pasteurella spp, Newcastle disease virus (including vaccine strains), avian pneumovirus, infectious bronchitis virus, E. coli and Mycoplasma spp, immunodeficiency conditions and environmental factors (such as excess ammonia, dust, hot or cold temperatures)”*.

Following spread within flocks at the affected farm, the virus also has the ability to rapidly spread to other poultry farms (Capua and others, 2002). This is primarily caused by movements of humans or shared contaminated equipment. Webster (1998) considers that the AI outbreaks in chicken and turkeys in Pennsylvania (USA) in 1983 to 1984 and Mexico in 1993 *“could have been prevented if domestic poultry had been raised in ecologically controlled houses with a high standard of security and limited access”*.

It is also considered that vaccination will increase the likelihood that AI infection will go unnoticed since there will be no clinical signs. However, vaccination is likely to further reduce quantities of LPNAI virus particles in faeces (Alexander, 2004).

**5.1.3. *There is a low likelihood that commercial poultry for slaughter will be infected and that infection will remain undetected during loading, transport and lairage***

***Assumptions:***

- *If birds are incubating disease before being selected for slaughter, the stress of transportation may cause clinical disease to develop;*
- *It is unlikely that birds would be infected and that infection would remain undetected during loading, transport and lairage;*
- *If LPNAI virus is introduced from wild birds during loading, transport and lairage there is insufficient time for incubation and spread within poultry selected for slaughter.*

**Supporting evidence**

Withholding feed for 6-10 hours prior to slaughter is aimed to reduce the crop content to a minimum. There is no data in the available literature to indicate the prevalence of LPNAI (H5 or H7) virus in faeces of poultry presented for slaughter or possible external contamination (e.g. feathers) during transport and lairage.

Poultry are transported to a slaughterplant and prepared for slaughter within a relatively short period of time (usually less than 12 hours in total after loading on the farm) (Northcutt, 2004). There is a remote possibility that cross-contamination by over flying free-living birds may occur during the transport and lairage.

Poultry are kept in a holding area for a very short period of time to avoid loss of carcass yield. Keeping poultry in the holding area for longer than 12 hours after the last feeding may have a harmful impact on the gastrointestinal tract. In this case, poultry start losing the mucosal lining resulting in a much weaker

intestine that may easily break during evisceration (Northcutt, 2004). The holding area at the slaughterplant is designed to protect birds from stress (e.g. birds are protected from the weather and well-ventilated). This environment allows time for the poultry to settle down before being sent to the processing line (Barker and others, 2000)

**5.1.4. *There is a low likelihood that infected commercial poultry for slaughter will pass ante-mortem or pre-slaughter health inspection***

***Assumptions:***

- It is unlikely that a high proportion of poultry will be incubating disease without a proportion of them showing clinical signs when presented for ante-mortem inspection,*
- It is unlikely that such poultry would pass ante-mortem inspection and qualify as a consignment fit to be slaughtered for export purposes.*

**Supporting evidence**

According to the Codex (Codex Alimentarius Commission, 1976) guidelines ante-mortem inspection and post-mortem inspection should be carried out by the appropriate official agency, under veterinary supervision. In some countries ante-mortem inspection may be replaced by pre-slaughter health inspection at the premise of origin.

Unfit poultry should be segregated and disposed of in an appropriate manner in order to prevent the spread of disease. Poultry found to be moribund or dead on arrival, appearing unhealthy or rejected for any other reason are not slaughtered.

**5.1.5. There is a low likelihood that stunning, killing or bleeding will result in cross-contamination of slaughtered poultry with LPNAI virus**

**Assumptions:**

- *It is unlikely that stunning, killing or bleeding would cause cross-contamination of slaughtered poultry*
- *Virus replication requires live host cells, therefore, it is highly unlikely that the virus replication will continue in slaughtered poultry.*

**Supporting evidence**

Available data does not provide evidence of the extent that stunning, killing and bleeding of poultry may have on LPNAI virus survival or cross-contamination of slaughtered poultry. However, it is a fundamental property of viruses that they require living host cells in order to replicate so the number of viable virus particles can only decrease from this point. This is in contrast to some known bacterial contaminants (i.e. *Salmonella*, *Campylobacter*) which could multiply on poultry carcasses.

Slaughter processes are designed to minimise the number of birds that are not properly bled and dead before scalding. Should these processes fail, the respiratory and internal organs may become contaminated and result in a change in carcass appearance (red in colour) and collapsed lungs which may be difficult to remove (Northcutt, 2004). Such poultry are unlikely to pass as being fit for human consumption at inspection during processing.

---

**5.1.6. There is a low likelihood that scalding, plucking and ‘whole bird inspection’ will result in contamination of poultry carcasses with LPNAI virus****Assumptions:**

- *It is likely that cross-contamination of carcasses will occur during scalding and plucking,*
- *It is highly likely that the number of present LPNAI virus particles will be reduced through removal of soiled feathers and washing, particularly if hot or sanitised water is used,*
- *It is likely that a few residual virus particles would be retained in feather follicles,*
- *It is highly likely that any visibly abnormal carcasses will be removed during ‘whole bird inspection’ inspection.*

**Supporting evidence**

Scalding is the process where “*turbulent hot water*” (usually heated to 52<sup>0</sup>C or 58<sup>0</sup>C to 60<sup>0</sup>C) “*is used to transfer heat to the feather follicles, which then relax allowing feathers to be removed mechanically in the pluckers*” (Barker and others, 2000, p.95). This procedure poses an increased risk for the cross-contamination of carcasses which is well recognised under public health considerations. Thus, the process is designed to minimise cross-contamination as much as practically possible by adjusting the flow of water (e.g. chlorinated or not chlorinated) into the scald tank (i.e. dilution factor). This rate of flow will depend on the type and species of poultry slaughtered and number of poultry per minute. As a general rule, the scald tank is cleaned and disinfected on a daily basis following completion of processing.

*“Recent developments have concentrated on reducing running costs, making scald tanks easier to clean, and most importantly, on reducing microbial contamination of carcasses. To this end many processors have installed multiple scalding systems with birds moving into ever cleaner water”* (Barker and others, 2000, p.96)

Although scalding is generally considered to pose an increased risk of cross contamination, certain studies on bacterial pathogens (e.g. *Campylobacter*) (quoted in Keener and others, 2004) have found significant reduction in the bacterial count on broiler carcasses. This reduction ranged from 4.73 log<sub>10</sub> on carcasses sampled pre-scald to 1.80 log<sub>10</sub> after the carcasses were scalded.

Although scalding may contribute to a reduction of the microbial load on carcasses due to the dilution factor, certain studies on bacterial pathogens (e.g. *Campylobacter*) (quoted in Keener and others, 2004) have found that de-feathering may contribute to an increased bacterial count on broiler carcasses. This count was found to increase to 3.70 log<sub>10</sub>. According to studies (quoted in Keener and others, 2004, p.108-109) *“the rubber fingers in the mechanical picker act to cross-contaminate birds”*. In addition, bacteriological studies have found that certain bacteriological pathogens (i.e. *Campylobacter*) may be introduced the subcutaneous layer via feather follicles (various authors cited in Keener and others, 2004).

Therefore, maximum attention is paid to avoiding cross-contamination during de-feathering. Mechanical pluckers must prevent the scattering of feathers and be easy to clean. If waxing is used, it must be carried out using wax approved as edible grade and stored under hygienic conditions (Radakovic, 2004). A spray or a continuing flow of clean water or water containing an approved solution is used to wash poultry carcasses during de-feathering. This is a decontamination practice designed primarily to reduce the rate of the attachment of microorganisms to the carcass for public health purposes.

In addition, “on the ‘whole bird inspection’ point all visibly abnormal carcasses are removed before the evisceration thus minimising further the risk of cross-contamination. At this point, the birds affected with generalised conditions such as septicaemia are rejected” (Radakovic, 2004).

**5.1.7. There is a negligible likelihood that number of LPNAI virus particles sufficient to transmit infection will remain on the internal and external surfaces of the carcass following evisceration, post-evisceration inspection and final washing**

**Assumptions:**

- *It is highly likely that the major concentration of LPNAI virus particles will be contained in the respiratory and alimentary tracts;*
- *It is highly likely that the most of LPNAI virus particles will be removed from poultry carcasses during evisceration (i.e. removal of the respiratory and alimentary tracts);*
- *It is highly likely that the number of LPNAI virus particles that remain on carcass surfaces will further be significantly reduced due to extensive washing used to minimise carcass contamination for reasons of food safety;*
- *It is highly likely that any visibly abnormal or contaminated carcasses or viscera will be removed during post-evisceration inspection for reasons of food safety;*
- *There is scientific evidence to demonstrate the absence of LPNAI infectious particles in poultry meat or bone.*

**Supporting evidence**

Evisceration may be mechanical or manual. Significant effort is put into the clean removal of the alimentary tract and other internal organs. If mechanical evisceration is used, the equipment is configured to suit a particular size or

type of carcass and is washed regularly. If manual evisceration is used, particular attention is given to cleaning by water. GIBLETS for human consumption are removed in a way that prevents cross-contamination of carcasses or equipment.

During evisceration, there is a high potential for the rupture of the intestine with resulting contamination of carcasses with faecal material (Radakovic, 2004). In addition, operator handling, speed of processing or aerosol spread may also contribute to potential cross-contamination.

Complete prevention of cross-contamination is impossible to achieve in practice. However, these processes are designed to minimise the contamination of carcass surfaces. Northcutt (2004) considers that visibly contaminated carcasses will have to be removed and then washed, trimmed or vacuumed at a reprocessing station which significantly increases the costs of processing.

Tollis and Di Trani (2002) and Suarez and Schultz-Cherry (2000) consider that the main difference between HPNAI and LPNAI viruses is systemic versus local replication, respectively. In experimental conditions, inoculation of live birds with *“LPNAI viruses resulted in predominant infection in the upper and lower respiratory tract... while infection in gastrointestinal tract varied from poor to strong predilection for replication. LPNAI viruses failed to produce viraemias and no virus was isolated from bone or breast and thigh meat”*. This study also concluded that residual virus may have remained in the thoracic region of slaughtered birds after lung removal (Swayne and Beck, 2004a: In press).

Codex Alimentarius Recommended Code of Hygienic Practice for Poultry Processing (CAC/RCP 14-1976) (Codex Alimentarius Commission, 1976) requires that *‘after evisceration and inspection carcasses should be washed’*.

Following evisceration, carcasses are washed using potable water, or water

containing an approved sanitiser, using spray or immersion washing to ensure that carcasses are free from visible external (including faecal) contamination (Kenner and others, 2004). Immersion washing may be performed subject to meeting approved specific conditions.

Considering the presence of LPNAI viruses in poultry carcasses, it should be emphasised that no LPNAI virus have been found in poultry meat or bone (Swayne and Beck, 2004a: In press). This would be expected given the absence of viraemia in live poultry following infection with LPNAI viruses. In addition, it should also be emphasised LPNAI virus, if present, will not multiply during slaughter and processing of poultry because any virus requires live cells to multiply. The vast majority of LPNAI virus, if present, would be removed during evisceration. Evisceration is therefore regarded as the most important risk reduction measure because it removes the organs (respiratory and gastrointestinal tract) considered to be the major sites of LPNAI virus replication in live birds. In addition, the various wash systems that are used at commercial poultry plants generally consume between 100 to 200 litres (25-50 US gallons) per minute of water (Kenner and others, 2004). This amount of water will further reduce the number of LPNAI virus particles on the carcass surface due to the high dilution factor.

#### **5.1.8. Completion of release assessment**

According to the OIE methodology, if the likelihood is assessed negligible at any step of the release assessment pathway and exposure assessment pathway the risk assessment may be completed at that step.

In our release assessment pathway, we have concluded that several risk reduction steps have resulted in a negligible likelihood of LPNAI virus presence on poultry carcasses after evisceration and final inspection, and final washing (Step: 5.1.8).

## **5.2. Risk estimation and conclusion**

The risk of introducing LPNAI infection to a country which imports fresh poultry meat produced for human consumption in accordance with international guidelines from a country not known to be free from LPNAI is considered negligible.

This conclusion is also supported by practical experience. That is, no introduction of AI (LPNAI or HPNAI) to poultry operations in any country has ever been attributed to imports of infected poultry meat.

*In addition, “migratory bird flyways crossing...high density ...poultry farms concentrated in limited area of the country, and typical features such as live-bird markets and outdoor rearing of domestic poultry are considered as major factors for the introduction of AIVs and transmission from the wild aquatic bird reservoirs to land-based poultry” (Di Trani and others, 2004, p.382).*

## **6. References**

- Alexander, D.J. (2004). Personal Communication, December 2004, Veterinary Laboratory Agency, New Haw, Addlestone, Surrey, KT15 3NB, United Kingdom.
- Alexander, D.J. (2000). A review of avian influenza in different bird species. *Veterinary Microbiology*, 74, 3-13.
- Banks, J., Spiedel, E.M., Capua, I., Fioretti, A., Piccirilo, A., Moore, E.H., Plowright, L., Alexander, D.J. (2001). Changes in the HA and NA genes prior to the emergence of HPAI H7N1 avian influenza viruses in Italy. *International Congress Series*, 1219, 363-367.
- Barker, D., Stork, P.M.T., Lankhaar, J., Stals, P (2000). Primary processing of poultry. In: *Poultry meat processing and quality*. Woodhead Publishing Limited, Abington Hall, Abington, Cambridge CB1 6AH, England, 90-107
- Capua, I., Alexander, D.J. (2002). Review Article: Avian influenza and human health. *Acta Tropica*, 83, 1-6.

- Capua, I., Mutinelli, F., Dalla Pozza, M., Donatelli, I., Puzelli, S., Cancellotti, F.M. (2002). The 1999-2000 avian influenza (H7N1) epidemic in Italy: Veterinary and human health implications. *Acta Tropica*, 83, 7-11.
- Campitelli, L., Mogavero, E., De Marco, M.A., Delogy, M., Puzelli, S., Frezza, F., Facchini, M., Chiapponi, C., Foni, E., Cordioli, P., Webby, R., Barigazzi, G., Webster, R.G., Donatelli, I. (2004). International Congress Series, 1263, 766-770.
- Codex Alimentarius Commission, (1976). Recommended Code of Hygienic Practice for Poultry Processing (CAC/RCP 14-1976). Accessed on 8 December 2004  
([http://www.codexalimentarius.net/download/standards/163/CXP\\_014e.pdf](http://www.codexalimentarius.net/download/standards/163/CXP_014e.pdf))
- Cheng, M.C., Wang, C.H., Kida, H. (2004). Influenza A virological surveillance in feral waterfowl in Taiwan from 1998 to 2002. International Congress Series, 1263, 745-748.
- De Marco, M.A., Campitelli, L., Foni, E., Raffini, E., Barigazzi, G., Delogu, M., Guberti, V., Di Trani, L., Tollis, M., Donatelli, I. (2004). Influenza surveillance in birds in Italian wetlands (1992-1998): is there a host restricted circulation of influenza viruses in sympatric ducks and coots? *Veterinary Microbiology*, 98, 197-208.
- Di Trani, L., Bedini, B., Cordioli, P., Muccillo, M., Vignolo, E., Moreno, A., Tollis, M. (2004). Molecular characterisation of low pathogenicity H7N3 avian influenza viruses isolated in Italy. *Avian Diseases*, 48, 376-383.
- Easterday, B.C., Hinshaw, V.S., Halvorson, D.A. (1997). Influenza: In: Diseases of Poultry, 10<sup>th</sup> ed. Calnek, B.V. and others (Ed), Iowa State University Press, Ames, 583-605.
- European Commission, (2000). The definition of avian influenza. The use of vaccination against avian influenza. European Commission Health & Consumer Protection Directorate-General, Scientific Committee on Animal Health and Animal Welfare. Draft report for possible adoption on 27 June 2000, SANCO/B3/AH/R17/2000.

- Ferguson, N.M., Bush, R.M., (2004). Influenza evolution and immune system. International Congress Series, 1263, 12-16.
- Halvorson, D.A. (2002). The control of low pathogenic avian influenza in Minnesota: successful and economical. Accessed on 8 December 2004 (<http://www.veterinaria.uchile.cl/cd/VIIIpatologia/SEMINARIOS/semi12.pdf>)
- Hinshaw, V.S., Webster, R.G., Turner, B. (1980). The perpetuation of orthomyxoviruses and paramyxoviruses in Canadian waterfowl. *Canadian Journal of Microbiology*, 26, 622-629.
- Ito, T., Okazaki, K., Kawaoka, Y, Takada, A., Webster, R.G., Kida, H. (1995). Perpetuation of Influenza A viruses in Alaskan waterfowl reservoirs. *Archive of Virology*, 140(7), 1163-1172.
- Lu, H., Dunn, P.A., Wallner-Pendelton, E.A., Henzler, D.J., Kradel, D.C., Liu, J., Shaw, D.P., Miller, P. (2004). Investigation of H7N2 avian influenza outbreaks in two broiler breeder flocks in Pennsylvania, 2001-02. *Avian Diseases*, Jan-Mar; (48(1), 26-33.
- Lvov, D.K., Yamnikova, S.S., Gambaryan, A.S., Fedyakina, I.T., Matrosovich, M.M. (2001). Isolation of influenza viruses from wild birds in the Volga River basin and in the North Caspian region. International Congress Series, 1219, 251-258.
- Keener, K.M., Bashor, M.P., Curtis, P.A., Sheldon, B.W., Kathariou, S. (2004). Comprehensive review of *Campylobacter* and poultry processing. Institute of Food technologists. *Comprehensive Reviews in Food Science and Food Safety*, Vol.3, 105-116.
- Northcutt, J.K. (2004). Reference guide for solving poultry processing problems. The University of Georgia Cooperative Extension Service, Athens, GA. Accessed on 7 December 2004 (<http://www.ces.uga.edu/pubcd/b1156-w.html>)
- OIE, (2004). Handbook on Import Risk Analysis for Animals and Animal Products. Vol.1. Office International des Epizooties, 12 rue de Prony, Paris, France.

- OIE, (2004a). Chapter 2.7.12. Highly pathogenic Avian Influenza. Terrestrial Animal Health Code, Thirteenth Edition, Office International Des Epizooties, 12 rue de Prony, Paris, France.
- Radakovic, M. (2004). Personal communication, December 2004. Food Standards Agency, Holborn, London, United Kingdom.
- Ritchie, B.W., Carter, K. (1995). Avian viruses function and control. Winger Publishing, Inc, Lake Worth, Florida, 351-364.
- Suarez, D.L. (2000). Evolution of avian influenza viruses. *Veterinary Microbiology*, 74, 15-27.
- Suarez, D.L., Schultz-Cherry, S, (2000). Immunology of avian influenza virus: A Review. *Developmental and Comparative Immunology*, 24, 269-283.
- Swayne, D.E., Beck, J.R. (2004). Heat inactivation of avian influenza and Newcastle disease virus in egg products. *Avian Pathology*, 33(5), 512-518.
- Swayne, D.E., Beck, J.R. (2004a) (In press). Experimental study to determine if low-pathogenicity and high-pathogenicity avian influenza viruses can be present in chicken breasts and thigh meat following intranasal virus inoculation. In press, *Avian Diseases*, 49: 000-000, 2005
- Tollis, M., Di Trani, L. (2002). Review. Recent developments in avian influenza research: epidemiology and immunoprophylaxis. *The Veterinary Journal*, 164, 202-215.
- Wallensten, A., Munster, V.J., Fouchier, R.A.M., Olsen, B. (2004). Avian Influenza A virus in ducks migrating through Sweden. International Congress Series, 1263, 771-772.
- Webster, R.G. (1998). Influenza: An emerging disease. *Emerging Infectious Diseases*, Vol.4., No.3, July-September 1998. Accessed on 8 December 2004 (<http://www.cdc.gov/ncidod/eid/vol4no3/webster.htm>).