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SCIENTIFIC REPORT

Animal health and welfare aspects of Avian Influenza

Adopted on 13/14 September 2005
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1 GLOSSARY

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<tr>
<td>AI</td>
<td>Avian Influenza</td>
</tr>
<tr>
<td>AIV</td>
<td>avian influenza virus</td>
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<tr>
<td>BIP</td>
<td>EU Border inspection post</td>
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<tr>
<td>DIVA</td>
<td>strategy which allows: Differentiating Infected from Vaccinated Animals</td>
</tr>
<tr>
<td>DPPA</td>
<td>densely populated poultry areas</td>
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<tr>
<td>EC</td>
<td>European Community</td>
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<td>EFSA</td>
<td>European Food Safety Authority</td>
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<tr>
<td>FVO</td>
<td>Food and Veterinary Office - specialised inspection service of the EC</td>
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<tr>
<td>HPAI</td>
<td>highly pathogenic avian influenza</td>
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<tr>
<td>HPAIV</td>
<td>highly pathogenic AI virus</td>
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<tr>
<td>HRP</td>
<td>high risk period</td>
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<tr>
<td>LBM</td>
<td>live bird market</td>
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<tr>
<td>LPAI</td>
<td>low pathogenic avian influenza</td>
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<tr>
<td>LPAIV</td>
<td>low pathogenic AI virus</td>
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<td>MS</td>
<td>Member States of the European Union (15 before and 25 after 1 May 2004)</td>
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<tr>
<td>ND</td>
<td>Newcastle disease (OIE listed poultry disease)</td>
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<tr>
<td>SCAHAW</td>
<td>Scientific Committee on Animal Health and Animal Welfare – Directorate in the Directorate General on Health and consumer protection in the European Commission (DG SANCO) that issued scientific opinions before EFSA was created</td>
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<tr>
<td>SCOFCAH</td>
<td>Standing Committee on the Food Chain and Animal Health</td>
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<td>SVC</td>
<td>Scientific Veterinary Committee</td>
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<td>TOR</td>
<td>Terms of reference</td>
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2 BACKGROUND

Highly pathogenic avian influenza (HPAI) is an extremely contagious viral disease that can affect all species of birds. It is a notifiable disease within the European Union according to Council Directive 82/894/EEC (EC, 1982) and Community measures to control HPAI are laid down in Council Directive 92/40/EEC (EC, 1992a). The disease is listed by the World Organisation for Animal Health (OIE, 2004a). From 1 January 2006 on and for the purpose of the OIE Terrestrial Code in its notifiable form, notifiable Avian Influenza (NAI) is defined as an infection of poultry caused by any influenza A virus of the H5 or H7 subtypes or by any AI virus with an intravenous pathogenicity index (IVPI) grater than 1.2 (or as an alternative at least 75% mortality). NAI virus can be divided into highly pathogenic notifiable avian influenza (HPNAI) and low pathogenic notifiable avian influenza (LPNAI).

The viruses causing HPAI are placed in the Influenza virus A genus of the Orthomyxoviridae family and these are negative-strand, segmented RNA viruses. Influenza A viruses can be divided into subtypes on the basis of the possession of 1 of 16 antigenically distinct haemagglutinin antigens (H1–H16) and 1 of 9 neuraminidase antigens (N1–N9). Virtually all haemagglutinin and neuraminidase combinations have been isolated from birds. The genetic pool for all AI viruses is primarily in aquatic birds, which are responsible for the perpetuation of these viruses in nature.

Influenza A viruses infecting poultry can be divided into two distinct groups on the basis of their ability to cause disease. The very virulent viruses cause highly pathogenic avian influenza (HPAI), which may result in mortality within a flock as high as 100%. These viruses have been restricted to subtypes H5 and H7, although not all viruses of these subtypes cause HPAI.

All other viruses cause a much milder disease, known as low pathogenicity avian influenza (LPAI), consisting primarily of mild respiratory disease and egg production problems in laying birds. LPAI infections may be completely inapparent, particularly when the virus has been recently introduced from the wild to the domestic host, however, sometimes secondary infections or environmental conditions may cause an exacerbation of LPAI infections leading to more serious disease.

Current evidence strongly supports the hypothesis that HPAI viruses are not normally present in wild bird populations and only arise as a result of mutation after H5 or H7 LPAI viruses have been introduced to poultry from wild birds. In recent times, however, following the unusual situation of endemicity which is present in some Asian countries, HPAI of the H5N1 subtype has spilled over to the wild bird population. This situation has never occurred in the past, and therefore the consequences of this epidemiological situation could be unpredictable.

Since 1959 only 24 primary isolates of HPAI viruses from domestic poultry have been reported, but six of these having the greatest socio-economic impact have occurred in the last five years (e.g. outbreaks in 1999/2000 in Italy, 2003 in The Netherlands) along with the unprecedented outbreak currently affecting several Asian countries. Recently the USA and Canada have also experienced HPAI disease outbreaks.

With the exception of some Asian countries, where the disease is currently not yet under control, a rigorous stamping out policy was applied by national authorities. This strategy includes in general the rapid culling of infected poultry and those suspected of being infected together with the implementation of movement restrictions for live poultry and poultry products, increased monitoring and biosecurity measures.
In 2000 the former Scientific Committee on Animal Health and Animal Welfare (SCAHAW) issued an opinion on the definition of avian influenza and vaccination against this disease (SCAHAW, 2000) and in April 2003 adopted a scientific opinion on recent advances in diagnostic techniques and vaccines for several important OIE List A diseases, including avian influenza (SCAHAW, 2003).

3 TERMS OF REFERENCE

In view of the above, the Commission asks the European Food Safety Authority (EFSA) to review 2000 and 2003 scientific opinions (SCAHAW, 2000 and 2003) on avian influenza in the light of more recent scientific data.

The EFSA scientific opinion should in particular describe:

1. an assessment of the risk of the introduction, and possible secondary spread, of LPAI and HPAI into the EU via different commodities, such as live poultry, ornamental birds, hatching eggs, table eggs, fresh poultry and other poultry products. In addition the scientific opinion should describe the risk factors for disease introduction into poultry holdings and surveillance tools and procedures available for early detection of AI in poultry holdings in relation to those risks;

2. the role of “backyard” poultry flocks in the epidemiology of avian influenza and available disease control tools for this specific population;

3. the risk of disease transmission between certain avian species in particular with respect to pigeons and anseriformes;

4. the risk of virus persistence in poultry manure and farm waste and a description of the possible inactivation and disinfection procedures that could be applied to these materials;

5. the animal welfare aspects of avian influenza including the implications of the different control strategies.

4 INTRODUCTION

Avian influenza (AI) is an OIE listed disease, which in its highly pathogenic avian influenza (HPAI) form has become a disease of great importance for animal health and with serious potential implications for human health. Until 1999, AI of the HPAI form was considered a sporadic disease with only 18 outbreaks occurring in domestic poultry world-wide since 1959. The total number of birds involved in all outbreaks over this 40-year period was approximately 23 million. From 1999 onwards, HPAI infections cannot be considered sporadic any longer. Including estimations of the ongoing Asian H5N1 epidemic, in five years over 200 million birds have been affected by this disease. Some outbreaks have maintained the characteristic of minor relevance but others, such as the Italian 1999-2000, the Dutch 2003, the Canadian 2004 and the Asian 2003-2004 have lead to devastating consequences for the poultry industry, negative repercussions on public opinion and in some cases created significant human health issues, including the risk of generating a new pandemic virus for humans via the avian-human link.

The increased relevance of AI in the fields of animal and human health, has highlighted the lack of scientific information on several aspects of the disease, which has hampered the adequate management of some of the recent crises thus resulting in millions of dead animals and concern over loss of human lives and over management of the pandemic potential.
The former Scientific Committee on Animal Health and Animal Welfare of the EU had been asked to address selected issues concerning AI, following the Italian H7N1 epidemic. Two reports, were issued in 2000 and 2003 addressing “The definition of avian influenza and the use of vaccination for avian influenza” and “Diagnostic tools and Vaccination for FMD, CSF, AI and other OIE List A diseases”. Both of these documents address the issue of the definition of AI, and conclude that Low Pathogenicity Avian Influenza (LPAI) viruses should be included in the definition of AI since it has been shown that they are the progenitors of Highly Pathogenic Avian Influenza (HPAI). The issue of vaccination instead is addressed in a rather conservative manner in the first of these documents and with a more open approach in the second document. The reason for this change in approach is based on field evidence supporting the use of vaccination in as a tool to achieve eradication in Densely Populated Poultry Areas (DPPA). The 2003 report addresses also the issue of diagnosis of AI and highlights the areas in which information is lacking.

Between the end of 2003 and the beginning of 2004, evidence of extensive circulation of the highly pathogenic H5N1 virus present in Asia was made available. In addition to this, fatal human cases were reported. The awareness of the endemicity of the H5N1 virus in the Asian avian population coupled with the concerns for the generation of a new pandemic virus for humans has generated responses from international organisations including concern on scientific issues.

Although AI has now become one of the leading emerging infectious diseases in veterinary and human health, the generation of scientific data to support decision makers and risk assessors requires time and resources. For this reason some of the issues that should find a clarification in the present report remain only partly addressed or unanswered.

It is likely that the international effort that is being carried out in the medical and veterinary scientific communities in analysing data from the Italian, Dutch, Canadian and Asian outbreaks will generate significant amounts of data in the short-medium term which will broaden our current knowledge on AI.

For the sake of clarity, the report has been subdivided into three parts: Part I will address biological factors, risks of introduction of AI via wild birds and importation of live poultry, birds and avian products, Part II will focus on prevention and control options, biosecurity, vaccination and virus persistence and Part III will deal with animal welfare aspects and culling methods.
PART I
Biological factors, risks of introduction of AI via wild birds and importation of live poultry, birds and avian products

5 BIOLOGICAL FACTORS

5.1 VIRUS CHARACTERISTICS

5.1.1 AETIOLOGY
Influenza viruses are segmented, negative strand RNA viruses that are placed in the family Orthomyxoviridae in three genera: Influenzavirus A, B and C. Only influenza A viruses have been reported to cause natural infections of birds. Type A influenza viruses are further divided into subtypes based on the antigenic relationships in the surface glycoproteins haemagglutinin (H) and neuraminidase (N). At present 16 H subtypes have been recognised (H1-H16) and nine neuraminidase subtypes (N1-N9). Each virus has one H and one N antigen, apparently in any combination; all subtypes and the majority of possible combinations have been isolated from avian species.

5.1.2 PATHOTYPES

5.1.2.1 HPAI and LPAI
Influenza A viruses infecting poultry can be divided into two distinct groups on the basis of the severity of the disease they cause. The very virulent viruses cause HPAI in which flock mortality in some susceptible species may be as high as 100%. These viruses have been restricted to subtypes H5 and H7, although not all viruses of these subtypes cause HPAI. There have been 24 reported primary isolates of HPAI viruses from domestic poultry since 1959 (Table 5.1.) All other viruses cause a much milder disease consisting primarily of mild respiratory disease, depression and egg production problems in laying birds. Sometimes other infections or environmental conditions may cause exacerbation of influenza infections leading to much more serious disease. For example, in outbreaks of LPAI in Italy in 1999, high mortality was often recorded in young turkeys, reaching 97% in one flock (Capua et al., 2000a).

5.1.2.2 Molecular basis of virulence
The haemagglutinin glycoprotein for influenza viruses has two important functions that are imperative for the infectivity of the virus. First it brings about attachment to host cell and then fusion between the host cell membrane and the virus membrane so that the viral genetic material is introduced into the host cell. This glycoprotein is produced as a precursor, HA0, which requires post translational cleavage by host proteases before it is able to induce membrane fusion and virus particles become infectious (Rott, 1992). The HA0 precursor proteins of LPAI viruses have a single arginine at the cleavage site and another at position -3 or -4. These viruses are limited to cleavage only by certain host proteases such as trypsin-like enzymes and are thus restricted to replication at sites in the host where such enzymes are found, i.e. the respiratory and intestinal tracts. The HA0 proteins of HPAI viruses possess multiple basic amino acids [arginine and lysine] at their HA0 cleavage sites either as a result of apparent insertion or apparent substitution (Vey et al., 1992, Wood et al., 1993, Senne et al., 1996) and appear to be cleavable by a ubiquitous protease[s], probably one or more proprotein-processing subtilisin-related endoproteases of which furin is the leading candidate
(Stieneke-Grober et al., 1992). These viruses are able to replicate throughout the bird, damaging vital organs and tissues which results in disease and death (Rott, 1992). For example, all H7 subtype LPAI viruses have the amino acid motif at the HA0 cleavage site of either -PEIPKGR*GLF- or -PENPKGR*GLF-, whereas examples of cleavage site amino acid motifs for HPAI H7 viruses are: -PEIPKKKKR*GLF-, PETPKRRKR*GLF-, -PEIPKKREKR*GLF-, -PETPKRRRR*GLF-, -PEIPKGSVRR*GLF-.

Although 23 of the HPAI viruses in Table 2.1 have multiple basic amino acid motifs, as do all HPAI viruses sequenced that were isolated prior to 1959, this is not true of the viruses isolated from the HPAI outbreaks in Chile in 2002. The H7N3 viruses isolated in these outbreaks had motifs with insertion of 11 amino acids but without the apparent minimum requirement of basic amino acids, as their sequences were either PEKPKTCPSLSCRETR*GLF (4372) or PEKPKTCPSLSCRKTR*GLF (4957) (Suarez et al., 2004).

Current theories suggest that AI subtype H5 and H7 viruses of high virulence emerge from viruses of low virulence by mutation (Garcia et al., 1996, Perdue et al., 1998) although there must be more than one mechanism by which this occurs. This is supported by phylogenetic studies of H7 subtype viruses, which indicate that HPAI viruses do not constitute a separate phylogenetic lineage or lineages, but appear to arise from non-pathogenic strains (Rohm et al., 1995; Banks et al., 2000a) and the in vitro selection of mutants virulent for chickens from an avirulent H7 virus (Li et al., 1990). It appears that such mutations occur only after the viruses have moved from their natural wild bird host to poultry. However, the mutation to virulence is unpredictable and may occur very soon after introduction to poultry, as in the case of outbreaks 1-4, 6, 8-12, 14, 15, 17, 19, 20 and 22 in Table 2.1, or after the LPAI virus has circulated for several months, as in the case of outbreaks 7, 13, 16 and 18. This hypothesis is further strongly supported by a recent study of Munster et al. (2005) who have demonstrated that there is minor genetic and antigenic diversity between H5 and H7 LPAI viruses found in wild birds and those having caused HPAI outbreaks in domestic poultry in Europe. The virus responsible for the Chile isolate apparently arose as the result of mutation by a different mechanism than other HPAI viruses since studies have shown that the 11 amino acid insertion occurred by recombination that introduced a section of the NP gene into the HA gene (Suarez et al., 2004).

5.1.2.3 Clinical Signs

Low Pathogenic Avian Influenza

The severity of the disease produced by viruses inducing little or no disease in chickens infected experimentally and without multiple basic amino acids at the HA0 cleavage site (LPAI viruses) is greatly influenced by: the strain of 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: Reimerella spp, Newcastle disease viruses (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).

At one extreme the disease seen may be inapparent or slight. For example, Alexander and Spackman (1981) reported that an LPAI infection in a turkey laying flock resulted in only transient mild respiratory signs and 2% white-shelled eggs. Other LPAI outbreaks occurring in turkeys at about the same time produced 20-40% egg production drops and respiratory disease with low but significant mortality.
At the other extreme infections with LPAI viruses may be associated with severe disease and with high mortality. In outbreaks in chickens in Alabama in 1975 with a LPAI virus of H4N8 subtype up to 69% mortality was recorded in infected flocks (Johnson et al., 1977). In 1995 major outbreaks caused by LPAI viruses of H7N3 subtype affected turkeys in Utah USA and was associated with significant mortality especially in young birds, with about 40% mortality in 0- to 4-week-old birds (Halvorson et al., 1998). In most cases mortality was associated with dual infections with *Escherichia coli* or *Pasteurella multocida*. During the LPAI H7N1 infections in Italy in 1999 turkeys were particularly affected. In turkeys reared for meat the severity of the clinical and post mortem disease varied considerably, clinical signs were dominated by respiratory distress with mortality ranging from 5% to 97% depending on the age of the affected birds (Capua et al., 2000a). In young meat birds the signs were usually sufficiently severe to result in 40-97% mortality in infected flocks. In turkey breeders a milder form of the same clinical condition was observed that consisted of rales, coughing, swelling of the infraorbital sinuses and a febrile condition associated with loss of appetite. Egg production dropped by 30% to 80% during the acute phase, but partially recovered to subnormal levels within three weeks from the onset of the disease. Mortality rates ranged from 5 to 20% (Capua et al., 2000a). Equally serious problems have been reported in recent years associated with widespread outbreaks of viruses of H9N2 subtype particularly in Pakistan and Iran, but also in the Middle East and Asian countries through to China.

**Highly Pathogenic Avian Influenza**

Often the first sign of HPAI in flocks of chickens or turkeys, especially birds not in cages, is the sudden onset of high mortality, which may approach 100% within a few days. Clinical signs that may be associated with high mortality are: cessation of egg laying, respiratory signs, rales, excessive lachrymation, sinusitis, oedema of the head and face, subcutaneous haemorrhage with cyanosis of the skin, particularly of the head and wattles, and diarrhoea, occasionally neurological signs may be present. Usually, these signs are most marked in birds that take some time to die. Death generally occurs within 12-48 hours following the onset of clinical signs.

In other species, clinical signs are not as clear-cut as in chickens and turkeys. HPAI in ducks and geese was thought to be asymptomatic, however in some instances clinical signs and mortality can be observed (Capua and Mutinelli, 2001a). Mortality of Muscovy ducks (*Cairina moschata*) and domestic geese (*Anser anser var.domestica*) following natural infection with highly pathogenic avian influenza of the H7N1 subtype (Capua and Mutinelli, 2001b; Sturm-Ramirez et al., 2004). In other birds such as ostriches and quail mortality rates vary, but generally do not reach 100% (Capua and Mutinelli, 2001a). For this reason, in birds other than chickens and turkeys HPAI may be misdiagnosed, leading to a delay in the notification to relevant authorities.

### 5.1.2.4 Definition of avian influenza

The marked variation in disease caused by LPAI and HPAI viruses of the same subtype and the fact that, to date, two subtypes H5 and H7 have been shown to be responsible for HPAI means that careful, specific definition is required for statutory control and trade purposes.

Current EU legislation for statutory control purposes (EC, 1992a) defines AI as `an infection of poultry caused by any influenza A virus that has an intravenous pathogenicity index in 6-week-old chickens greater than 1.2 or any infection with influenza A viruses of H5 or H7 subtype for which nucleotide sequencing has demonstrated the presence of multiple basic amino acids at the cleavage site of the haemagglutinin’. However on the basis of the evidence that HPAI viruses emerge in domestic poultry from LPAI progenitors of the H5 and H7 subtypes there is a case that not only HPAI viruses but also their LPAI progenitors should be
controlled in domestic poultry (Capua and Marangon, 2000; Alexander, 2003). As a result the European Union Scientific Committee on Animal Health and Animal Welfare put forward a proposal for a new definition (SCAHAW, 2000) which is: ‘an infection of poultry caused by either any influenza A virus that has an intravenous pathogenicity index in 6-week-old chickens greater than 1.2 or any influenza A virus of H5 or H7 subtype’. A very similar definition has recently been adopted by the World Organisation for Animal Health (OIE) during its 73rd General Session (OIE, 2005a).

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

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

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

Trade requirements now also apply for LPAI of H5 and H7 subtypes (OIE, 2005a). However, these differ between LPAI and HPAI and are recommended proportionate to the risks posed by the various commodities. Linked to the application of these new requirements guidelines for AI surveillance (OIE, 2005d) and for the implementation of the concept of “compartmentalisation” have now been included in the Terrestrial Code for 2005 (OIE, 2005a).

A proposal (EC, 2005a)¹ for a revised definition and control measures for avian influenza has been adopted by the European Commission on 28 April 2005. It takes into account lessons learned during the experiences gained with AI outbreaks in the last years and is now under discussion in working groups at the Council and European Parliament with the aim of its adoption before the end of 2005.

Table 5-1 Primary HPAI virus isolates from poultry\(^1\) since 1959

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Year</th>
<th>HN</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A/chicken/Scotland/59 (H5N1)</td>
<td>1959</td>
<td>H5N1</td>
</tr>
<tr>
<td>2</td>
<td>A/turkey/England/63 (H7N3)</td>
<td>1963</td>
<td>H7N3</td>
</tr>
<tr>
<td>3</td>
<td>A/turkey/Ontario/7732/66 (H5N9)</td>
<td>1977</td>
<td>H5N9</td>
</tr>
<tr>
<td>4</td>
<td>A/chicken/Victoria/76 (H7N7)</td>
<td>1976</td>
<td>H7N7</td>
</tr>
<tr>
<td>5</td>
<td>A/chicken/Germany/79 (H7N7)</td>
<td>1979</td>
<td>H7N7</td>
</tr>
<tr>
<td>6</td>
<td>A/turkey/England/199/79 (H7N7)</td>
<td>1979</td>
<td>H7N7</td>
</tr>
<tr>
<td>7</td>
<td>A/chicken/Pennsylvania/1370/83 (H5N2)</td>
<td>1983</td>
<td>H5N2</td>
</tr>
<tr>
<td>8</td>
<td>A/turkey/Ireland/1378/83 (H5N8)</td>
<td>1983</td>
<td>H5N8</td>
</tr>
<tr>
<td>9</td>
<td>A/chicken/Victoria/85 (H7N7)</td>
<td>1985</td>
<td>H7N7</td>
</tr>
<tr>
<td>10</td>
<td>A/turkey/England/50-92/91 (H5N1)</td>
<td>1991</td>
<td>H5N1</td>
</tr>
<tr>
<td>11</td>
<td>A/chicken/Victoria/1/92 (H7N3)</td>
<td>1992</td>
<td>H7N3</td>
</tr>
<tr>
<td>12</td>
<td>A/chicken/Queensland/667-6/94 (H7N3)</td>
<td>1994</td>
<td>H7N3</td>
</tr>
<tr>
<td>13</td>
<td>A/chicken/Mexico/8623-607/94 (H5N2)</td>
<td>1994</td>
<td>H5N2</td>
</tr>
<tr>
<td>14</td>
<td>A/chicken/Pakistan/447/94 (H7N3)</td>
<td>1994</td>
<td>H7N3</td>
</tr>
<tr>
<td>15</td>
<td>A/chicken/NSW/97 (H7N4)</td>
<td>1997</td>
<td>H7N4</td>
</tr>
<tr>
<td>16</td>
<td>A/chicken/Hong Kong/97 (H5N1)</td>
<td>1997</td>
<td>H5N1</td>
</tr>
<tr>
<td>17</td>
<td>A/chicken/Italy/330/97 (H5N2)</td>
<td>1997</td>
<td>H5N2</td>
</tr>
<tr>
<td>18</td>
<td>A/turkey/Italy/99 (H7N1)</td>
<td>1999</td>
<td>H7N1</td>
</tr>
<tr>
<td>19</td>
<td>A/chicken/Chile/2002 (H7N3)</td>
<td>2002</td>
<td>H7N3</td>
</tr>
<tr>
<td>20</td>
<td>A/chicken/The Netherlands/2003 (H7N7)</td>
<td>2003</td>
<td>H7N7</td>
</tr>
<tr>
<td>21</td>
<td>A/chicken/East Asia/2003-2005 (H5N1)</td>
<td>2005</td>
<td>H5N1</td>
</tr>
<tr>
<td>22</td>
<td>A/chicken/Canada-BC/2004 (H7N3)</td>
<td>2004</td>
<td>H7N3</td>
</tr>
<tr>
<td>23</td>
<td>A/chicken/USA-TX/2004 (H5N2)</td>
<td>2004</td>
<td>H5N2</td>
</tr>
<tr>
<td>24</td>
<td>A/ostrich/S. Africa/2004 (H5N2)</td>
<td>2004</td>
<td>H5N2</td>
</tr>
</tbody>
</table>

\(^1\)Where outbreaks were widespread and affecting more than one species, the isolate from the first outbreak identified is listed.

\(^2\)Cambodia, China, Indonesia, Japan, Lao PDR, Malaysia, Republic of Korea, Thailand and Viet Nam reported disease in this period; the relationship of these viruses to A/Hong Kong/97 (H5N1) remains unclear at present.

\(^3\)This virus did not kill chickens infected experimentally, but had multiple basic amino acids at the HA0 cleavage site.

5.2 HOSTS

5.2.1 AVIAN HOSTS

The prevalence of AI viruses in different avian hosts and their susceptibility has been the subject of several reviews (e.g. Alexander, 2000; Alexander, 2001) and will be dealt with in detail in chapter 6 of this report. Influenza viruses have been isolated from avian species
representing most of the major Families of birds. It seems likely that the viruses are perpetuated in free-living birds, particularly migratory waterfowl (Hinshaw et al., 1980a). Surveillance studies have indicated active infection rates in migratory ducks, especially mallards (Anas platyrhyncos) as approaching 20% (see section 6.1), although this depends on temporal and geographical factors relating to migration. Isolation rates from other wild birds such as passerines, gulls and shore birds have been between 0-3% in surveillance studies.

Studies by Sharp et al., (1993), suggest that waterfowl do not act as a reservoir for all avian influenza viruses. It seems likely that part of the influenza gene pool is maintained in shorebirds and gulls, from which the predominant number of isolated influenza viruses are of a different subtype to those isolated from ducks (Kawaoka et al., 1988).

Until the recent H5N1 epidemic in Asia, it was thought that wild waterfowl could harbour both LPAI and HPAI strains without exhibiting any clinical signs. To current knowledge, wild waterfowl do not exhibit any clinical sign with LPAI viruses, but may develop clinical disease and eventually die of HPAI infection (Chen et al., 2005; Liu et al., 2005).

It would appear that host range may vary with the strain of virus.

5.2.1.1 Commercial ducks

The role of commercial ducks in infections, maintenance and spread of AI viruses requires separate consideration.

The influenza status of commercial ducks in most countries is poorly understood or has not been investigated. When surveillance of commercial ducks has been undertaken, enormous pools of LPAI virus and many subtype combinations have been detected, especially from meat birds, which are usually fattened on open fields. For examples, (Alexander and Stuart, 1982) reported the isolation of 32 LPAI viruses of several different subtypes from 60 pools each of 10 cloacal swabs taken from ducks at slaughter in England; studies in Hong Kong in the late 1970s and early 1980s on carcases at duck dressing plants or on duck farms indicated about 6% of the ducks were infected with AI viruses of various subtypes (Shortridge, 1982). In 1997 H5 viruses were isolated from 2.5% of ducks sampled in Hong Kong (Shortridge, 1999) in contrast to an H5 virus isolation rate from ducks of 0.25% five years earlier. In 1997 H9 viruses were also isolated from 0.9% of ducks compared to 0.19% five years earlier. On many duck farms, where birds are raised outdoors, the continual presence of influenza viruses seems most likely to be due to the repeated introduction of susceptible ducklings to fields where virus is already present in infective faeces or water, and influenza viruses may be considered enzootic in some commercial duck flocks, particularly fattening ducks. However, Shortridge (1982) and Sandhu and Hinshaw (1982) reported considerable variation in the subtypes present in commercial flocks and it is probable that fresh introductions by wild birds also occur regularly.

Commercial ducks are also of potential significance in the spread and maintenance of HPAI viruses. There is no evidence that HPAI viruses have emerged by mutation from LPAI virus in commercial ducks. However, experimentally ducks are readily infected with HPAI viruses, but usually show no clinical signs following such infections (Slemons and Easterday, 1972; Alexander et al. 1978; Westbury et al., 1979; Alexander et al.,1986; Wood et al., 1985; Perkins and Swayne, 2002; Chen et al., 2004). However, it is possible that some HPAI viruses may cause disease and deaths in ducks, for example (Alexander et al. 1978) reported that in experimental infections with A/chicken/Germany/34 (H7N1) HPAI virus 9/10 two-week-old ducks infected intranasally showed clinical signs and two died and 6/7 two-week-old ducks placed in contact with these showed clinical signs and 3 of this group died. In similar experiments with two other H7 HPAI viruses these authors recorded no clinical signs. Sturm-
Ramirez et al. (2004) in infection studies in mallards with H5N1 viruses (1997-2003) of high genetic homology showed that in contrast to those from 1997 and 2001 viruses isolated in 2002 caused: acute disease, systemic infection, pathology in multiple organs, high virus titres, neurological dysfunction and death. In addition there was efficient transmission between ducks. However, in the field, disease and death in commercial ducks has not been a feature of the ongoing HPAI H5N1 infections in East Asia.

Despite the prevalence of LPAI AI virus infections in commercial ducks, until the recent South East Asian outbreaks there was only one report of a significant commercial duck flock infected with HPAI virus, this was of H5N8 subtype in Ireland (Alexander et al., 1987). In November 1983 a highly pathogenic virus of H5N8 subtype was obtained from turkeys on three farms (Murphy, 1986; 1987). A further 44 poultry farms within a five kilometre radius were monitored with no evidence of disease. However, early in 1984 viruses of H5N8 and H3N2 subtypes were isolated from apparently healthy birds on a large commercial duck farm situated between the first and second turkey farm to be affected, the 250,000 ducks were slaughtered. This outbreak highlights the greatest concern of HPAI infections in commercial ducks, that the virus will enter and circulate amongst the commercial duck population going unnoticed or ignored because of the absence of clinical signs and represent a pool of endemic virus capable of spread to susceptible hosts, including humans. There is some evidence that this is the current situation in some countries in South East Asia.

5.2.2 AVIAN INFLUENZA INFECTIONS OF MAMMALS

5.2.2.1 Pigs

Kida et al., (1994) demonstrated experimentally that pigs were susceptible to infection by at least one virus representative of each of the subtypes H1-H13.

The introduction of classical swine H1N1 influenza viruses to turkeys from infected pigs has been reported to occur regularly in the USA, and in some cases, influenza-like illness in pigs has been followed immediately by disease signs in turkeys (Halvorson et al., 1992; Mohan et al., 1981; Pomeroy; 1982). Genetic studies of H1N1 viruses from turkeys in the USA has revealed a high degree of genetic exchange and reassortment of influenza A viruses from turkeys and pigs in the former species (Wright et al., 1992). In Europe, avian H1N1 viruses were transmitted to pigs, became established, and were subsequently reintroduced to turkeys from pigs (Ludwig et al., 1994; Wood et al., 1997). An independent introduction of H1N1 virus from birds to pigs occurred in Europe in 1979 (Pensaert et al., 1981) A similar introduction occurred in Asia in the early 1990s; and these latter viruses are genetically distinct from the viruses in Europe (Guan et al., 1996). H9N2 viruses were introduced into pigs in South-East Asia (Peiris et al., 2001). Serological evidence has been obtained of infections of pigs with viruses of H4, H5 and H9 subtypes (Ninomiya et al., 2002). During the HPAI H7N7 epidemic in The Netherlands in 2003 13 pig herds on farms with infected poultry were shown to have antibodies to H7 subtype, though no virus was detected (Loeffen et al., 2003, 2004). In Canada, however, avian viruses of H3N3 and H4N6 subtypes were isolated from pigs (Karasin et al., 2000, 2004). Clearly the introduction of avian influenza viruses to pigs is not an uncommon occurrence. But despite this, the only subtypes to have become truly established in pig populations are H1N1, H3N2, and the reassortant H1N2, although genotype analysis of isolates of these subtypes suggests that they can be the result or reassortment of viruses from different progenitor host species (pig, human and avian).
5.2.2.2 Horses

Although there have been isolated reports of evidence of infection of horses with viruses of subtypes H1N1, H2N2 and H3N2 (Tumova, 1980), influenza infections of horses have been restricted essentially to H7N7 and H3N8 subtypes of influenza A and these viruses form distinct lineages in phylogenetic studies. However, examination of H3N8 viruses isolated from severe epidemics in horses occurring in the Jilin and Heilongjiang Provinces in the north-east of the People’s Republic of China in 1989 and 1990, showed these to be antigenically and genetically distinguishable from other equine H3N8 viruses and Guo et al. (1992) concluded that this virus was of recent avian origin and had probably spread directly to horses without reassortment. This virus does not appear to have become established in the horse population.

5.2.2.3 Marine mammals

During 1979 and 1980, approximately 500 deaths [about 20% of the population] occurred in harbour seals (Phoca vitulina) around the Cape Cod Peninsula in the USA as a result of acute haemorrhagic pneumonia. Influenza A viruses of H7N7 subtype were isolated repeatedly from the lungs or brains of dead seals (Lang et al., 1981). The virus infecting the seals was shown to be closely related both antigenically and genetically to avian influenza viruses (Webster et al., 1981a) and appeared to represent direct transmission to the seals without reassortment.

In 1983, further deaths (2%-4%) occurred in harbour seals on the New England coast of the USA and an influenza A virus of subtype H4N5 was isolated. Once again, all eight genes of this virus were demonstrably of avian origin (Webster et al., 1992). Following surveillance of seals on the Cape Cod peninsula Callan et al. (1995) reported isolates of two influenza A viruses of H4N6 subtype made in 1991 and three of H3N3 subtype in 1992 all from seals found dead with apparent viral pneumonia. Antigenic and genetic characterisation, revealed these too were avian viruses that had entered the seal population.

Two viruses of H13N2 and H13N9 subtypes were isolated from a single beached pilot whale, and genetic analysis indicated that the viruses had been introduced recently from birds (Chambers et al., 1989, Hinshaw et al., 1986).

5.2.2.4 Mink

In October 1984, outbreaks of respiratory disease affected approximately 100,000 mink on 33 farms situated in close proximity along a coastal region of southern Sweden, with 100% morbidity and 3% mortality (Klingeborn, et al., 1985). Influenza A viruses of H10N4 subtype were isolated from the mink and genetic analysis indicated that the viruses were of avian origin and were very closely related to a virus of the same subtype isolated from chickens and a feral duck in England in 1985 (Berg et al., 1990). Earlier experimental infections had suggested that mink were susceptible to infection with various subtypes of avian influenza viruses (Okazaki et al., 1983).

5.2.2.5 Cats/Felidae

Studies of Hinshaw et al. (1981) have shown the ability of LPAI virus as to infect and replicate in cats without showing clinical signs. However, during the 2003 – 2004 HPAI H5N1 outbreak in Asia, there have been occasional reports of fatal H5N1 virus infections in domestic cats and zoo felids after they had been fed virus-infected chickens (ProMED, Mail 2004). In experimental studies, cats excreted virus and developed lung pathology after intratracheal inoculation with H5N1 or after feeding on H5N1 virus-infected chickens (Kuiken et al. 2004). In addition, the virus was transmitted from the infected to sentinel cats.
Thus, cats may become infected with AI after consumption of fresh poultry meat and they may spread the virus to other cats.

5.2.2.6 Humans

Although it has been known for sometime that the human pandemic viruses of 1957 and 1968 appeared to arise by reassortment between viruses present in the human population and avian influenza viruses (Gething, et al., 1980; Kawaoka, et al., 1989; Scholtissek, et al., 1978), because of the apparent “barriers” to human influenza viruses infecting birds and avian influenza viruses infecting humans it was suggested that pigs, which both human and avian viruses are known to infect readily, acted as “mixing vessels” in which reassortment between human and avian influenza viruses could take place with the emergence of viruses with the necessary gene(s) from the virus of human origin to allow replication and spread in the human population, but with a different haemagglutinin surface glycoprotein, so that the human population could be regarded as immunologically naive.

However, there has been a significant change in our understanding of infections of humans with avian influenza viruses as indicated by the now known reported infections of humans with avian influenza viruses, which are summarised in Table 5.2. As indicated, until 1996 there had been only three reported infections and these had been the result of unknown contact, in 1959, and two laboratory accidents in 1977 and 1981 (with the seal isolate). This was in keeping with the findings of Bear and Webster (1991) that in experiments human volunteers produced at best only transitory infections when challenged with avian influenza viruses. The first reported infection of a human known to have contact with birds was the isolation of an avian virus of H7N7 from a woman in England who kept ducks and presented with conjunctivitis (Kurtz, et al., 1996, Banks et al., 1998). This was the vanguard of the series of isolations from people having contact with poultry shown in Table 5.2. The impact these subsequent human infections on public health issues was greatly enhanced by the high death rate in those shown to be infected. These deaths usually occurred as a result of severe respiratory disease and although there were other symptoms there was no evidence that virus replicated outside the respiratory tract (Yuen et al., 1998) and were not comparable to the systemic infections seen in poultry.

The biggest threat of the demonstration of direct natural infections of humans with avian influenza viruses is that pandemic viruses could emerge as a result without an intermediate host. The main danger appears to be not directly the viruses that have spread from avian species, but if the people infected with the avian influenza viruses had been infected simultaneously with a “human” influenza virus, reassortment could have occurred with the potential emergence of a virus fully capable of spread in the human population, but with H5, H7 or H9 haemagglutinin, resulting in a true influenza pandemic. However, it seems likely that during the widespread outbreaks of H9N2 virus since the mid-1990s and the H5N1 outbreaks in Asia since 1996 many more people than those listed in Table 5.2 would have been infected with these viruses. For example, a serological survey of poultry workers in Hong Kong after the 1997 outbreak identified 10% seroprevalence of H5 antibodies, but without any known occurrence of clinical disease (Buxton Bridges et al., 2002). In relation to serological investigations in humans during the Dutch 2003 H7N7 epidemic which also caused one human fatality see also section 7.4. Despite this no reassortant virus has emerged and it may well be that other, unknown, factors limit the chances of a pandemic virus arising in this way.
### Table 5-2 Reports of human infections with avian influenza viruses (Updated to 19 May 2005)

<table>
<thead>
<tr>
<th>Year</th>
<th>Subtype</th>
<th>HPAI/LPAI¹</th>
<th>Country</th>
<th>Number infected</th>
<th>Symptoms</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1959</td>
<td>H7N7</td>
<td>HPAI</td>
<td>USA</td>
<td>1</td>
<td>hepatitis?</td>
<td>Campbell et al., (1970)</td>
</tr>
<tr>
<td>1977</td>
<td>H7N7</td>
<td>HPAI</td>
<td>Australia</td>
<td>1</td>
<td>conjunctivitis</td>
<td>Taylor and Turner, (1977)</td>
</tr>
<tr>
<td>1981</td>
<td>H7N7</td>
<td>LPAI</td>
<td>USA</td>
<td>1</td>
<td>conjunctivitis</td>
<td>Webster et al., (1981b)</td>
</tr>
<tr>
<td>1997</td>
<td>H5N1</td>
<td>HPAI</td>
<td>Hong Kong</td>
<td>18</td>
<td>influenza-like illness</td>
<td>Shortridge et al., (2000)</td>
</tr>
<tr>
<td>1999/98</td>
<td>H9N2</td>
<td>LPAI</td>
<td>Hong Kong/China</td>
<td>2 (+5?)</td>
<td>influenza-like illness</td>
<td>Peiris et al., (1999)</td>
</tr>
<tr>
<td>2003</td>
<td>H5N1</td>
<td>?</td>
<td>Hong Kong</td>
<td>2 (+1?)</td>
<td>influenza-like illness, 1 (+1?) death</td>
<td>WHO website:²</td>
</tr>
<tr>
<td>2003</td>
<td>H7N7</td>
<td>HPAI</td>
<td>The Netherlands</td>
<td>83</td>
<td>conjunctivitis, some cases of influenza-like illness, 1 death</td>
<td>Koopmans et al., (2004)</td>
</tr>
<tr>
<td>2004 -</td>
<td>H5N1</td>
<td>HPAI</td>
<td>Thailand</td>
<td>17</td>
<td>influenza-like illness</td>
<td>WHO website²</td>
</tr>
<tr>
<td>ongoing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2004 -</td>
<td>H5N1</td>
<td>HPAI</td>
<td>Viet Nam</td>
<td>76</td>
<td>influenza-like illness</td>
<td>WHO website²</td>
</tr>
<tr>
<td>ongoing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2004</td>
<td>H7N3</td>
<td>HPAI</td>
<td>Canada [BC]</td>
<td>2</td>
<td>conjunctivitis</td>
<td>Health Canada website³</td>
</tr>
<tr>
<td>2005-</td>
<td>H5N1</td>
<td>HPAI</td>
<td>Cambodia</td>
<td>4</td>
<td>influenza-like illness</td>
<td></td>
</tr>
<tr>
<td>ongoing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6 RISK OF INTRODUCTION OF AI INTO EU POULTRY HOLDINGS

For the sake of clarity of the present chapter dealing with classification of risks (hazard identification) the following terminology will be used (OIE, 2004b):

Table 6-1-1: Definitions for the purpose of risk assessment

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

6.1 RISK OF INTRODUCTION OF AI BY WILD BIRDS

6.1.1 PERIODS AT RISK IN EUROPE ACCORDING TO GEOGRAPHICAL CONSIDERATIONS

As seen in the summary in table 13-1 of Annex I, only very few investigations of AIV in wild migratory birds in Europe, and elsewhere, have been performed in a longitudinal and standardised manner. Therefore it is important to notice that the body of background knowledge is fragmentary. However, although it does appear that mallards may play a significant role, it is noteworthy to mention that all dabbling ducks are genetically closely related and have a behaviour of interspecies mixing during migration and wintering. Furthermore, male dabbling ducks often show a non-philopatric behaviour e.g. a bird may be born in the Russian tundra but can the next year breed in virtually any European country. Therefore, to identify with certainty certain “risk
species” or state that the main risk period for transmission of AIV from waterfowl to domestic birds is during the autumn migration may be premature and hazardous. The temporal pattern with higher prevalence in early migratory birds compared to wintering or spring migrating birds can indicate that the period at risk is in the early phase of the autumn migration. On the other hand, even if the prevalence of AIV in wintering birds may be low, the congregations of huge numbers of wild birds in proximity to domestic poultry farm in their winter quarter may be a substantial risk. More research on this topic is needed.

Recently, however surveillance in wild birds has increased in the EU. One particular study conducted in Italy, is based on virus isolation attempts from wild birds, domestic waterfowl and game birds in northern Italy, yielded 9 H7 viruses between 2003 and the beginning of 2005. Seven of these were isolated from wild birds (mallards and teals) and two from domestic waterfowl in backyard flocks. Preliminary sequence analysis of the H7 gene showed a 99.3 homology at the nucleotide level between the isolates from the backyard flocks and the isolates obtained from the wild birds (Terregino et al., 2005). This preliminary data could support the theory that backyard poultry may represent one of the links that connect the AI wild bird population to poultry. For this reason it is imperative that the further transmission step (from backyard to intensively reared poultry) is avoided. This can be achieved through the implementation of rigorous biosecurity measures.

6.1.2 ROLE OF BACKYARD/HOBBY FLOCKS IN THE EPIDEMIOLOGY OF AI

As mentioned above, backyard or hobby flocks reared in the open may represent the contact point from the wild reservoir to the domestic host. However, field evidence gained during major outbreaks in Europe suggests that they play a negligible role in the spread of infection. This is most probably due to the fact that basic biosecurity measures require a complete separation of the industrial circuit from the backyard circuit. Since the major risk of spread from a backyard to an industrial farm is linked to mechanical transfer of the virus by human beings or equipment carried by human beings it is a well-respected general rule that farm staff must not have any contact with birds at risk of infection, such as backyard or hobby flocks. For this reason, the risk of hobby or backyard flocks that are situated within the restriction zones to be infected with AI is negligible. Thus, mass culling of backyard or hobby flocks situated within the restriction zones is unnecessary, although education of the owners on prevention and on the clinical signs of AI and surveillance are essential to avoid that any outbreaks remain undetected.

6.1.3 ROLE OF FREE-RANGE FARMS

In recent years, there has been a significant trend to develop outdoor systems for farming chickens, including free-range establishments. This type of farming, which has been primarily developed in the layer industry, enables birds to be able to wander in the shed and also outside, in a fenced outdoor paddock. Often, wild birds, invited by open water, food, presence of domestic ducks and geese or other attractions may fly into the paddock and mingle with the farmed birds and possibly deposit droppings on the ground which may represent a source of infection. Thus possibility of having access to outdoor premises, increases the risk of having contacts with wild birds that are known to carry
several pathogens, including avian influenza viruses, and this should be kept in mind when developing biosecurity programmes for this type of farming.

6.2 RISKS OF AI INTRODUCTION BY THE IMPORTATION OF LIVE POULTRY

6.2.1 EU IMPORT LEGISLATION

A comprehensive set of rules exists in the EU detailing the requirements to be met by third countries in order to export live poultry, birds other than poultry and products of avian origin to the EU. Information on import requirements is available on the website of the European Commission and in particular in a document produced by the Food and Veterinary Office (FVO) which is conceived as a guidance for third country authorities when applying for access to the European market (EC, 2003a).

6.2.2 THIRD COUNTRY APPROVAL FOR IMPORTATION INTO THE EU

Live poultry including hatching eggs can only be imported from a rather short list of approved third countries, currently 11 countries or parts thereof: Australia, Brazil (some regions), Bulgaria, Canada, Chile, Croatia, Israel, New Zealand, Romania Switzerland and the United States of America (EC, 1996). In addition live ratites (i.e. ostriches and emus) for breeding and production and their hatching eggs can also be imported from Botswana, Namibia, South Africa and Tunisia (EC, 2001a).

Third country EC approval is dependent on the animal health status of the country including the one of its neighbouring countries, the capabilities of the veterinary services and diagnostic laboratories and their effectiveness to prevent and control certain infectious or contagious diseases, the third country’s own disease control measures and its import policy. The FVO carries out on the spot missions to these countries before approval is given and after approval to inspect if the EU requirements are complied with. Inspection reports can be consulted on the internet.

A country's approved importer status approval may be suspended in the event of an outbreak of a specified disease by protection measures adopted at Community level. EC trade restrictions in relation to recent AI outbreaks in Asia, Canada, USA and South Africa and the chronology of events can be found on internet.

If the animal health situation in a third country has substantially changed by a disease has having become endemic or due to lack of credibility of the veterinary services and their certification a more definite action by “delisting” the country from the list of authorized countries will be considered.

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2  http://europa.eu.int/comm/food/animal/index_en.htm
3  http://europa.eu.int/comm/food/fvo/ir_search_en.cfm
6.2.3 REQUIREMENTS FOR THIRD COUNTRIES AS THEY APPLY SPECIFICALLY TO THE CONTROL OF HPAI

So far, EU disease control measures and international trade standards are focussed on HPAI and therefore no specific import requirements currently exist for LPAI such as e.g. compulsory surveillance for LPAI in the third country of origin or testing requirements after importation. Consignments of animals and animal products destined for the importation into the EU must be accompanied by health certification as laid down in EU legislation. In relation to HPAI live poultry and fresh poultry meat imports can only take place from a third country which:

a) is listed as an EC approved country/region for such importation
b) is free from HPAI for 12 months or for at least for 6 months after an outbreak of HPAI, when a stamping out policy has been applied
c) has not carried out vaccination against H5 or H7 subtypes within the last 12 months

The specific requirements for poultry holdings, breeding establishments and hatcheries from which live poultry and poultry products must originate and for the official veterinary inspections to be carried out in the country of origin are detailed in the relevant sections below.

6.2.4 SPECIFIC REQUIREMENTS FOR IMPORTS OF LIVE POULTRY INCLUDING FARmed FEATHERED GAME, AND RATITES

6.2.4.1 Definition for live poultry

According to EU legislation (EC,1990a) 'poultry' shall mean fowl, turkeys, guinea fowl, ducks, geese, quails, pigeons, pheasants and partridges and ratites (ratitae) reared or kept in captivity for breeding, the production of meat or eggs for consumption, or for restocking.

In the scientific reports SCAHAW (1998, 2000) the following definition not referring to specific poultry species has been recommended:

“Poultry is defined as ‘all birds reared or kept in captivity for the production of meat or eggs for consumption, for the production of other commercial products, for restocking supplies of game, or for breeding these categories of birds’. This definition has also been recently adopted by OIE in the new AI Terrestrial Code Chapter. (OIE, 2005a)

6.2.4.2 Definition for hatching eggs

‘Hatching eggs’ are defined as eggs for incubation laid by poultry as defined in paragraph 6.2.4.1. above. Animal health requirements for intra-Community trade and imports of hatching eggs are included in the rules laid down for live poultry i.e. they are

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5 Recently adopted OIE rules making trade in vaccinated birds and their products an option if linked to appropriate surveillance will apply as of 1/1/06. Therefore provided that the third country applies a vaccination programme that would meet these standards and give EU equivalent animal health guarantees, this requirement might be modified.
not considered as products, but as ‘live poultry’ and therefore more stringent requirements than for table eggs apply.

6.2.5 PRE-IMPORT REQUIREMENTS FOR LIVE POULTRY AND HATCHING EGGS

Live poultry as defined in the above paragraphs are imported as hatching eggs, day-old chicks, breeding or productive poultry, slaughter poultry or as poultry for restocking game supplies (EC, 1996).

6.2.5.1 Requirements for establishments and certification for live poultry and hatching eggs

In addition to the requirements for the third country of origin as detailed in 6.2.2 with regard to its authorisation for importation and the country’s health status for HPAI, live poultry imports - here reference is made to breeding and productive poultry - must fulfil the following requirements:

a) they must have been kept in the country of origin for at least 3 months or since hatching if younger; or if imported they must have originated from a country with the same stringent requirements;

b) they must come from approved establishments, where they have been kept since hatching or at least for 6 weeks, and which
   - are not subject to any animal health restrictions
   - are not located within a radius of 25 km, where there has been an outbreak of HPAI (this refers to neighbouring countries, as the country itself or the approved region must be free of HPAI);

c) during the period indicated in b) they may not have been in contact with any other poultry that does not meet these requirements nor with wild birds;

d) they must be clinically inspected on the day of loading and show no clinical sign or suspicion of disease

e) they must be transported in crates or cages:
   - containing only poultry of the same species, category and type and coming from the same establishment
   - which are cleansed and disinfected and closed in a way to avoid any possibility of substitution
   - which must bear the approval number of the establishment of origin
   - which have been cleaned and disinfected and which are designed to preclude the loss of excrement and minimise the loss of feathers during transport and which allow visual inspection

6.2.6 APPROVAL OF LIVE POULTRY ESTABLISHMENTS FOR LIVE POULTRY AND HATCHERIES

Approval of establishments as indicated in b) above is given by the third country’s veterinary services on basis of compliance with detailed rules on lay-out, facilities and operation of these establishments, hygiene and disease surveillance programmes
including testing for Salmonella and Mycoplasma and on procedures for suspension and withdrawal of approval.

6.2.7 ANIMAL HEALTH CERTIFICATION

All requirements detailed in 6.2.5, including the indication of origin and destination, numbers of poultry, identification of packaging etc. are contained in the animal health certificate that accompanies the consignment during transport to the border inspection post and to the final destination within the EU. The official veterinarian issuing the certificate must verify and guarantee with his signature that all these requirements are met.

As stated above the listed requirements are fully applicable to breeding and productive poultry whereas some requirements differ with regard to the to the category of poultry; e.g. for slaughter poultry which is to be transported directly to an abattoir for subsequent slaughter and therefore not coming into contact with any live poultry kept on holdings within the EU, requirements for the establishments as regards specific approval and disease programme requirements are less stringent.

In the case of hatching eggs the animal health requirements as listed above refer to the poultry flocks from which they are derived and in addition they are marked individually with the approval number of the hatchery.

6.2.8 REQUIREMENTS FOR LIVE RATITES AND THEIR HATCHING EGGS

The requirements for the importation of live ratites and their hatching eggs are laid down in separate Community rules (EC, 2001a).

With respect to HPAI the animal health requirements are identical to the rules for other live poultry, however more stringent rules for pre-export quarantine and testing needed to be laid down in relation to the possible occurrence of Crimean Congo haemorrhagic fever.

6.2.9 SPECIFIC REQUIREMENTS FOR IMPORTS OF SINGLE CONSIGNMENTS OF LESS THAN 20 UNITS

For less than 20 heads of live poultry or less than 20 hatching eggs no harmonised EU import certificates are laid down. However, the third country from which Member States may authorise such importation must appear on a third country list established for other live animal species and importation has not been banned because of outbreaks of AI or Newcastle disease (1995, EC). The conditions of such imports must include a requirement for post-import isolation or quarantine and are not applicable to arties and hatching eggs thereof.

6.2.10 MEANS AND CONDITIONS OF TRANSPORT

As detailed in the animal health certificates transport of live poultry must be carried out in crates or cages and vehicles which must have been cleansed and disinfected before loading the consignment or in the case of hatching eggs in perfectly clean, disposable boxes which are used for the first time must.

Conversely to other animal species (EC, 1979) no detailed transit rules apply; however, the validity of the animal health certificates for live poultry and hatching eggs is limited to five days and transport over long distances mainly takes place as day-old poultry
which is usually performed by aircraft under closed conditions (according to temperature requirements), thus minimising the risk of contact to other consignments.

### 6.2.11 Veterinary checks on EU borders

Checking imports at border inspection posts (BIPs) is one of the main safeguards for ensuring that imports, both of live poultry and of avian products are in compliance with the required legal standards. Information on import requirements can be found on the EC website⁶. Both live animals and products of animal origin must undergo veterinary checks.

According to Council Directive 91/496/EEC (EC, 1991b) checks of live animal imports from third countries always include a “documentary check” of the accompanying certificates and an “identity check” to ensure that the animals are as described in the documentation, and may include a “physical check” to ensure the animals do not show any signs of poor health.

Again at this stage, the “physical checks” of live poultry cannot avoid the introduction of AIV infected poultry that do not exhibit clinical signs (infection with no visible signs during the incubation period). Whereas the later situation can be monitored thanks to the implemented controls after importation at the holding of destination (see 6.2.15). However, no measures are in force at this moment to avoid the introduction of LP and (even HP) AIV in poultry species that are clinically far less susceptible such as e.g. ducks.

For products of animal origin from third countries veterinary checks are carried out in accordance to Council Directive 97/78EEC (EC, 1997). Again, the three main types of documentary, identity, and physical veterinary checks are carried out. The documentary check is carried out on all consignments, to ensure all documentation is present and fully completed. The identity of all consignment is also verified by checking that the description on the consignment matches that on the documentation. In particular health marks identifying the country and establishment of origin must be present. For sealed containers, either an official seal is present, or the container is opened and checked as above. However, Community legislation foresees a reduction of the frequency physical checks for products of animal origin. Depending on the animal health situation of the country of origin, previous outcomes of FVO inspections and the third country’s overall compliance with EU legislation the physical checks for poultry products may be reduced to the levels below (EC, 1994a):

- whole eggs, hatching eggs: 20% of consignments
- poultry meat, poultry meat products and egg products: 50% of consignments
- feathers, game trophies, processed petfood: between 1% and 10% of consignments

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⁶ [http://europa.eu.int/comm/food/international/trade/index_en.htm](http://europa.eu.int/comm/food/international/trade/index_en.htm)
The Member States must organise the physical checks in such a way that it is not possible for an importer to predict whether any particular consignment will be subjected to a physical check.

6.2.12 BORDER INSPECTION POST APPROVAL AND THEIR EFFICACY CONTROL

The Commission approves BIPs where the Member State in which the BIP is located has given the adequate guarantees that the BIP is in conformity with EC requirements. BIPs are operated under the Member States’ authority and controls of imports are carried out under their responsibility. In order to ensure that BIPs are functioning efficiently and that uniform rules are applied, the FVO carries out inspections of these BIPs. The frequency of these inspections is determined by an assessment of the potential risks for animal health and public health in the Community, the quantitative and qualitative patterns of trade including the type and species of animals or products of animal origin concerned, relevant information concerning possible illegal imports and the potential risk of introduction of disease, data collected under the TRACES system - see third paragraph below - and taking into account the history of previous inspections made.

The conclusions reached in the BIP inspection reports of the FVO are that these safeguards are not 100% effective in ensuring that everything which enters the EU 'legally' conforms to EU animal and veterinary public health requirements. For example, “consignments posing a potential risk cannot be fully excluded” (EC, 2002c) and - referring to potential risks for public and animal health – “these risks cannot be completely excluded” (EC, 2002d).

In addition, the first cited FVO report referring to control in 8 Member States pointed out deficient facilities (5/8), deficient staffing (2/8), insufficient or not reliable completion of incoming and rejected consignments (7/8), acceptance of consignments without original certificates and/or without accompanying original certificates (3/8), occurrence in all BIP of consignments by passing the veterinary checks, inadequate respect of notifications of rejected consignments from other Member States. No details are available for the rejection of consignments that would allow a conclusion on a specific risk posed for the introduction of AI.

The introduction of the TRACES\(^7\) system by 1 January 2005 - a computerized network linking Member States’ veterinary authorities replacing the previous ANIMO system – will allow for an improved rapid tracing back of all movements of live animals and certain kinds of animal products between Member States and those which have been imported at EU borders.

More systematic information on identification and the reasons for the rejection of consignments at EU borders will become available, which should allow a better assessment of the risks associated with legal and attempted illegal importation.

Provided such data will be made available in the future these data might provide useful information for the assessment of spread of infection following its introduction into the EU.

\(^7\) [http://europa.eu.int/comm/food/animal/diseases/animo/index_en.htm](http://europa.eu.int/comm/food/animal/diseases/animo/index_en.htm)
There is a total of 293 BIPs approved in EU Member States and thereof 103 for live poultry.

**Table 6-2-1**: below shows the numbers and types of approved BIPs in EU Member States. BIPs may be approved for imports of both live animals (A) and products of animal origin (P) in different inspection centers located at one BIP. Therefore where both (A and P) are handled BIPs are set in brackets respectively in order not to be counted twice.

Whenever the BIP is approved for live poultry this BIP is indicated in bold e.g. (2/2) means that out of 2 BIP’s approved for live animals both are approved for poultry and (-/1) that there is only 1 BIP, which is approved for live poultry only.

BIPs may further be approved only for specific live animals species and during a certain time of the year and for various products for human and non human consumption and depending whether those products are chilled or frozen or do not require any specific temperatures.
Table 6-2-1: Type and numbers of border inspection posts by EU Member States (EC, 2001b).

<table>
<thead>
<tr>
<th>Code</th>
<th>Country name</th>
<th>Port</th>
<th>Airport</th>
<th>Rail</th>
<th>Road</th>
<th>Total</th>
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<td>4</td>
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<td>(0/1)</td>
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<td>-</td>
</tr>
<tr>
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<td>-</td>
<td>(1/1)</td>
<td>1</td>
<td>-</td>
</tr>
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<td>9</td>
<td>(10/10)</td>
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<td>1</td>
</tr>
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<td>(2/2)</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
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<td>3</td>
<td>-</td>
<td>-</td>
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</tr>
<tr>
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<td>2</td>
<td>(2/1)</td>
<td>2</td>
<td>3/2</td>
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<td>(11/11)</td>
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<td>(-/1)</td>
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</tr>
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<td>(7/7)</td>
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</tr>
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<td>-</td>
<td>(1/1)</td>
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<td>(-/1)</td>
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<td>-</td>
</tr>
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</tr>
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<td>(1/1)</td>
<td>3</td>
<td>(1/1)</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
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<td>(4/4)</td>
<td>5</td>
<td>-</td>
</tr>
<tr>
<td>SE</td>
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<td>5</td>
<td>3/2</td>
<td>(2)</td>
<td>-</td>
</tr>
<tr>
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<td>(-/1)</td>
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<td>-</td>
</tr>
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<td>United Kingdom</td>
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<td>21</td>
<td>(6/3)</td>
<td>9</td>
<td>-</td>
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<td></td>
<td>TOTAL of live poultry*</td>
<td>11*</td>
<td>-</td>
<td>66*</td>
<td>-</td>
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6.2.13 ILLEGAL IMPORTATION

6.2.13.1 Definition of illegal imports

Illegal importation of animals and their products includes all attempted third country importations at places other than BIPs, and importations at BIPs which attempt by whatever means to evade the normal import requirements. Responsibility for detection lays usually within the Member States’ custom services, however enforcement agencies will differ between Member States.

6.2.13.2 Illegal importation at recognised international entry points

For imports declared at BIPs, including cargo in ships and aircraft, details of the checks undertaken are given in 6.2.11. Illegal importation may be attempted via incorrect and misleading documentation. Some illegal imports may comprise items concealed within cages, containers etc., which will only be discovered if the contents are physically examined. As indicated in 6.2.11. not all consignments declared as products of avian origin are physically examined. The proportion of consignments declared to be of non-animal origin which are physically examined is even lower than that for those declared to be of animal origin (DEFRA International Animal Trade Division).

Illegal importation may be attempted simply by concealment without any attempt to make a declaration, and this may occur either at a BIP or other recognised international border entry point, including those for land, sea and airport entry. Detection then relies upon the vigilance of the enforcement authorities.

6.2.13.3 Other entry routes

Illegal importation may be attempted via routes not recognised as border entry points. Such routes include small boats or aircraft entering at places other than ports or international (customs) airports, and land border crossings not at border posts. Again, detection then relies upon the vigilance of the enforcement authorities. Although a potential route, no information is currently available, on probability or frequency of smuggling by this route.

6.2.13.4 Eastern borders

The eastern side of the EU is characterised by a long land border, lending itself to opportunities to effect illegal importation. It might be assumed that a land border is more easily crossed than sea or air. Although a potential route, no information is currently available, on probability or frequency of smuggling by this route.

6.2.13.5 Illegal imports: GB as a case study

Data on illegal importation collected by DEFRA (2002) as a consequence of the major outbreak of foot and mouth disease experienced in early 2001 are detailed in Annex III. The estimated scale factors in this case study indicate that only a small percentage of attempted illegal imports are detected, which means that a high proportion is successful. There is no data available for the remainder of the EU. However, the general conclusion that illegal imports are thus a potential route for the introduction of avian products contaminated with AI viruses, and the quantity into one country alone is not negligible. However the probability of entry of any virus via this route depends upon many additional factors including place of origin, product type, viability of the virus under processing and transport conditions pertaining etc. (VLA report, 2004).
6.2.14 POST-IMPORT REQUIREMENTS FOR LIVE POULTRY AND HATCHING EGGS

After clearance through the BIP live poultry and hatching eggs must be transported to the establishment at the place of final destination. With exception of slaughter poultry directly transported to the abattoir as stated above, live poultry and hatching eggs must be kept separate from other poultry or hatching eggs on the holding of destination for a defined period of time. A clinical inspection must be carried out by an authorised veterinarian and if necessary samples for laboratory investigations have to be taken at least at the end of this period (EC, 1996). For live poultry this period is six weeks, but can be reduced to 3 weeks provided that sampling and virological testing of cloacal swabs for AI as laid down in Council Directive 92/40/EEC (EC, 1992a) is carried out and has given favourable results. This derogation is not applicable to ratites. For day-old chicks hatched from imported eggs the period is 3 weeks respectively. The periods are extended in case of suspicion of HPAI.

6.2.15 IDENTIFICATION OF RISKS FOR THE INTRODUCTION OF HPAI AND LPAI VIA IMPORTATION OF LIVE POULTRY AND HATCHING EGGS

6.2.15.1 Live poultry (apart from day-old poultry and hatching eggs)

All species of poultry (pigeons being a possible exception see 6.3.3) can be potentially infected by every AIV subtype including H5/H7 (Easterday and Beard, 1984), even if they do not display overt clinical signs. Thus infected live poultry are theoretically potential agents for introduction of AIV.

HPAI – Domestic ducks represent possible asymptomatic carriers of HPAI viruses (Stallknecht, 1990a)

Furthermore, in other poultry species (e.g. quails and ostriches and possibly geese) HPAI virus infection could induce overt clinical signs or could evolve without clinical signs depending of the virus strain involved and other factors such as the age of the infected bird (Alexander et al., 1978; Alexander et al., 1986; Manvell et al., 1998; Capua at al., 2000b; Capua and Mutinelli, 2001a; 2001b).

It is therefore possible that clinical examinations carried out on live poultry prior to export might miss the presence of HPAI infection, either because it is a poultry species that does not show overt clinical signs or these have not yet developed as birds are incubating the infection. However, once HPAI is detected in a country this should lead to immediate notification of disease and cessation of exports due to legal EU and international requirements.

LPAI – LPAI infections may induce only mild clinical signs in the affected poultry farms, and thus remain active and undetected in an infected country (Capua and Marangon, 2000). This may result in apparently healthy birds originating from a country not known to be LPAI free to harbouring and spreading the infection.

Since it will not be until 1/1/2006 that exporting countries will be required according to OIE rules (OIE, 2005a) to prove LPAI freedom for their country or region or specific compartment, presently there is an absence of knowledge of countries’ LPAI status and this represents a risk of LPAI virus introduction in the importing country.
6.2.15.2 Day-old-poultry

As AI viruses are generally embryo lethal the AIV-infected eggs would most likely not hatch in the hatchery of the country of export. Day-old chicks will have had only a very limited exposure time to post-hatching virus. Therefore they are much less likely to be infected than older birds. In addition, legal export and transport conditions apply.

Furthermore, it must be noted that they have hatched from eggs that have been in the incubator in the country of origin for 21 days, i.e. means (unless asymptomatic in certain species), the infection should most likely have become overt in the parent flock before export takes place.

As mentioned in 6.2.3. earlier imports of live poultry into the EU vaccinated against AI is currently not authorised, but will become an option in view of recent modifications to the OIE code (OIE, 2005a) and upcoming revision of EC legislation (EC, 2005a). Therefore, it must be noted that vaccinated birds, disease free but possibly infected by a field strain, might pose a risk of AIV introduction. However this risk is reduced because vaccinated birds may only be imported if specific surveillance to detect virus circulation has been carried out.

6.2.15.3 Hatching eggs

**HPAI** - HPAI viruses have been reported as present on the surface and in the contents of eggs laid by infected hens on most occasions this has been investigated (see 6.4.2.3). HPAI virus introduction in a country via importation of hatching eggs is possible, when eggs have been laid during the incubation period before cessation of lay.

Before hatching eggs are put into the incubators fumigation with formaldehyde or other sanitisation measures in accordance with Community legislation (EC, 1990a) and OIE recommendations (OIE, 2005c) are common industry practice and should therefore avoid that AI viruses are present on the hatching eggs’ surface.

**LPAI** - Concerning LPAI, Ziegler et al. (1999) reported the isolation of virus from the oviduct in hens infected with H7N2 LPAI during the 1996-1998 Pennsylvania outbreaks, and LPAI viruses are excreted in large amounts in the faeces of infected birds and faecal material frequently contaminates the outside of eggs shells (see 6.4.2).

The risk of introducing LPAI infection to a country which imports hatching eggs and day-old-poultry from a country not known to be free from LPAI is mainly related to faecally contaminated materials (e.g. trays, packaging materials etc.) which may be re-used in the importing country; however, legal requirements for fumigation and egg packaging are likely to reduce these risks to negligible levels.

Observations and conclusions on the risk for the introduction of AI (both HPAI and LPAI) via hatching eggs have been made essentially for eggs from chickens and turkeys; thus hatching eggs derived from other species might be considered as potential agents for introduction of AIV.

As stated above for imports of live poultry, vaccination against parent flocks from which hatching eggs or day-old poultry are derived might have an influence on their
infectious status, but should be minimised by the surveillance requirements for vaccinated flocks.

6.3 RISKS OF AI INTRODUCTION BY BIRDS OTHER THAN POULTRY, (ORNAMENTAL BIRDS, PET BIRDS, FIGHTING COCKERELS, RACING PIGEONS, BIRDS FOR SHOWS AND EXHIBITIONS

It should be noted that under the current EU definition, AI viruses (even if HPAI or LPAI H5 and H7) infecting birds that fall outside the definition of poultry are not notifiable AI.

6.3.1 CAPTIVE CAGED BIRDS - PET, ZOO AND SHOW BIRDS

The first isolations of avian influenza viruses from captive caged birds were reported in the 1970s. These had been usually made as a result of monitoring for Newcastle disease [ND], as trade in caged birds had been associated with spread of ND virus during the panzootic of that disease in the early 1970s. In view of the enormous trade in wild-caught captive birds many countries imposed quarantine on imports during the 1970s and as a result many more isolations of avian influenza viruses from both passerine and psittacine species have been obtained since that time. The isolates of avian influenza viruses obtained from birds in quarantine in Great Britain since 1975 are listed in Table 3.4.1. The vast majority of AI viruses were of H3 and H4 subtypes, usually H3N8 and H4N6 combinations. It is worth noting that there were periods, often covering a number of years when no AI viruses were isolated from this source. In 1979 [H7N1] and again in 1989 [H7N7] H7 subtype LPAI viruses were obtained from birds in quarantine in Great Britain.

Table 6-3.1.: Isolations of AIV from birds in quarantine in UK (Alexander, 1994-2004)

<table>
<thead>
<tr>
<th>Date</th>
<th>Subtype</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>1975</td>
<td>H4N6</td>
<td>29</td>
</tr>
<tr>
<td>1976-06.1977</td>
<td>H3N8</td>
<td>58</td>
</tr>
<tr>
<td>07.1977-1978</td>
<td>NONE</td>
<td></td>
</tr>
<tr>
<td>1979</td>
<td>H4N6</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>H10N7</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>H7N1</td>
<td>1</td>
</tr>
<tr>
<td>1980-06.1987</td>
<td>NONE</td>
<td></td>
</tr>
<tr>
<td>1987</td>
<td>H3N8</td>
<td>1</td>
</tr>
<tr>
<td>1988</td>
<td>H3N8, H3N6</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>H4N6</td>
<td>4</td>
</tr>
<tr>
<td>1989</td>
<td>H3N8</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>H4N2, H4N3, H4N6</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>H7N7</td>
<td>1</td>
</tr>
</tbody>
</table>
In other countries where captive caged birds have been monitored, very similar patterns and similar subtypes have been obtained. In the USA Panigrahy and Senne (1998) noted that the majority of AI viruses from caged birds came from passerine species. It may well be that passerines are the natural reservoir for these AI viruses (Ibrahim et al., 1990) and spread to psittacine species occurs at holding stations or during transit.

From time to time there have also been reports of AI infections of caged birds outside quarantine. Two of the earliest of these was the isolation of a LPAI of H7N1 from a pet African grey parrot (Psittacus erithacus) in N. Ireland in 1973 (McFerran et al., 1974) and the isolation of an H7N7 virus [IVPI 0.0] from a pet macaw (Ara sp.) in 1980 (Alexander 1982b).

The isolations of H5 and H7 viruses from EU countries since 1991 is summarised in Table 6.3. None have been reported since 1994. Interestingly all the H7 isolates have been made from psittacine species.

<table>
<thead>
<tr>
<th>Year</th>
<th>Country</th>
<th>Host</th>
<th>Subtype</th>
<th>Virulence [IVPI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>1991</td>
<td>Italy</td>
<td>monk parrot [Meopsitta monachus]</td>
<td>H7N2</td>
<td>0.85</td>
</tr>
<tr>
<td>1991</td>
<td>Italy</td>
<td>monk parrot [Meopsitta monachus]</td>
<td>H7N2</td>
<td>0.45</td>
</tr>
<tr>
<td>1991</td>
<td>Italy</td>
<td>cockatiel [Nymphicus hollandicus]</td>
<td>H7N2</td>
<td>0.27</td>
</tr>
<tr>
<td>1991</td>
<td>England</td>
<td>turaco [Touraco musophagida]</td>
<td>H5N2</td>
<td>0.00</td>
</tr>
<tr>
<td>1994</td>
<td>Netherlands</td>
<td>parakeet</td>
<td>H7N1</td>
<td>0.00</td>
</tr>
<tr>
<td>1994</td>
<td>England</td>
<td>sun conure (Aratinga solitialis)</td>
<td>H7N1</td>
<td>0.00</td>
</tr>
<tr>
<td>1994</td>
<td>England</td>
<td>parrot</td>
<td>H7N1</td>
<td>0.00</td>
</tr>
<tr>
<td>1994</td>
<td>England</td>
<td>painted conure</td>
<td>H7N1</td>
<td>0.00</td>
</tr>
</tbody>
</table>

None of these birds were in quarantine when the viruses were isolated. Source (Alexander, 1994-2004).
EU legislation for the importation of birds other than poultry from third countries is encompassed in Commission Decision 2000/666/EC (EC, 2000c), which is primarily concerned with restricting the introduction of Newcastle disease or avian influenza. It applies for commercially traded caged birds and essentially requires:

- The birds must originate from OIE member countries (currently 167 members8)
- The birds must come from a holding in the country of origin where they have been kept for at least 21 days prior to export.
- The birds are transported in cages or crates that contain only one species of bird or one species per compartment if compartmentalised.
- The birds are moved to designated officially approved quarantine premises where they are held for 30 days and subjected to at least two veterinary inspections at the beginning and the end of this period before release.
- During the 30 day period either the imported birds or sentinel birds must undergo laboratory testing for ND and AI. In addition, birds becoming sick or those that have died have to be tested according to the procedures described in the Directive 92/40/EEC (EC, 1992a).

These quarantine provisions however do not apply for imports of pet birds that are accompanied by their owner (EC, 2003b) or to imports of birds (EC, 1992b) for exhibitions, show or contest or which form part of a zoo or circus. This falls within the competence of national legislation of Member States.

There is no evidence that AI viruses have spread from captive caged birds to poultry. However, during the 1970-73 Newcastle disease [ND] panzootic it was considered that movement of captive caged birds, particularly psittacines, was responsible for the introduction of that ND virus to poultry in California (Walker et al., 1973) and there was the report of ND virus spread from a pet imported parrot to a backyard poultry flock in Great Britain in 1978. This suggests that caged birds as a source of AI infecting poultry cannot be ruled out.

According to the above testing procedures, all birds other than poultry coming into the EU via a BIP should be transported directly to approved quarantine facility or centre in cages. Upon arrival the birds are clinical inspected by an official veterinarian who also has to check the mortality records. In addition, virological examination has to be carried out. Swabs must be collected of all birds if the consignment is less than 60 birds and of 60 birds when the consignment is larger. Using this sample size, one positive sample will be detected with a probability of 95% when the prevalence of infected birds is 5% of more. However, consignments may consist of many different bird species with unknown susceptibility for AI. The decision does not prescribed that samples should be taken of all species and or from all cages in the consignment.

In the EU a total of 189 bird quarantine facilities in 17 Member States are approved by the national veterinary services.

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8 OIE list of members countries and delegates http://www.oie.int/eng/OIE/PM/en_PM.htm
6.3.2 OTHER SHOW BIRDS

Other show birds that are not classified as captive caged birds may include birds of the Phasianiformes (Galliformes) and Anseriformes orders as well as pigeons and as such are as likely to be able to be infected and spread AI viruses as their corresponding poultry. The question here is whether or not they represent any additional hazard, and the main cause for concern is the gathering of such birds from different areas and countries when they congregate for shows and fairs. There is a historical record implicating such birds in the spread of HPAI. HPAI seems to have been endemic but sporadic in Germany from 1890-1930 (Todd and Rice, 1930), but one incident was notable. In 1901 birds at a poultry show in Brunswick were observed to be sick and dying. The organisers insisted that all exhibits should be removed from the show. Stubbs (1926) recalls that this was such an effective method of spreading the disease to the farms of origin and beyond that it became known as “Brunswick disease” throughout Germany.

6.3.3 RACING PIGEONS

Historically, pigeons (Columba livia) have been considered resistant to avian influenza viruses and in experiments infections of pigeons have been difficult to establish. Doyle (1927) used the susceptibility of pigeons to infection with Newcastle disease virus with disease and death as one of the criteria used to distinguish that virus from “fowl plague” [HPAI] virus (Doyle, 1927). In experiments with the HPAI H5N9 virus ty/Ontario/7732/66 Narayan et al., (1969) showed intravenously infected pigeons seroconverted but no virus shedding or clinical disease was detected. Using the same virus Slemons and Easterday (1972) reported disease and death in 1/19 pigeons infected intranasally and isolation of virus from the trachea of two of the infected pigeons. Panigrahy et al., (1996) infected pigeons experimentally with HPAI and LPAI viruses of H5N2 subtype and HPAI H7N7 and LPAI H7N1, by oculonasal and intravenous routes and in contact with these infected birds. They failed to produce disease or seroconversion in any of the infected pigeons and only one tracheal swab, taken on day 3 from a LPAI H7-infected pigeon, yielded virus, which the authors considered to be residual inoculum. Similarly, Perkins and Swayne (2002) failed to show any virus excretion, disease, lesions or seroconversion in pigeons infected intranasally with HPAI A/chicken/Hong Kong/220/97 H5N1 virus.

These experimental data are consistent with field studies during the 1983-84 HPAI H5N2 Pennsylvania epizootic in which Nettles et al., (1985) reported failure to isolate any AI virus from 473 pigeons, 7 doves and 81 samples of material contaminating the feet of pigeons that were sampled in the infection quarantine area (Nettles et al., 1985). However, it should be noted that H5N1 viruses were reported as isolated from feral pigeons in Hong Kong in 2002 and 2003 (Li et al., 2004). Similarly HPAI of the H7N1 subtype was isolated from a dead collared dove (Streptopelia decaocto) during the 1999-2000 Italian epidemic (Capua et al., 2000c).

In experiments aimed at assessing the ability of the HPAI H7N7 virus responsible for the outbreaks in The Netherlands in 2003 to infect pigeons administration of $10^5$ EID$_{50}$ to individual pigeons intranasally failed to result in the excretions of virus, clinical or histological signs or seroconversion (Shell, W. 2005).
The conclusion from these data could be that pigeons are very unlikely to become infected with AI viruses and therefore pose very little threat of introducing AI viruses into an area. However, some caution should be exercised in dismissing racing pigeons as potential agents for introduction and spread of AI viruses. Host range may be very much related to specific virus strains and could evolve during an epizootic. Equally racing pigeons could act as mechanical vectors if contaminated with infective faecal material while invading farms with affected poultry. The nature of racing pigeons over large distances and from country to country plus the procedure of gathering them together for release represent introduction risks that are unique to these birds.

A detailed review of HPAI virus infections of pigeons has recently been published by (Kaleta and Honicke, 2004).

6.3.4 Other pigeons and doves [not poultry or racing pigeons]

Other pigeons and doves may be kept as pets, show birds (either similar to captive caged birds or active show birds such as ‘tumblers’) or working birds (e.g. as magicians’ props). In view of the probable lack of susceptibility of pigeons to AI infections the risk of introductions by such birds may be regarded a very low, but should not be entirely ignored in any legislation aimed at control.

6.3.5 Birds of prey

Hunting with falcons is practised in a number of countries around the world. The birds have close contact with humans and are highly domesticated and yet the nature of the purpose for which they are kept means they also have contact with feral birds. There have been two recent reports of AI infections of falcons. Manvell et al. (2000) reported the isolation of a HPAI virus of H7N3 subtype from a peregrine falcon (Falco peregrinus) dying in the United Arab Emirates. The virus showed close homology with the H7N3 viruses responsible for the outbreaks in Pakistan four years earlier (Banks et al., 2000a,b). During the HPAI outbreaks in Italy in 2000, an H7N1 virus was isolated from a saker falcon (Falco cherrug) that died three days after normal hunting activity (Magnino et al., 2000). There was a single isolation of the H5N1 HPAI virus in Hong Kong in January 2004 from a peregrine falcon (Falco peregrinus) that was found dead.

The significant threat that illegal movement of birds of prey has for the introduction of HPAI was highlighted by the recent incident in Belgium. On 18 October 2004, a Thai man travelling from Bangkok to Brussels was apprehended by customs officials at Brussels international airport, and found to be illegally carrying two mountain hawk eagles (Spizaetus nipalensis) in his hand luggage. These birds were seized and killed humanely. Although they had shown no clinical signs one bird was shown to have bilateral pneumonia and H5N1 HPAI was isolated from lung material (Van Borm et al., 2005).

6.3.6 Fighting cockerels

Fighting cockerels (cocks) are likely to be as susceptible as other domestic fowl. However, in view of the largely illegal nature of such birds and their movement, it may well be that they represent a real and difficult risk in introduction of AI virus. There do not appear to be any specific reports of AI infections in fighting cocks, but their role in the propagation and spread of Newcastle disease virus in Western states of the USA in 2002-03 is an indication of how important such birds could be in the introduction and spread of AI (Senne, 2004).
6.4 ASSESSMENT OF THE RISK OF INTRODUCTION OF AI BY AVIAN PRODUCTS

6.4.1 GENERAL COMMENTS

When assessing the risk of introduction of AI viruses by avian products, it is necessary to consider the pathogenesis of the disease in the infected host as this will determine in which organs or products these viruses are present during the acute course of infection and will be highly influenced by the type of virus (LPAI or HPAI), by the strain of virus, by the animal species involved and possibly, within a species, by other factors such as age and exacerbating factors.

Infections with HPAI viruses, particularly in chickens and turkeys, have a generalised course with extensive viraemia and virus may be detected not only in the respiratory and enteric tracts but also in internal organs such as spleen, pancreas, heart, liver, kidney, nervous system as well as muscle and skin. (Starick and Werner, 2003)

Theoretically, LPAI viruses are restricted to replication in the respiratory and intestinal tracts and infections should not result in infective material outside these areas. However, under exacerbating conditions more generalised LPAI virus infections have been reported, especially in turkeys (Mutinelli et al., 2003) and therefore the theoretical absence of LPAI viruses in some poultry products cannot be guaranteed. In addition the extensive replication of LPAI viruses in the intestinal tract and large amounts of virus excreted in the faeces, means there is the potential that products could be contaminated with such infective faeces and therefore pose a risk to susceptible birds if adequate hygienic measures are not practised.

The assessments and comments in sections 6.4.2. to 6.4.13. are necessarily limited to the risk that infectious virus may be present in the product. In addition there is also the need when estimating the likelihood that this will lead to the introduction of AI to assess the risk that the presence of virus will lead to its introduction to poultry or other bird populations. For example, imported meat may be infective or contaminated with infective material, but the chance of that meat reaching poultry or other birds still containing infectious virus may be very small and will depend on restrictions imposed on feeding swill etc. in the importing country.

In 1993 the use of swill originating from means of international transport, such as ships, land vehicles or aircraft, became prohibited for the feeding of poultry with entry into force of Council Directive 92/66/EEC (EC, 1992c) for the control of Newcastle disease. It further foresees that the use of other swill or poultry scraps, could only be authorized for the feeding of poultry after a heat-treatment in appropriate facilities ensuring that the disease is not transmitted and the Newcastle-disease virus is destroyed.

However, since October 2002, (EC, 2002b) the so-called “intra-species recycling ban” prohibits the uses of animal by-products and processed products for the feeding of a species with processed animal protein derived from the bodies or parts of bodies of animals of the same species and the feeding of farmed animals (thereby including poultry) other than fur animals with catering waste or feed material containing or derived from catering waste.

The probability of exposure to these avian commodities will depend on the likelihood that illegal swill feeding occurs and secondly, whether this swill contains raw scraps or is prepared from kitchen waste that has undergone some form of heat treatment for...
preparation as food. No information is available on the actual amounts of waste at exposure, or virus in waste. In addition the titre of viable virus will decrease further due to environmental and dilution effects.

6.4.2 EGGS FOR CONSUMPTION

6.4.2.1 Legal definition:

Concerning marketing standards of eggs, Council Regulation (EEC) 1907/90 (EC, 1990b) in conjunction with Commission Regulation (EC) 2295/2003 (EC,2003c) apply and define 'Eggs' as hen eggs in shell, suitable for direct human consumption or for use in the food industries, except for broken eggs, incubated eggs and cooked eggs.

These standards define two grades (classes) of table eggs (A and B) according to different physical characteristics: (i) Grade (class) A eggs (“fresh eggs”) should have a “normal, clean and undamaged” shell and cuticle; they will not be washed or cleaned before or after grading, and will be not chilled or treated for preservation. (ii) Grade (class) B eggs, i.e. eggs “which do not meet requirements applicable to eggs in grade A”. Such eggs may only be used by the food or non-food industries. Table eggs are normally grade A eggs. A fresh egg of grade A has to be produced in a way that ensures it is fit for human consumption. It must comply with the minimum characteristics set by the above cited legislation (EC, 1990b; EC, 2003c,) in particular, its shell and cuticle must be normal, naturally clean and undamaged. Imports from third countries must comply with these European marketing standards.

As of 1/1/2006 (EC, 2004a) the following definition will apply in relation to specific EU rules for the hygiene of foodstuffs:

‘Eggs’ means eggs in shell - other than broken, incubated or cooked eggs - that are produced by farmed birds and are fit for direct human consumption or for the preparation of egg products.

6.4.2.2 Legislation for imports of eggs for consumption

Eggs for consumption may be imported from countries from which the import of fresh poultry meat is authorised (see 6.4.6.2) and in addition from Iceland, South Korea, Madagascar, Malaysia and Turkey. However, none of the additionally listed countries can currently comply with the residue requirements (EC, 2004c) or have been suspended due to recent outbreaks of HPAI, so that imports do not take place.

Besides the EC third country approval no further EC animal health requirements are currently applicable to eggs for consumption.

6.4.2.3 Risk of LPAI/HPAI presence in eggs for consumption

HPAI. HPAI viruses have been reported as present on the surface and in the contents of eggs laid by infected hens on most occasions this has been investigated (Moses et al., 1948; Beard et al., 1984; Narayan et al., 1969; Cappucci et al., 1985; Bean et al., 1985; Starick and Werner, 2003). In experiments M. Brugh (cited by Swayne and Beck, 2004) was able to demonstrate the presence of H5N9 HPAI virus in eggs laid 3-4 days after infection with titres up to $10^{4.9}$ EID$_{50}$/ml of egg product.
Table eggs from HPAI infected hens and egg trays and other fomites that may come in contact with such eggs therefore represent a very high risk for the potential spread of HPAI virus.

**LPAI**: There has been no report of a natural infection of laying birds with LPAI viruses that has resulted in eggs containing virus internally. (Swayne and Beck 2004) cited P. Dunn as reporting failure to isolate AI virus from the albumen of 9930 eggs tested during the monitoring of three layer flocks in Pennsylvania infect with H7N2 LPAI during 1996-1998. Equally, (Lu et al., 2004) failed to demonstrate the presence of LPAI H7N2 virus in egg shell swabs, albumen or yolk of eggs laid by hens with respiratory signs and egg production problems despite the virus being present in tracheal and cloacal swabs. In contrast Ziegler et al. (1999) reported the isolation of virus from the oviduct in hens infected with H7N2 LPAI during the 1996-1998 Pennsylvania outbreak. It would appear that while there may be the potential for table eggs to become infected with LPAI viruses internally, the marked absence that this has occurred suggests that this risk is very low.

However, LPAI viruses are excreted in large amounts in the faeces of infected birds and faecal material frequently contaminates the outside of eggs shells. It would seem a wise precaution that the outside of table eggs are treated in some way to reduce the likelihood of faecal and/or virus contamination, either as a routine measure or when the parent flock is known to be or suspected of being infected with LPAI virus. Egg trays and other packaging material, in particular if packaging procedures take place in close proximity to the laying flocks, may also be contaminated with faeces/virus or infective egg fluids from cracked or broken eggs and it would be a wise precaution that these should be disposed of after use or thoroughly washed and disinfected. Similarly other fomites that may come in contact with eggs should be thoroughly disinfected after each use.

### 6.4.3 Egg products

**6.4.3.1 Legal definition**

The current definition for egg products (EC, 1989) is in line with the definition applicable as of 1/1/2006 (EC, 2004a) which says:

‘Egg products’ means processed products resulting from the processing of eggs, or of various components or mixtures of eggs, or from the further processing of such processed products.

**6.4.3.2 Legislation for imports of egg products**

In addition to the countries authorised for imports of eggs for consumption, the following countries are authorised for the importation of egg products (EC, 2003d): currently Albania, Greenland, Hong Kong, India, Former Yugoslav Republic of Macedonia, Mexico, New Caledonia, Russia, Serbia and Montenegro, Singapore provided they can comply with the hygiene requirements for egg processing products and the residue monitoring (2004c). No specific EU animal health requirements exist for this product.
6.4.3.3  Risk of LPAI/HPAI presence in egg products

Egg products are frequently obtained from eggs downgraded from table eggs, often due to cracked shells. As a result these products may be considered to have a greater risk of contamination with faeces/virus than intact table eggs if they have not been treated in a way that would reduce the likelihood of virus survival to an acceptable level.

Most egg products are whole eggs or parts of the egg that have been liquefied or homogenised and subjected to some form of heat treatment, or are products that contain egg material treated in this way. Very few studies have been published that assess the survival of HPAI or LPAI viruses in egg materials subjected to heat treatments normally applied during commercial processing. Swayne and Beck (2004) conducted a series of experiments aimed at assessing the heat inactivation of a H7N2 LPAI virus and a H5N2 HPAI virus in various egg products at temperatures used commercially. They calculated $D_t$ values (the time taken for the treatment to inactivate $1 \log_{10}$ for the two viruses in each of the products and concluded that for homogenised whole egg, liquid egg white and 10% salted yolk the temperature and time applied in standard industrial pasteurisation was likely to reduce a level of $10^{4.9} \text{ EID}_{50}/\text{ml}$ of egg product to below or very close to the probability of 1:100 that 1ml of product would contain 1 EID$_{50}$. However, they considered that the industrial standard of 54.4°C for 7 days for dried egg white would be inadequate for acceptable heat inactivation of virus.

Assessing the risk of treated products depends on several factors i.e. starting titre of virus, the acceptable level of probability of virus survival in what quantity of product, even when the $D_t$ value of the virus in the product is known. Those suggested by Swayne and Beck (2004) do not seem unreasonable, but some recipients of the products may demand greater assurance under some circumstances.

6.4.4 SEMEN OF POULTRY

6.4.4.1  Legislation for imports of semen of poultry

Currently no harmonised EU legislation is laid down for this product.

6.4.4.2  Risk of LPAI/HPAI presence in semen of poultry

There have been no reports of LPAI or HPAI virus in the semen of birds. However, since it is well documented that in most infections of birds with HPAI virus there is viraemia, it seems possible that HPAI virus may be present in the semen of infected poultry. Whereas, with the caveat that strain variation may occur [see 6.4.1] LPAI infected birds should pose no risk of transmitting LPAI through semen unless contaminated with faeces or other body fluids during the collection process.

Using standard artificial insemination procedures, Samadieh and Bankowski (1970; 1971) were able to show turkey hens became infected when given semen contaminated experimentally with AI virus.

6.4.5 SEMEN OF BIRDS OTHER THAN POULTRY

6.4.5.1  Legislation for imports of semen of birds other than poultry

Currently no harmonised EU legislation is laid down for this product.
6.4.5.2 Risk of LPAI/HPAI presence in semen of birds other than poultry

There is trade and movement of semen from birds other than poultry from one country to another, usually zoo birds or endangered species. The risk of semen being infective is probably slightly higher than for poultry because of the uncertainty of the status of the donor birds in terms of AI infections. In addition, little is known about the pathogenesis of AI infections in these species, and particularly for birds belonging to the Anseriformes, there may be the risk of HPAI being systemic although asymptomatic, thus semen could represent a means of spreading the infection.

6.4.6 Fresh meat of poultry

6.4.6.1 Legal definitions

In EU legislation “meat” means edible parts of the slaughtered animals including blood. “Fresh meat” is defined as meat that has not undergone any preserving process other than chilling, freezing or quickfreezing, including meat that is vacuum-wrapped or wrapped in a controlled atmosphere (EC, 2004a). ‘Carcase’ means the body of an animal after slaughter and dressing. ‘Offal’ means fresh meat other than that of the carcase, including viscera and blood. ‘Viscera’ means the organs of the thoracic, abdominal and pelvic cavities, as well as the trachea and oesophagus and, in birds, the crop. The carcase, offal and viscera are all considered as edible parts and therefore fall under the requirements for fresh meat. ‘Minced meat’ means boned meat that has been minced into fragments and contains less than 1 % salt. ‘Mechanically separated meat’ or ‘MSM’ means the product obtained by removing meat from flesh-bearing bones after boning or from poultry carcases, using mechanical means resulting in the loss or modification of the muscle fibre structure.

As regards animal health rules for the imports of ‘poultry meat’ (EC, 1991a) this refers to meat of fowl (chicken), turkeys, guinea fowl, ducks, geese, quails, pigeons, pheasants and partridges and ratites thereby including farmed feathered game species.

In addition, meat of wild feathered game, which is hunted/killed in the wild in the country of origin, can be imported from most third countries from where also poultry meat imports are authorised (EC, 2000a). This can occur in unplucked, unviscerated form and needs to be performed without undue delay upon arrival in the game-handling establishment at destination. It is further specified in which cases (countries distant to the EU) transport must be carried out by aeroplane.

As wild feathered game meat is sourced from wild birds that can move freely, also transboundary, their health status in relation to AI infections is difficult to assess; therefore fresh meat of such species might pose a significant greater risk than fresh meat sourced from poultry or farmed feathered game.

6.4.6.2 EC approved third countries

In addition to the 10 countries approved for live poultry [see 6.2.2.] imports for fresh poultry meat are also authorised from a small region of the PRC (China)9, Thailand2 and

9 PRC suspended for public health (residues) since 2002 and both PCR and Thailand for HPAI occurrence since early 2004.
Tunisia (EC, 1994b). Meat of farmed feathered game and wild feathered game can be imported from countries listed in 2000/585/EC (EC, 2000a). For fresh ratite meat the four African countries approved for live ratite imports and Zimbabwe are authorised for importation (EC, 2000b).

Slaughterhouses, cutting plants, establishments for meat products and other products of animal origin approved in third countries for imports into the EU can be found on internet listed by country or type of product:

6.4.6.3 Animal health requirements as they refer to HPAI

Fresh poultry meat can only be imported from countries or parts thereof that are free from HPAI [see 6.2.3] and when the meat has been obtained from poultry that:

- has been held in the territory of the third country since hatching or has been imported as day-old chicks;

- comes from holdings:
  - which have not been placed under animal restrictions in connection with any disease for which poultry is susceptible,
  - around which, within a radius of 10 km, including where appropriate the territory of a neighbouring country, there have been no outbreaks of HPAI for at least 30 days:

- has not been slaughtered in the context of any animal health scheme for the control or eradication of poultry diseases;

- during transport to the slaughterhouse, did not come into contact with poultry infected with HPAI

- has been slaughtered in EC approved slaughterhouses which, at the time of slaughter, are not under restrictions due to a suspect or confirmed outbreak of HPAI and around which, within a radius of 10 km, there have been no outbreaks of HPAI for at least 30 days:

The meat obtained must not have been in contact, at any time of slaughter, cutting, storage or transport with meat which does not fulfil these requirements.

A health mark must be applied to the wrapping and the packages of fresh poultry meat which contains the ISO code reference of the country of origin, the veterinary approval number of the slaughterhouse or, where appropriate, the cutting premises and in addition it must enable the identification of the veterinarian who carried out the health inspection of the meat.

An animal heath certificate signed by an official veterinarian who must thereby guarantee that all EC requirements have been met, must accompany the consignment of the fresh meat to the BIP.

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10 [http://forum.europa.eu.int/irc/sanco/vets/info/data/listes/list_all.html](http://forum.europa.eu.int/irc/sanco/vets/info/data/listes/list_all.html)
6.4.6.4  Hygiene requirements for EC approved slaughterhouses

Detailed EC hygiene rules are laid down for the approval of slaughter and cutting plants, ante- and post mortem inspection of slaughter animals and hygiene requirements at slaughter, storage and transport (EC, 2004 a,b). Establishments approved in third countries must either comply with these requirements or must have their own controls in place that have been assessed as equivalent to EC rules by the Commission inspection services.

These rules shall provide for detection of clinical signs of infection or a change in production data (included in “food chain information”) which might indicate presence of disease before the animals are presented to slaughter.

In case these findings lead to a suspicion of HPAI infection animals will not be authorised for slaughter for export to the EU and animal health rules in relation to suspicion of disease will apply.

Post-mortem inspection after slaughter provides for detection of pathological signs, which might indicate presence of AIV infections (HPAI and LPAI, in particular in highly susceptible species and when exacerbated by secondary bacterial infections). In many cases post-mortem findings will result in the immediate discard of the carcase.

Stunning, bleeding, skinning or plucking, evisceration and other dressing must be carried out without undue delay in such a way that contamination of the meat is avoided. In particular, measures must be taken to prevent the spillage of digestive tract contents during evisceration.

All along the pre- and post slaughter process, if findings appear that cannot rule out a condition that leads to suspicion of any condition which might adversely affect human or animal health, paying particular attention to OIE listed diseases (OIE, 2004a), additional examinations (including laboratory examinations) have to take place to determine presence of disease or other factors that might require the meat to be declared as unfit for human consumption.

However, the influences on AIV presence, reduction and dilution factors of the different steps of the slaughter process and of subsequent chilling, freezing, storage and maturation have not been addressed in this report, due to lack of data available for AIV.

6.4.6.5  Risk of LPAI/HPAI presence in fresh meat

In line with the EU definition fresh meat includes frozen meat, chilled meat, minced meat and mechanically recovered meat (OIE, 2005b; EC, 2004a). However, how the meat is prepared after slaughter may have significant effects on the survival of infectious virus. For example all influenza viruses are considered to be extremely sensitive to acid pH. On the other hand it is known that poultry meat does not always experience a significant drop in pH, which might however be species dependant (e.g. ratite meat). There appear to be no adequate studies on these aspect in the literature, but it may well be that chilled meat poses less of a risk than frozen meat and the speed at which meat is frozen or chilled after slaughter may influence the survival of infectious virus.

HPAI. In most poultry species HPAI viruses cause viraemia and systemic infections with virus replication in muscle tissues and it has long been recognised that HPAI viruses may be detected in the muscle tissues of infected birds and numerous
experiments have shown this. For example, Purchase (1931) was able to show that chickens fed on muscle tissues from HPAI infected birds became infected. More recently there have been numerous reports of the detection of virus in meat/muscles of HPAI-infected poultry (Mo et al., 1998; Perkins and Swayne, 2001; Tumpey et al. 2002, 2003; Lu et al., 2003b; Swayne and Beck 2004).

There seems little doubt that meat from chickens, turkeys, ducks and other poultry slaughtered during an active HPAI infection will contain infectious virus and although titers may be low there may be sufficient virus present to infect other birds if fed to them untreated (Swayne and Beck, 2004).

Particular consideration should be given to fresh duck meat. Ducks usually remain healthy when infected with HPAI viruses although they do become viraemic and virus may be isolated from internal organs (Wood et al., 1995). Infected ducks may well pass veterinary inspection at and prior to slaughter and in recent years HPAI H5N1 virus was isolated from duck meat imported into Korea (Tumpey et al., 2002, 2003).

LPAI. There have been very few reports in which the presence of LPAI virus in meat has been estimated in either experimental or field infections of poultry. In keeping with the assumed lack of systemic virus replication following LPAI infections, Mo et al. (1998) using immunohistochemical techniques, failed to detect the presence of LPAI virus in the skeletal muscles of infected birds. Swayne and Beck (2004) failed to detect any virus in the meat of chickens infected experimentally with LPAI H5N2 or H7N2 viruses. But, in contrast, Kishida Kishida et al. (2004) reported the isolation of LPAI H9N2 virus from imported chicken meat and were able to demonstrate virus in the muscles of chickens infected experimentally with the isolated virus.

Without the report of Kishida et al., (2004) the conclusion would almost certainly be that in keeping with theory the risk of the presence of LPAI viruses in fresh meat is likely to be very low to negligible even from birds excreting infectious virus at the time of slaughter and that a greater risk would be the contamination of meat by infective faeces at or after slaughter. However, the presence of the H9N2 virus in meat and confirmation that it is present in muscle tissues during infections suggests that the presence of LPAI viruses in meat may be strain specific and that the risk may need to be assessed on a case by case basis.

The potential for contamination with faeces and other potentially infective body fluids would appear to be greater for whole carcases than meat cuts.

6.4.7 MEAT PRODUCTS FROM POULTRY

6.4.7.1 Legislation on imports of meat products and definitions

‘Meat product’ means processed products resulting from the processing of meat or from the further processing of such processed products, so that the cut surface shows that the product no longer has the characteristics of fresh meat.

The fresh meat used for processing of these products must comply with the general animal health requirements as laid down in Council Directive 2002/99/EC (EC, 2002a) Commission Decision 2005/432/EC (EC, 2005b) lists the third countries from where importation of meat products is authorised and the specific animals health requirements. It indicates if depending on the animal health status of the third country in relation to
the species from which the meat has been obtained for further processing which treatment must be applied to mitigate the risks of disease transmission via this product. Meat for the manufacture of meat products may not be obtained from animals which have been kept on a holding which is under animal health restrictions due to a suspicion or the presence of disease, but where in the case of restrictions on the country’s territory or a part thereof a certain treatment is foreseen.

Meat products consisting of or containing poultry meat may be imported from the countries meeting the requirements for fresh poultry meat [see 6.4.6.2] and additional from 10 countries or parts thereof (Hong Kong, Iceland, Madagascar, Malaysia, Mexico, Serbia and Montenegro, Singapore, South Korea, Turkey, Uruguay) under the condition that the products have undergone a heat treatment of at least 70°C. This temperature must have been reached throughout the meat during the processing of the product. However, it appears that actually only Hong Kong and Singapore are authorised for such imports on the condition that only use meat is used that has been imported form a country which is approved for the import of fresh poultry meat to the EU and not meat that is sourced from poultry flocks on their own territory. The animal health requirements are the first step that needs to be complied with before a meat product manufacturing establishment can be listed as EC approved. Hygiene requirements for the slaughterhouse, the processing plant and requirements for monitoring for residues must also be met. Imports can only take place when all the aspects of EU legislation are fulfilled; this explains why certain countries listed on animal health grounds may actually not be exporting to the EU, because they e.g. have not submitted a residue monitoring programme that has obtained EC approval (EC, 2004b).

6.4.7.2 Risk of LPAI/HPAI presence in poultry meat products

In line with the EU definition poultry meat products have undergone some form of treatment (OIE, 2005b; EC, 2004a) and the assessment must therefore be whether or not that treatment is likely to reduce the potential level of viable virus contamination to an acceptable level. Most treatments for poultry products involve heat treatment. Influenza viruses are usually considered to be heat labile. The figures usually quoted are that influenza viruses are inactivated by heat-treating for 15 minutes at 56°C or for 5 minutes at 62°C (King, 1991; Easterday and Beard, 1984). However, there has been no proper study of the inactivation of AI viruses by heat treatment in which inactivation curves have been constructed and Dₜ values determined from which a proper assessment can be made of heat inactivation of AI viruses in meat. Alexander and Manvell (2004) investigated the heat inactivation of Newcastle disease virus in artificially infected meat and calculated D₆₅ as 120 secs, D₇₀ as 82 secs, D₇₄ as 40 secs and D₈₀ as 29 secs. In the absence of any similar data on AI viruses Newcastle disease virus could be considered sufficiently similar to AI viruses that these figures could serve as a guide for estimating the efficacy of heat treatments at reducing the risk of infective poultry meat. However, as mentioned in section 6.4.3.2. other factors such as starting titre of virus, the acceptable level of probability of virus survival and in what quantity of product will need to be assessed.

6.4.8 POULTRY VISCERA

By definition poultry viscera are included within the definition of meat (OIE, 2005b).
6.4.9 MEAL CONTAINING MEAT, FEATHERS OR BONES OF POULTRY
The likelihood that such meal would contain virus is probably low because of the normal processing of meal. However, there should be some assurance that the treatment is likely to reduce the potential level of viable virus contamination to an acceptable level.

6.4.10 FEATHER AND DOWN FROM POULTRY
6.4.10.1 Legislation on imports of feather and down from poultry
According to EU legislation (EC, 2002a) on animal by products not intended for human consumption ‘unprocessed feathers and parts of feathers’ means feathers and parts of feathers that have not been treated with a steam current or by some other method that ensures that no pathogens remain.

Imports of unprocessed feathers may only be authorised if they are securely enclosed in packaging and dry; and sent directly to the technical plant or to an intermediate plant in conditions such that any spread of pathogenic agents is avoided.

6.4.10.2 Risks of LPAI/HPAI transmission by feathers and down from poultry
Feathers and down from poultry especially ducks and geese are used as a filling for duvets, pillows, thermal clothing and other textiles. The feathers being removed from the carcases after slaughter, or harvested from live birds i.e. down feathers from the breasts of geese [this is a common practice in N. China and some other northern Asian countries]. The most likely reason that feather or down would be infective is due to contamination with infective faeces or other body fluids.

However, these plumage products are usually processed in ways that can vary from simple cleaning with soap-containing solutions, with or without disinfectant, to more sophisticated treatment involving thorough cleaning and steam treatment. There is no reason for not subjecting feather products to treatments likely to reduce the potential level of viable virus contamination to an acceptable level and it would seem prudent only to trade in feather products that had undergone such treatment.

6.4.11 PRODUCTS OF POULTRY ORIGIN INTENDED FOR ANIMAL FEEDING, AGRICULTURAL OR INDUSTRIAL USE
The only product in this category that is not covered in the preceding ten sections is poultry faeces. Faeces and litter from poultry houses may be traded as agricultural manure and as such represents a significant risk if from birds infected with AI virus. Even if traded for industrial rather than agricultural purposes, the movement of large quantities of faeces would appear to represent a significant risk.

6.4.12 MEAT OF FEATHERED GAME OR OTHER PRODUCTS FROM BIRDS OTHER THAN POULTRY
Meat of feathered game or other products from birds other than poultry represents a much higher risk than poultry meat as the disease status at slaughter or sourcing is rarely known. The risk is sufficiently high to justify a recommendation that trade in such meat should be limited to products that have been processed to reduce the potential level of viable virus contamination to an acceptable level.
### 6.4.13 Classification of risks

The classification of risks the above products pose for HPAI and LPAI viruses are summarized in the following table. This classification does not represent a qualitative risk assessment, because, as stated in the general comments at 5.5.1. there is a need to take into consideration such factors as whether or not the avian product will reach poultry and whether existing control measures will prevent this occurring.

For LPAI infections the likelihood of LPAI virus being present in the commodities products has been taken into account; in making a detailed risk assessment it will also be necessary to consider the possibility that mutation to HPAI could take place in the LPAI infected birds or flocks. However, at present it is unknown when this is likely to occur.

Table 6-4: The risk of virus being present in avian commodities given HPAI and LPAI infections in the bird or poultry

<table>
<thead>
<tr>
<th>PRODUCT</th>
<th>HPAI</th>
<th>LPAI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eggs for consumption</td>
<td>Very high</td>
<td>Negligible</td>
</tr>
<tr>
<td>Egg products</td>
<td>Will depend on the effectiveness of processing</td>
<td>Negligible</td>
</tr>
<tr>
<td>Semen of poultry</td>
<td>Remote</td>
<td>Remote</td>
</tr>
<tr>
<td>Semen of birds other than poultry</td>
<td>Unknown probably low</td>
<td>Remote</td>
</tr>
<tr>
<td>Fresh meat from poultry</td>
<td>Very high</td>
<td>Remote</td>
</tr>
<tr>
<td>Meal containing poultry products</td>
<td>Will depend on the effectiveness of processing</td>
<td>Negligible</td>
</tr>
<tr>
<td>Poultry viscera</td>
<td>Very high</td>
<td>Negligible(^1)</td>
</tr>
<tr>
<td>Poultry containing meal</td>
<td>Will depend on the effectiveness of processing</td>
<td>Remote</td>
</tr>
<tr>
<td>Feathers and down from poultry</td>
<td>Will depend on the effectiveness of processing</td>
<td>Will depend on the effectiveness of processing</td>
</tr>
<tr>
<td>Commercial manure</td>
<td>Very high</td>
<td>Very high</td>
</tr>
<tr>
<td>Meat from other birds</td>
<td>Very high</td>
<td>High</td>
</tr>
</tbody>
</table>

\(^1\)Poultry viscera are classified as meat (EC, 2004a; OIE, 2005b).
PART II

Prevention and control options, biosecurity, vaccination and virus persistence

7 PREVENTION AND OPTIONS FOR AI CONTROL STRATEGIES

7.1 HYGIENIC/BIOSECURITY MEASURES AT DIFFERENT POULTRY PRODUCTION LEVELS

Bio-security is the first line of defence against an introduction of AI and probably the only defence as long as preventive/prophylactic vaccination of flocks at risk is excluded. Bio-security can be very effective as is demonstrated by the production of specific pathogen free chickens mainly for veterinary vaccine production and diagnostic purposes. Specific pathogen free chickens are raised in filtered air positive pressure houses. Unfortunately these housing systems are expensive and therefore economically unfeasible for intensive poultry farming.

Bio-security comprises two elements: bio-containment and bio-exclusion. Bio-containment means the prevention of virus spread from infected premises and will be dealt with in 7.4. Bio-exclusion refers to measures to exclude infectious agents from uninfected premises.

Good bio-security depends on the formation of a barrier between farms and the outside environment. This sounds simple but can be difficult to implement successfully in practice. Many items and people routinely enter poultry farms, including replacement birds, feed, water, farm workers, consultants, veterinarians, poultry buyers, poultry catchers and vaccination crews. Moreover, it is difficult to exclude free range farming and this type of husbandry is increasing for welfare reasons. In addition, it is impossible to completely prevent the access of vermin to poultry houses.

Bio-exclusion requires the prevention of direct or indirect contacts of wild birds to poultry which is of particular importance because wild birds harbour AI viruses as discussed in 5.2.1. and 6.1. Wild birds may come directly into contact with poultry when the latter are free ranging or indirectly via feed or when open water is used untreated as source for drinking water. Preventing direct or indirect exposure to potentially AI virus-infected birds is very important. Between 1978 and 2000, poultry farmers in Minnesota experienced 108 introductions of LPAI viruses of various haemagglutinin and neuraminidase subtypes from migratory ducks into turkeys (Halvorson, 2002). These Minnesota cases resulted from close direct contact between seasonal migratory juvenile ducks (September to November) with range-reared turkeys, or usage of AI virus contaminated lake or pond water for indoor reared turkeys. Although, the range reared or semi-confinement methods has represented historically less than 5% of turkey production, this minor production method has been the introduction point for LPAI viruses into Minnesota commercial turkeys with disastrous results. With the H5N1 HPAI poultry outbreak and human infections in Hong Kong in 1997, the Minnesota production companies agreed to stop range rearing of turkeys to eliminate introduction of waterfowl LPAI viruses and a potential public relations
problem should an outbreak of LPAI or HPAI occur in Minnesota. As a result, from 1997-2000 only 28 flocks had infections with LPAI viruses, mostly from swine H1N1 influenza virus. Another example is the outbreak in Chile in 2002 on a farm managed at a high bio-security level (Rojas et al. 2002). This outbreak most likely was caused by using drinking water from a pond on the premises frequented by wild birds, demonstrating how easily breaches in bio-security barriers can be overlooked. The outbreak of Newcastle disease in the 80s is another example of breaches in bio-security. This outbreak was caused by infected pigeons that had access to feed storages in the docks of Liverpool (Alexander et al. 1984).

Small flocks of domestic waterfowl (ducks and geese) raised outside could also form a possible route of introduction in particular when they are mixed with other species of domestic poultry and are maintained under common management. Opportunity for AI virus introduction is provided by the tendency of domestic ducks to attract wild ducks. Such introduction can remain unnoticed because infection of ducks even with HPAI mostly does not involve disease. Partial depopulation and restocking often practiced on these kind of premises will enable the maintenance and selective adaptation of influenza viruses. In addition, trading or exchanging of live birds may be responsible for the perpetuation of infection and the spread to other holdings.

In some Member States free ranging of poultry is greatly stimulated for welfare reasons. This trend will most likely increase in the near future throughout the European Union. Where there is a move towards free range farms it is important that farmers are educated in basic principles to discourage wild bird contact with their poultry.

The main goal of bio-exclusion programmes is to manage the risk posed by animals, people and equipment that cross the barriers erected to protect the flock. This is achieved through careful planning and design of farms, use of restrictions, appropriate disinfection and use of protective clothing bound to the farm. Commercial poultry farms should develop a plan in writing that is appropriate to the farm. The plan should specify those that are responsible for its maintenance. Bio-exclusion programmes have been developed in some member states to control campylobacter and salmonella infections.

Still the efficacy of bio-security plans depends greatly on the compliance of the farmer and his personnel. Compliance will be enhanced when they have a basic understanding of the purpose of the measures. Thus effective bio-security demands that nearly every aspect of farm management is controlled causing farmers to feel in a straitjacket of rules. Thus, breaches of bio-security do likely occur no matter how stringently the measures are implemented. The risk of bio-security breaches is higher the more people and items routinely enter the farm.

In some regions the live bird market system forms a potential risk for the transmission of viruses from one species to the other. In particular, those markets at which free-ranged poultry is traded form a risk for virus spreading. Live birds markets often exist for ethnical and cultural reasons and are more prevalent in Asia and in some cities in the United States. Live-birds markets that exist in Europe are much smaller and probably do not form a great risk.
7.2 STRUCTURE AND ORGANISATION OF PRODUCTION SYSTEMS

The poultry industry has a complex structure that varies from country to country and region to region and its organisation is related to consumer requirements in terms of type and seasonal availability of products. In general the poultry industry is vertical or semi-vertical organised with primary breeders on the top and production birds at the bottom. Between the two there are about 5 generations. At the bottom of the chain, independently managed hatcheries are linking the different generations that are bred separately.

In the layer industry, pullets are most often raised till 3 weeks before the start of lay and then transferred to layer houses. The units of these production columns are geographically intermingled. A peculiarity of the layer industry is that multi-age farms represent a large portion of the establishments. This basically means that replacements are present on the same farm as the adult birds and this determines poor biosecurity as the all-in-all-out system is not implemented and thus the farm, under normal circumstances cannot be cleaned and disinfected properly. In addition, poultry is delivered to an ever decreasing number of slaughter houses that are sometimes located at large distances. Moreover, feed delivery, egg traders, poultry workers, veterinarians, service crews, collection of carcasses provide daily visits often governed by production rules rather than by biosafety issues. These daily visits form the horizontal contacts between production columns. Spread of avian influenza virus is facilitated by the high integration of the poultry industry. Any form of compartmentalisation of the poultry industry and its supply companies will reduce the spread of AI. Compartmentalisation will particularly be effective to reduce the spread of LPAI because it tends to spread imperceptibly.

7.2.1 SYNDROME SURVEILLANCE SYSTEM TO DETECT AI IN AN EARLY STAGE

Bio-security is the first line of defence against all AI viruses (Beard, 1981). However, occasionally AI is introduced into a commercial poultry population. When it does, the level of standard bio-security is mostly inadequate and an enhanced level of bio-security is necessary to control the spread. In general, introductions of either LPAI or HPAI strains, into poultry are not notified immediately after introduction. This occurs in the case of HPAI because of the length of flock incubation time i.e. the time between the flock being infectious and the onset of clinical signs. Moreover, even after the appearance of the first clinical signs the disease may remain unreported, because some of the clinical signs are not pathognomonic (Elbers et al., 2004a). With reference to LPAI, a similar situation may exist, or it may occur that initial stages of viral circulation are completely asymptomatic - particularly in chickens. Therefore, practitioners that are confronted with an unclear clinical condition will be inclined to think of other diseases (Elbers, et al. 2004b), and submission of clinical samples originating from such farms is therefore crucial to the early identification of infection. The period between introduction of the virus and notification is indicated as the high-risk period (HRP). During the Dutch outbreak, in the HRP the virus could almost spread freely between flocks, since insufficient biosecurity measures were in place (Stegeman et al., 2004).

The HRP can be reduced by implementing syndrome surveillance programmes compelling poultry farmers to report disease problems to their veterinarians. The veterinarian should assess the cause of the problems and perform differential diagnosis
to exclude AI. The clinical signs used to decide whether to send material to the
laboratory will determine the sensitivity and specificity of detecting AI. Criteria that
include non specific signs like decrease in water and food consumption, increase in
daily mortality, respiratory problems and decrease in egg production will increase
sensitivity but will result in increased costs (Elbers et al., 2004b). The use of highly
sensitive on-site tests detecting viral antigen can be very useful in these cases (Akey,
2003). However, it is important that the limits of the currently available tests are
considered in interpreting the results. Rapid antigen tests do not attain the sensitivity of
rapid tests like the real time reverse transcriptase (RT) PCR (SCAHAW, 2003).

Diagnosis and confirmation of AI in the field may be obtained within a short time,
currently within 6-12 h) by using the RT-PCR (Spackman et al., 2002; Cattoli et al.,
2004). The advantage of this test is that it exhibits a sensitivity which is equal or higher
than the standard virus isolation test and results are obtainable in less than a day. On the
other hand, the RT-PCR test must be performed within the setting of a laboratory, and
in addition it is not yet an official test as it is not yet mentioned in Annex III of Council

From the experience gathered within and outside the EU, it is clear that farmers are
reluctant to report suspicions of AI. There are several reasons for this including the
education level of the farmer, underestimation of the problem, type of contract with
private veterinarian, fear of bankruptcy or of unjustified restriction policies. It is
however crucial to avoid secondary spread from the index case that the owners/farmers
and veterinarians understand the importance of excluding AI in a very early phase.
Investigations should be performed to find ways to increase the readiness to quickly
exclude HPAI in a very early stage using appropriate laboratory tests and to understand
the importance of notification and active collaboration with the public veterinary
services.

Syndrome surveillance programmes are aimed at monitoring and identifying HPAI at
early stages of HPAI infections which are not characterised by pathognomonic signs
and clinically non overt LPAI infections. In this way notification to the competent
authorities at an early stage will contribute to the adequate management of the crisis.

7.3 SEROLOGICAL MONITORING SYSTEMS

Serological monitoring systems are not suited to detect an infected flock at an early
stage firstly because it takes at least 7-10 days for the flock to begin seroconverting
and secondly because the testing frequency required obtaining such result is unpractical and
not cost-effective. The aim of serological monitoring systems is to prove that a certain
region is free of disease. A second aim of serological monitoring programme is to detect
subclinical LPAI infections in case syndrome surveillance programmes are not
implemented or as a complementary measure to syndrome surveillance programmes.

‘Free from AI infection’ cannot be guaranteed in absolute terms because this would
require frequent testing of all flocks within that region. Therefore, “free” implies that
the prevalence of the disease is below the level specified in the monitoring programme.
The level depends on the size of the poultry population, the sample size used at the
moment of sampling and the characteristics of the test used.

However, surveillance by random sampling with statistical assessment of prevalence is
a static approach, often assuming a stable (endemic) situation, whereas the spread of
infection is a dynamic process. Consequently, population-dynamic aspects should also be considered when developing surveillance programmes aiming at the detection of subclinical AI infections. For this purpose mathematical models can be used to incorporate the dynamic behaviour of infectious diseases in the development of a surveillance programme. (Anderson and May, 1991; Becker, 1989; Diekman and Heesterbeek, 2000; May and Anderson, 1987). These types of models have been applied in previous epidemics and showed to be valuable for understanding the course of the epidemic. They also provided parameters for the development of future control and monitoring programmes (see e.g. Bouma et al., 2003; Graat et al., 2001; Keeling et al., 2001; Klinkenberg et al., 2005; Stegeman et al., 1999).

The data required to develop such mathematical models can only be obtained by analysing data collected in recent and future AI outbreaks. A standardised format to collect the proper data is recommended.

OIE has recently developed Surveillance Guidelines for AI adopted during the 73rd General Session in May 2005 (OIE, 2005d).

7.4 RETROSPECTIVE ANALYSIS OF CONTROL OPTIONS IMPLEMENTED DURING MAJOR AI OUTBREAKS

Once AI is confirmed competent authorities should take appropriate measures to eradicate the disease. This requires the complete isolation of a suspected flock (biocontainment measure) followed by its prompt culling after infection is confirmed in the laboratory. However, in the face of insufficient biosecurity measures, virus might spread rapidly already during the HRP, and measures can not be restricted to the infected flock but have to be extended to flocks at risk of becoming infected. For instance, analysing the dynamics of the outbreak in the Netherlands in 2003 showed that, due to insufficient biosecurity in the poultry industry 25-30 farms were already infected before restriction measures were issued. Earlier outbreaks have demonstrated that most secondary infected farms were located at distances of 1-3 kms and therefore to get ahead of virus spread pre-emptive culling of all susceptible farms within this region was required. Obviously, this approach has a different outcome on the basis of the poultry density in the area. In regions with a low poultry density only a few farms at risk will be depopulated, but in regions with a high density of poultry farms this approach will lead to massive culling of millions of mainly healthy birds (Capua and Marangon, 2003).

The HPAI in the intensive layer industry in Pennsylvania in 1984-1983 was the first outbreak which required the culling of about 17 000 000 birds to control it. The outbreak resulted particularly difficult to extinguish due to the extensive circulation of the LPAI virus prior to mutation to HPAI, which hampered and delayed diagnostic interventions. During this outbreak infected farms and farms with known exposure were culled and the control took about 5 months (Utterback, 1984). A LPAI outbreak of in Virginia and North Carolina in 2002 was controlled in a similar way, although on site tests instead of clinical signs and RT-PCR and virus isolation were used to detect and confirm infected farms. The control of the Virginia outbreak took 4 months and 5 000 000 birds’ lives (Akey, 2003). During HPAI outbreaks in Italy (1999-2000), the Netherlands (2003) and British Colombia, Canada (2004), in addition to infected and exposed farms, the farms at risk of becoming infected were pre-emptively culled; i.e. the farms in a region with a radius 1 up to 3 km. Still, the outbreak in Italy took 4.5 months (Capua et al. 2003) and
caused the death of 13 million birds, in the Netherlands 2 months and 30 000 000 birds and Canada 3 months and 19 000 000 birds (Capua and Alexander, 2004a). At first sight the different control strategies between these outbreaks did not result in any outbreak being significantly better controlled than the other. However, conclusions on the efficacy of different control measures can only be based on the proper analysis of the epidemics using data on the total number of susceptible and infectious farms, species reared, geographic localisation of the farms, characteristics of the strain and estimated incubation time.

European Community control measures in the case of an HPAI outbreak are laid down in Council Directive 92/40/EEC (EC, 1992a). This directive describes minimal requirements to be taken. Member States are always able to implement additional measures. In the Netherlands the stand-still for movements was extended to the whole country instead of protection zones of 3 km and surveillance zones of 10 km as indicated in the directive. The restrictions were only gradually lifted after surveillance had demonstrated that it was safe to do so. Stand-still and zoning reduces the number of contacts that are believed to form the principle routes of transmission of avian influenza (Utterback, 1984; Akey, 2003; Marangon and Capua, 2005). The greatest threat of spread of avian influenza viruses is by mechanical transfer of infective faeces, in which virus may be present at concentrations as high as 107 infectious particles/gram and that may survive for longer periods depending on the temperature (Utterback, 1984) Primary mode of transmission therefore is considered to be movement of people (caretakers, farm-owners and staff, trucks and drivers moving birds or delivering food, and artificial inseminators), fomites or birds. In the outbreak in Italy in 1999-2000 the origin of infection could be attributed to: movement of animals (1.0%), indirect contacts at the time of loading for slaughter of female turkeys (8.5%), neighbourhood spread (within 1 km radius) (26.2%), lorries for the transport of feedstuff, litter and dead carcasses (21.3%), and other indirect contacts (e.g. exchange of manpower, machinery, equipment (9.4%) (Marangon and Capua, 2005).

Analysis of data collected during the Dutch outbreak suggests that the number of farms infected by on average one farm was reduced from 5-6 to about 1 by issuing a stand still and the culling of infected farms only (Stegeman et al., 2004). Unfortunately, the effect of the contribution of individual measures such as movement restriction and pre-emptive culling could not be assessed by certainty, because measures came to an effect at the same time or in a period of time of insufficient duration for appropriate analysis. Still, because culling started rather late, results suggested that the stand still contributed more than the pre-emptive culling in decreasing the transmission rate. Movement restrictions also reduced the spread of AI in Pennsylvania dramatically (Fitchner, 1987). Moreover, the results of the analyses raised doubts on the efficacy of measures, such as the culling of all poultry in large areas, zoning, and creation of poultry-free buffer zones to control HPAI in densely populated poultry areas. This is in particular true if the virus is already widespread in the area (Capua and Marangon, 2000; Stegeman et al., 2004).

Due to the organisational, structural and functional diversities of the poultry industry in different parts of the world, some modes of spread that appear unlikely or negligible in one situation may instead represent a possible means of spread under other conditions. No evidence of air borne transmission was obtained during the HPAI outbreaks in Italy (Capua et al., 2000a) and Pennsylvania in 1984 (Utterback, 1984). In Italy virus sometimes did not spread between flocks that were separated by a fence only, in which
direct contact between birds was not possible. Air borne spread was suggested as a possible transmission mode between nearby farms during the recent outbreak in the Netherlands (Landman and Schrier, 2004) and Canada (CFIA, 2005). In the evaluation of outbreaks in Pennsylvania (Utterback, 1984) and the Netherlands, veterinarians reported that birds showing clinical signs first were observed nearby the ventilation inlet instead of near the entrance door. In the only attempt to measure airborne transmission infectious virus could be detected more frequently in air samples collected at distances of 5 m than at distance of 45 m from infected farms also demonstrating the dilution effect (Beard, et al., 1984, Brugh and Johnson, 1987). However, many farms use wood shavings or sawdust as bedding materials. These bedding materials contain extremely fine and light sawdust particles which can be readily become airborne if the bedding is disturbed. Large exhaust fans of modern poultry houses can eliminate high volumes of air and airborne dust that could be readily taken up by the intakes of adjacent farms. Future analysis of transmission dynamics of the Dutch and Canadian outbreaks is required to establish whether airborne route did contribute in significant way to the spread between farms.

People were always considered to be able to carry the virus between farms only mechanically. In attempts to isolate virus from nasal swabs obtained from people involved in depopulation of poultry during the Pennsylvania outbreak HPAI H5N2 was isolated from 2 of 110 nasal swabs that were collected from people immediately after leaving the poultry houses. Virus could not be isolated from the same 2 individuals 12 hours later. (Wood et al, 1984). In addition, no or few blood samples collected among poultry workers during a number of outbreaks were positive in the past. During many outbreak attempts to show AIV infections in humans either failed or no evidence for secondary transmission between humans were obtained (Capua and Alexander 2004b). During the Dutch outbreak, however, not only an unexpectedly high number of transmissions were observed of avian influenza A virus subtype H7N7 to people directly involved in handling infected poultry, but evidence for person-to-person transmission was also noted (Koopmans et al., 2004). About 50% of 500 sera of people exposed to infected poultry reacted positive with H7N7 virus in a modified haemagglutination inhibition test. In 63% of the cases, people did not have direct contact with infected poultry but were only exposed to infected asymptomatic humans. Infected people may be contagious for other humans and for poultry. People with symptoms did shed virus for more than 3 days which normally is used as the period in which no contact with other uninfected poultry is allowed. Since the antibodies against H7N7 could not be detected in haemagglutination inhibition tests using the conventional protocol, the prevalence of AI infections among people exposed to infected poultry may have been under estimated in the past. Although the observation may be limited to characteristics of the H7N7 virus, it must have consequence for the way HPAI outbreaks have to be controlled in the future. The use of protective equipment thus is not only essential to prevent disease in humans and to reduce the risk of starting a pandemic but also essential to prevent virus spread between flocks by poultry workers, veterinarians and vaccination crews.
7.5 ROLE OF HOBBY FLOCKS
The flock size and the contact structure between flocks are important variables that determine the transmission rate. Hobby poultry and pet birds are kept in small sized flocks and as such do shed much less virus than large commercial flocks. Moreover the contacts between hobby birds and commercial flocks should be accidental. It has not been established whether hobby or pet birds have contributed greatly to spread during the recent Dutch and Italian AI epidemics. However, it appears that once infection has reached the industrial circuit it is mainly spread via the direct and indirect contacts related to the functional organisation of the integrated system. As contacts between this system and amateurial poultry farming are accidental and for other (ethical, emotional etc.) reasons pre-emptive culling of hobby birds is most probably unnecessary and definitely undesirable. In addition such a policy will inevitably determine the illegal movement of birds out of the restriction zones. Increased bio-security and surveillance of hobby birds is most likely to be more effective and to have a more acceptable impact on the public opinion.

7.6 EDUCATION
Education is an essential component of AI prevention, control or eradication strategies. This involves providing information to the industry concerning the biology of avian influenza viruses, how the virus is introduced and spread between farms, and bio-security guidelines to prevent introduction of AI virus onto a farm (bio-exclusion practices) and to prevent secondary spread of AI (bio-containment practices). The education process must involve all employees who are provided information on what avian influenza is, how it is transmitted, identification and elimination of behaviours that put the farm at risk for AI introduction (e.g. owning backyard poultry, working or visiting other poultry farms). In addition basic bio-security measures to protect poultry (e.g. farm dedicated clothing and footwear left on the farm at the end of the workday, employee showering facilities before entry on the farm, cleaning and disinfection equipment used between farms), and consequences for the company and the employees jobs if an AI outbreak occurs should be part of the training programme.

Risk communication is essential between companies when an AI infected premise is identified.

8 VACCINATION AGAINST AI

8.1 INTRODUCTION
Vaccination has not historically been an option for the control of avian influenza infections of the H5 and H7 subtypes (AI). This situation arose because, until 1998, AI was a sporadic disease with only 18 outbreaks reported in domestic poultry since 1959. The total number of birds involved in all outbreaks over this 40-year period was approximately 23 million (Capua and Mutinelli, 2001a). The greatest contribution in number of dead birds to this figure was the Pennsylvania 1983-1984 outbreak in which over 16 million birds were affected. Over this period, the Pennsylvania outbreak represents the exception to the rule: a single outbreak with very serious consequences among outbreaks of minor or no relevance.
From 1999 onwards, AI infections cannot be considered sporadic. Including estimations of the ongoing Asian H5N1 epidemic, in five years over 200 million birds have been affected. Some outbreaks have maintained the characteristic of minor relevance but others, such as the Italian 1999-2000, the Dutch 2003, the Canadian 2004 and the Asian 2003-2004 have lead to devastating consequences for the poultry industry, negative repercussions on public opinion and in some cases created significant human health issues (Capua and Alexander, 2004a).

With retrospectively analyses, two main factors have been identified as crucial in the development of such major epidemics. Possibly, the most important point is the presence of areas of high poultry population densities (DPPA) and/or in situations in which poverty is widespread. In these situations it is impossible to impede the spread of AI only with restriction, biosecurity and stamping out.

In addition, some areas appear to be particularly at risk for AI due to the presence of migratory flyways over DPPAs - or - some countries are at higher risk of introduction from “reservoir environments” (e.g. live bird markets in the USA or Hong Kong).

For the reasons listed above, the concepts of emergency vaccination and of prophylactic vaccination have made their way in several countries worldwide. The outcome of these campaigns has been different, ranging from eradication to endemicity of the field virus. It is therefore imperative, since AI now represents not only an animal health issue but also a human health threat, that vaccination is used as a tool to support eradication and not as a tool to obtain endemicity.

### 8.2 Vaccines and Vaccination

An extensive document on vaccines that are available currently has been recently issued (SCAHAW, 2003). The major conclusions of this document are that currently only conventional oil emulsion inactivated products are suitable for field use. The vaccination system must allow the DIVA (Differentiating Infected from Vaccinated Animals) concept, either through appropriate serological tests or through unvaccinated sentinels left in the shed. This is primarily to deal appropriately with the flock if it becomes infected. For this reason, vaccination can only be seen as part of a control strategy based on biosecurity, monitoring, controlled marketing and stamping out.

Controlled marketing of slaughter birds is an alternative measure that can be applied as a means of reducing the economic impact of massive culling of birds in LPAI control. It has been estimated that about 4 weeks after the introduction of the virus in a flock, the infective cycle of the virus has come to an end, and the amount of virus shed from the convalescent birds is negligible. From this point in time the risk of spreading virus in the environment is low, and therefore sending these birds to a slaughter house under controlled conditions including rigorous bio-security measures during loading, transport and slaughter under official control represents a useful compromise in managing LPAI outbreaks in for which compensation is not foreseen.

It would seem appropriate to recommend that if vaccination is pursued it must enable the DIVA concept.

At present, EU legislation is under revision (EC, 2005a) and therefore it is not possible to make up-to date comments on regulatory aspects of vaccination. However due to the
significant impact that AI has had in the past few years and the recent adoption of OIE rules (OIE, 2005a) including the possibility of vaccination, it appears that a more flexible approach to the use of vaccination is envisaged. One of the recommendations of a conference on the “Material and immaterial costs of animal disease control” in Brussels on 15/16 December 2004 was: “Vaccination should be accepted as one of the regular options for the control of animal disease”. (EC, 2004e)

8.3 EMERGENCY VACCINATION

As explained above and in section 7.4, notwithstanding an efficient veterinary infrastructure and modern diagnostic systems, AI outbreaks in densely populated poultry areas are impossible to control without the culling of millions of mainly healthy birds.

In recent times in the EU, H7 vaccines have been used as emergency vaccination programmes that have obtained the approval of the European Commission and of Member States.

Pivotal work on this control option has been carried out in Italy, and the application of the “DIVA” vaccination strategy has resulted in the approval of use of vaccination as an additional tool for the eradication of two subsequent epidemics of AI (H7N1 and H7N3) without massive killing animals. Vaccination was used to complement restriction measures already in place and was integrated with an intensive monitoring programme, targeted at identifying viral circulation in the area (Capua et al, 2004a). In 2000, for the first time ever heterologous vaccination was used in the field against an H7 virus as a “natural marker vaccine” and subsequently in Hong Kong vaccination using a DIVA strategy was successful in preventing further spreading of HPAI to neighbouring farms in face of an outbreak of HPAI H5N1 (Ellis et al., 2004).

In the laboratory, vaccination resulted in increased resistance of birds to infection and in reduced shedding (Capua et al., 2004b), and the combination of these two effects, associated to a monitoring system and a territorial strategy was successful in achieving eradication in the field. In addition the use of such “DIVA” system enabled the continuation of international trade of poultry products (Capua et al., 2004a; Marangon and Capua, 2005).

For obvious reasons there is only limited experimental evidence on the use of vaccination and this lack of theoretical knowledge poses questions that do not have an answer. However, as proved in Italy, vaccination may be efficient in reducing virus spread. Recent experimental data indicate that SPF chickens that underwent a two-shot immunisation programme are resistant to challenge with $10^6$ logs of virus. This represents a 100 fold increase in resistance compared to the unvaccinated controls (Capua, 2005 unpublished). Moreover, the transmission of virus in SPF chickens vaccinated with a ‘heterologous’ vaccine did reduce virus transmission to a level that on average one bird infected less than one other bird which indicates that an outbreak within a flock will be extinguished and therefore this flock will not represent a source of infection for other flocks. Although at 7 days after vaccination birds still shed enough virus, to infect naive contact birds, contact infection of naïve birds was no longer observed at 14 days after vaccination. Difference in the level of transmission was observed between different vaccines demonstrating the importance of the vaccine
choice. From the results, it is predicted however that vaccination should induce immunity in at least 99% of the birds to obtain enough herd immunity to prevent a major outbreak (Goot et al, unpublished).

However, although an appropriate vaccination programme, based on the “DIVA” strategy has shown to be a powerful tool in supporting eradication, vaccination is not formally regulated under the current Council Directive 92/40/EEC (EC, 1992a) does foresee the use of emergency vaccination, but the consequences on movements of birds and products are not detailed. This regulatory deficiency results in the imposing of unjustified trade barriers that generate uncertainties in the decision making process on whether to vaccinate or not. In the face of an outbreak, exporting countries have not had the legislative support to implement vaccination and continue to trade, and this has resulted, for example, in opting for a stamping out policy, resulting in 30 000 000 dead birds in the Netherlands in 2003, with great costs for the industry and taxpayer.

There is field evidence, however that if vaccination is not complemented with biosecurity and surveillance, this may result in making the infection endemic. The efficacy of the vaccination programme and the implementation of adequate biosecurity measures should be under official control. Similarly, it is imperative that an intense monitoring programme to detect infected flocks by using a DIVA system is implemented. This enables to detect the circulation of the field virus and to appropriately manage infected flocks which should be slaughtered under controlled conditions or culled under official supervision.

Inadequate biosecurity or vaccination practices can lead to transmission between flocks and selection of variants that exhibit antigenic drift. Antigenic drift of viruses belonging to the Mexico lineage resulting in less homology to the vaccine strain has been recently described (Lee et al, 2004). It clearly appears that the extensive and uncontrolled use of vaccine in Mexico has resulted in the emergence of antigenic variants that escape the immune response induced by the vaccine. Mexico has been practicing vaccination since the HPAI outbreak in 1994 without applying the DIVA principle. However, no HPAI virus has been reported following the implementation of the vaccination campaign although LPAI viruses continue to circulate. Conversely, a similar approach in Pakistan has led to the continued presence of HPAI virus for over 10 years (Naeem and Siddique, 2005).

8.4 EMERGENCY VACCINATION PROGRAMMES

Emergency vaccination therefore could represent an alternative to pre-emptive culling in reducing the susceptibility of healthy flocks at risk by reducing the transmission rate (see 7.4.). The effectiveness of such policy depends on variables such as the density of poultry flocks in the area, the level of biosecurity and integration of the industry and on characteristics of the virus strain involved. In addition logistical problems such as vaccine availability and adequate administration must be kept in mind. The dimension of the vaccination zone in case of a ring vaccination depends not only on the transmission rate but also on the initial spread during the high risk period (HRP) and on the functional interconnections of the infected zone. Based on the preliminary analyses of the Dutch outbreak a zone up to 35 km around the index case should have been vaccinated. Still, the risk of spread of the virus by fomites over a longer distance
remains. It is generally accepted though that AI cannot be controlled when interventions strategies are based on geography only.

Another issue of relevance is that of the time interval necessary to obtain protective immunity. It is estimated that a minimum of 7-10 days are necessary for the initial development of the immune response, and over two weeks may be necessary to have protective antibody levels. This implies that the decision making process must be fast-tracked and vaccine must be available for immediate use. In the face of an emergency however, uncontrolled movement of vaccination crews will result in spreading of infection rather than in a means of controlling its spread. For this reason contingency plans that include decision making patterns under different scenarios should be envisaged.

It also appears rather clear that it is not possible to lay down general conditions for vaccination programmes which can be applied in all Member States. Although the industrial system often has overlapping points there are major differences in animal densities, species reared, husbandry systems and genetic profile across the EU. In all cases, as stated in Council Directive 92/40/EEC (EC, 1992a) all Member States should have prepared contingency plans. For the current revision of the Directive the creation of vaccine banks is envisaged and contingency plans should include different scenarios of vaccination campaigns, which should allow quick and easy access to high quality vaccine banks in case of an emergency.

Analysing past and future outbreaks should provide data that is required to design appropriate vaccination programmes in the different situations. In particular, these data are required to determine intervention strategies both in case of LP- and HPAI outbreaks.

8.5 VACCINATION VERSUS CULLING

The financial losses due to AI epidemics can be huge for the commercial and the public sectors, especially once AI viruses are introduced in areas that have high bird densities. In these areas, the high density of poultry farms, the organisation of the poultry production sector and the difficulties in applying rigorous biosecurity measures increase the risk of major AI epidemics. These epidemics are difficult to control despite the enforcement of draconian eradication measures based on the depopulation of farms that are infected, suspected of being infected, suspected of being contaminated or located in areas at risk of infection (buffer zones).

Unpublished field evidence indicates that despite the enforcement of massive stamping out and depopulation measures, both LPAI and HPAI viruses can persist undetected in domestic reservoirs or in the wild, re-emerge and rapidly spread after repopulation of poultry farms in previously affected areas. This means that frequent incursions or the re-emergence of AI viruses in densely populated areas can contribute to make these areas unsustainable in the long term.

Furthermore, the killing of large numbers of birds and the destruction of carcasses is being increasingly perceived by the public as unacceptable due to ethical, social and environmental reasons. Stamping out policies have also led to very high costs and
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The enforcement of stamping out measures on AI affected farms can be effectively applied in areas with a low poultry density, especially if first infected sites are promptly detected and adequately managed. If this is the case, the depopulation of infected premises can allow the rapid eradication of the disease, at acceptable costs for both the producers and the public.

Taking into account the high risk of major AI epidemics once AI viruses are introduced in areas with high poultry densities, alternatives to the application of stamping out in case of LP or HP avian influenza outbreaks in such areas should be pursued.

Controlled marketing of slaughter birds is an alternative measure that could be applied to depopulate LPAI virus infected holdings, to reduce the economic impact of massive culling of birds in LPAI control. About 4 weeks after the introduction of the virus in a flock, there is a significant decrease of viral shedding by the already fully immunized birds. At that time the risk of sending these birds to a slaughter house for controlled marketing could be considered as acceptable, provided that rigorous bio-security measures during loading, transport and slaughter procedures are enforced under official control. In comparison with non vaccinated/exposed birds, the amount of virus excreted by vaccinated/exposed birds is significantly reduced both in quantity and duration. Thus vaccination could decrease the risk of a further spread of the infection related with the application of controlled marketing of birds originating from LPAI affected holdings.

In densely populated areas control strategies based on a combination of stamping out/controlled marketing, restriction and emergency vaccination policies should also be implemented to maximize eradication efforts.

An accurate evaluation of the cost/benefit ratio of all the available eradication options should be carried out in peace time, to address in due times the actions to be developed in an emergency situation. This evaluation could produce different scenarios in different geographical areas taking into account: pathogenicity of the AI virus (LP/HP) possibly involved, poultry densities, bird species, type and organisation of the poultry sector, rendering and slaughtering capacities, organisation of veterinary services and the impact on trade.

8.6 PROPHYLACTIC VACCINATION

Prophylactic vaccination for viruses of the H5 and H7 subtypes is a completely innovative concept. This is primarily due to the fact that only recently situations which may find in this policy a cost-effective solution have been pinpointed and identified.

Prophylactic vaccination should increase the resistance of birds and, in case of virus introduction, reduce levels of viral shedding - at the same levels of biosecurity. It should be perceived as a tool to maximise biosecurity measures when a high risk of exposure exists. Ultimately it should result in preventing the index case, or alternatively in reducing the number of secondary outbreaks and thus minimising the negative aspects of animal welfare and economic losses.

Prophylactic vaccination for H5 and H7 subtype viruses is presently not allowed under the current EU legislation. Prophylactic vaccination should only be considered when
there is circumstantial evidence that country/area compartment is at risk of infection. Risk of infection may be subdivided into two categories:

1) High risk of infection with either H5 or H7 subtype (e.g. from migratory birds)
2) Risk of infection with a known subtype (e.g. LBM in the USA, Asian countries with H5N1)

In the first case a bivalent (H5 and H7) vaccination programme should be implemented. Italy has recently implemented such a programme in the DPPA at risk of infection (EC 2004d). In the second case, a monovalent (either H5 or H7) programme is also acceptable.

To reduce the risk that DIVA strategy can not be used vaccine strains that combine H5 and H7 with rare neuraminidase types should be used, either by using natural isolates with these characteristics or by generating prototype strains in the laboratory by reassortment or by using reverse genetics systems.

The rationale behind the use of prophylactic vaccination is that it should be able to generate a minimal level of protective immunity in the target population. The immune response may be boosted if there is evidence of the introduction of a field virus.

It should be clear that the population to be vaccinated must be clearly identified. Different countries have different production systems, types of birds, species, rearing systems etc. and a general rule applicable to every situation does not exist.

In addition prophylactic vaccination should be carried out as long as the risk of infection exists.

8.7 ECONOMIC ASPECTS OF VACCINATION

An accurate evaluation of the cost/benefit ratio of a vaccination programme should be carried out prior to the implementation of the campaign. This should include not only the costs of the vaccine and of vaccine administration but also the costs of monitoring, surveillance, laboratory testing and all other activities connected to vaccination.

Although the application of a DIVA vaccination strategy enables both the monitoring of the epidemiological situation in a vaccinated population and the prompt identification of infected/exposed birds and flocks, the main constraint on the use of AI vaccines is represented by the possible impact on trade.

Movement and trade restrictions to be enforced on live poultry and poultry products originating from areas where vaccination is applied should be modulated on the basis of a relevant risk assessment.

For obvious reasons the trade implications will be clarified when the EU Commission define guidelines for the use of vaccination in the documents of their competence.

8.8 THE PUBLIC OPINION

Public perception of the management of animal diseases has become a relevant issue. This is primarily due to the fact that media greatly emphasize the “brutal” aspect of culling of significant numbers of animals. In addition, animal welfare has become more important and often has a greater impact on public opinion than other animal health issues.
Vaccination of animals against epizootic diseases has a series of added values for the public which are summarised below:

a) the veterinary services are active in “preventing” the disease  
b) animal welfare is preserved  
c) mass culling is avoided

In addition, in the specific case of the “H5N1 bird flu crisis” in Asia, prophylactic vaccination against the H5 subtype may be perceived by the public as a means of safeguarding the consumer

9 PERSISTENCE OF AIV VIRUSES AND DISINFECTION METHODS

9.1 SURVIVAL OF AVIAN INFLUENZA VIRUSES IN FAECES

Survival of avian influenza viruses in faeces is likely to be influenced by: strain of virus, type of faeces i.e. hosts from which it came and the physical properties of the faeces and the temperature at which it is held. One of the problems in assessing the duration of retention of infectivity is that very little properly co-ordinated scientific evaluation has been done and sometimes observations are almost anecdotal. For example Fitchner (1987) states that during the 1983-85 Pennsylvania outbreak of HPAI, caused by H5N2 virus, under natural field conditions virus was still detectable in infective wet manure after 105 days but doesn’t state the ambient temperature, although it may be assumed that there was a considerable range. Similarly discussing the same outbreak Utterback (1984) concluded that virus may be present at concentrations as high as $10^7$ infectious particles/gram and may survive for longer than 44 days. Beard et al. (1984) held wet faeces from naturally-infected hens (same H5N2 virus) at 4°C and could detect infectivity >35 days (maximum time tested), but at 25°C infectivity could only be detected for two days. In a slightly more scientific approach Lu et al., (2003a) artificially infected SPF hens with a LPAI H7N2 virus (about $10^7$ EID50/gram) and showed that at 4°C they could still detect infectious virus in their faeces at 23 days (last day tested); at ambient temperature (15-20°C) they could detect virus at 19 days but not 23 and at 37°C they could detect virus at 14 but not 16 days. Interestingly when they used “field chicken” manure the times were at ambient temperature (15-20°C) they could detect virus at 4 days but not 6 and at 37°C they could detect virus at 12 hours but not 36 hours (Lu et al., 2003a).

Tyres, footwear, clothing, farm equipment, crates and cages, interiors of transport vehicles can be contaminated with infected faeces in which the virus levels for both LPAI and HPAI can be very high, if HPAI or LPAI infections are present in the serviced flocks and adequate precautions of cleaning and disinfection are not taken.
### Table 9-1: Reports on the survival of AIV in faeces

<table>
<thead>
<tr>
<th>Virus</th>
<th>Conditions</th>
<th>Starting material</th>
<th>Temperature</th>
<th>Conclusion</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPAI H5N2</td>
<td>Field</td>
<td>wet manure</td>
<td>ambient – not stated</td>
<td>infectivity detectable after 105 days</td>
<td>Fitchner, 1987</td>
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<td>[Pa USA]</td>
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<tr>
<td>HPAI H5N2</td>
<td>Field</td>
<td>faeces titre up to $10^7$ EID50</td>
<td>not stated</td>
<td>infectivity detectable after 44 days</td>
<td>Utterback, 1984</td>
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<tr>
<td>[Pa USA]</td>
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<tr>
<td>HPAI H5N2</td>
<td>Experimental</td>
<td>infective faeces from infected chickens</td>
<td>4°C</td>
<td>infectivity detectable after 35 days [max. time tested]</td>
<td>Beard, et al., 1984</td>
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<td>[Pa USA]</td>
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<tr>
<td>HPAI H5N2</td>
<td>Experimental</td>
<td>infective faeces from infected chickens</td>
<td>25°C</td>
<td>infectivity detectable up to 2 days</td>
<td>Beard et al., 1984</td>
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<td>[Pa USA]</td>
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<tr>
<td>LPAI H7N2</td>
<td>Experimental</td>
<td>artificially infected SPF chicken faeces ~$10^7$ EID50/g</td>
<td>4°C</td>
<td>infectivity detectable up to 23 days [last time tested]</td>
<td>Lu et al., 2003a</td>
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<td>[USA]</td>
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<td>LPAI H7N2</td>
<td>Experimental</td>
<td>artificially infected SPF chicken faeces ~$10^7$ EID50/g</td>
<td>ambient [15-20°C]</td>
<td>infectivity detectable at 19 but not 23 days</td>
<td>Lu et al., 2003a</td>
</tr>
<tr>
<td>[USA]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LPAI H7N2</td>
<td>Experimental</td>
<td>artificially infected SPF chicken faeces ~$10^7$ EID50/g</td>
<td>37°C</td>
<td>infectivity detectable at 14 but not 16 days</td>
<td>Lu et al., 2003a</td>
</tr>
<tr>
<td>[USA]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LPAI H7N2</td>
<td>Experimental</td>
<td>artificially infected field chicken faeces ~$10^7$ EID50/g</td>
<td>ambient [15-20°C]</td>
<td>infectivity detectable at 4 but not 6 days</td>
<td>Lu et al., 2003a</td>
</tr>
<tr>
<td>[USA]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LPAI H7N2</td>
<td>Experimental</td>
<td>artificially infected field chicken faeces ~$10^7$ EID50/g</td>
<td>37°C</td>
<td>infectivity detectable at 12 hours but not 36 hours</td>
<td>Lu et al., 2003a</td>
</tr>
</tbody>
</table>
9.2 SURVIVAL OF AVIAN INFLUENZA VIRUSES IN WATER

Webster et al. (1978) commented on the high levels of virus excreted by ducks and in a simple batch test experiment with A/duck/Memphis/546/74 (H3N6) showed that an initial virus concentration of $1 \times 10^{8.1}$ EID50/ml in lake water was reduced to $1 \times 10^{4.3}$ EID50/ml after 32 days at 0°C [D0 = 8.4 days] and that no virus could be detected after 4-7 days at 22°C. Stallknecht et al. (1990b) in a more detailed series of experiments using 5 LPAI viruses estimated that from an initial concentration of $10^6$ TCID50/ml of virus in distilled water infectivity was retained for up to 207 days at 17°C and 102 days at 28°C. Their results allowed the calculation of Dt values at the two temperatures used; these varied from 21-34.5 days at 17°C and 5-17 days at 28°C (Table 9-2) depending on the strain of virus.

**Table 9-2: Survival of AIV in water**

<table>
<thead>
<tr>
<th>Virus (all LPAI)</th>
<th>Water type</th>
<th>Temp. °C</th>
<th>Dt in days¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>A/duck/Memphis/546/74 (H3N6)²</td>
<td>lake</td>
<td>0</td>
<td>8.4</td>
</tr>
<tr>
<td>A/gadwall/LA/17G/87 (H3N8)</td>
<td>distilled</td>
<td>17</td>
<td>32</td>
</tr>
<tr>
<td>A/blue-winged teal/LA/44B/87 (H4N6)</td>
<td>distilled</td>
<td>17</td>
<td>34.5</td>
</tr>
<tr>
<td>A/mottled/duck/LA/38M/87 (H6N2)</td>
<td>distilled</td>
<td>17</td>
<td>29</td>
</tr>
<tr>
<td>A/green-winged teal/169GW/88 (H10N7)</td>
<td>distilled</td>
<td>17</td>
<td>24</td>
</tr>
<tr>
<td>A/blue-winged teal/LA/188B/87 (H12N5)</td>
<td>distilled</td>
<td>17</td>
<td>21</td>
</tr>
<tr>
<td>A/gadwall/LA/17G/87 (H3N8)</td>
<td>distilled</td>
<td>28</td>
<td>11</td>
</tr>
<tr>
<td>A/blue-winged teal/LA/44B/87 (H4N6)</td>
<td>distilled</td>
<td>28</td>
<td>12.3</td>
</tr>
<tr>
<td>A/mottled/duck/LA/38M/87 (H6N2)</td>
<td>distilled</td>
<td>28</td>
<td>16.3</td>
</tr>
<tr>
<td>A/green-winged teal/169GW/88 (H10N7)</td>
<td>distilled</td>
<td>28</td>
<td>5</td>
</tr>
<tr>
<td>A/blue-winged teal/LA/188B/87 (H12N5)</td>
<td>distilled</td>
<td>28</td>
<td>17</td>
</tr>
</tbody>
</table>

¹Approximate values determined from data presented.

²Webster et al. (1978) all others Stallknecht et al. (1990b). The Dt value is the time taken to reduce the virus infectivity titre by 90% [i.e. by 1 log10] at the specified temperature.

9.3 METHODS FOR MANURE TREATMENT

9.3.1 TREATMENT OF INFECTED LITTER

Litter obtained from infected premises represents a significant source of virus and needs to be managed appropriately. It may be either buried in pits, and subsequently covered with a layer of at least 40 centimetres of soil, or alternatively may be accumulated in heaps inside or outside the house. In the latter case particular attention must be paid to covering the heaps with a resistant sheet of plastic, which must be secured to the ground either with bricks or, more frequently with vehicle tires. This procedure must be implemented to prevent access to infected material to wild birds, rats and other animals, apart from ensuring that the plastic cover could not be lifted by the wind.
9.3.2 METHODS OF VERIFYING THE MATURATION OF LITTER HEAPS

Internal temperature and pH of the heaps should be measured with the aid of dedicated thermometers and pH-meters. The probe of the instrument is to be inserted at least 60 centimetres inside the heap, following removal of a cone of litter with an appropriate sampler. An internal temperature ranging between 42 and 55°C for 42 days is sufficient to allow virus inactivation.

In certain cases, virus isolation attempts may be performed on selected litter samples (obtained with the sampler) and as an additional measure, sentinel SPF birds can be introduced in the cleaned and disinfected houses for a period of time ranging between 15-30 days (Capua and Mutinelli, 2001a).

9.4 DISINFECTANTS FOR INACTIVATION OF AVIAN INFLUENZA VIRUSES

9.4.1 PREPARATORY WORK AND PRINCIPLES

Preliminary cleaning work is invariably needed before any chemical disinfectants are used. The natural processes of time, dehydration, warm temperature and sunlight will greatly assist the decontamination operation and should be considered in planning. A hot, dry, sunny day will cause rapid natural inactivation of an agent like Avian influenza virus whereas cold, damp, overcast conditions will assist its persistence. Simple cleaning of surfaces by brushing with a detergent solution is effective in removing contaminating viruses and is fundamental for achieving effective chemical decontamination. Most disinfectants have reduced effectiveness in the presence of fat, grease and organic dirt. Every effort should be made to remove such coverings from all surfaces to be decontaminated. Hot water and steam are effective in cleaning many cracks and crevices where pathogens are likely to linger. The insides of pipe work can often only be cleaned effectively by steam. If applied long enough for surfaces to approach 100°C, the interior pipe work will be effectively decontaminated. The choice of disinfectant depends on the method of application and how an adequate wet contact time is to be maintained.

It is most important to remember that, after having cleaned the surface, the time of contact with the disinfectant is of critical importance. For most applications, disinfectant must flood the surface and keep it thoroughly wet for at least 10 minutes.

9.4.2 ESTIMATION OF QUANTITIES REQUIRED

The amount of decontaminating agent necessary for particular jobs varies considerably. For a polished, non-porous floor, 100 ml of disinfectant/chemical applied per square meter is probably sufficient. However, for porous surfaces such as concrete or wood, the volume may need to be doubled or tripled. Generalisations are not useful as application of liquids to ceilings or vertical walls cannot be well controlled.

Use of chemicals may be subject to limited use because of environmental consideration or because they are considered hazardous chemicals. Therefore commercial available disinfectants have to be licensed in the member states. Often products contain more than one class of disinfectant. Licensing includes proven efficacy to the claimed range of micro-organism. Member States should include lists of effective disinfectants that have been licensed when this is required by the member state.
Table 9.3: **Disinfectants active against AI viruses.** From Capua and Mutinelli (2001a) and AUSVETPLAN (2000)

<table>
<thead>
<tr>
<th>Chemical product</th>
<th>Concentration</th>
<th>Recommended contact time</th>
<th>Recommended use</th>
<th>Limitations</th>
<th>Other information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium hypochlorite</td>
<td>2%</td>
<td>10 - 30 minutes</td>
<td>Equipment</td>
<td>Presence of organic material, corrosive for many metals</td>
<td>Reduced stability at temperatures above 15°C</td>
</tr>
<tr>
<td>Quaternary ammonium salts</td>
<td>4%</td>
<td>10 minutes</td>
<td>Walls, floors, ceiling equipment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postassium peroxomonosulphate+ sulphamic acid+ sodiumalkylbenzenesulphonate</td>
<td>2%</td>
<td>10 minutes</td>
<td>Walls, floors, ceiling equipment</td>
<td>corrosive</td>
<td></td>
</tr>
<tr>
<td>Calcium hydroxide</td>
<td>3% w/v</td>
<td>10 minutes</td>
<td>Walls, floors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cresolic acid</td>
<td>2.2% w/v</td>
<td>10 minutes</td>
<td>Floors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Synthetic phenols</td>
<td>2% w/v</td>
<td>10 minutes</td>
<td>Floors</td>
<td>Causes burns; absorbed through skin</td>
<td></td>
</tr>
<tr>
<td>Formaline +permanganate</td>
<td></td>
<td></td>
<td></td>
<td>Fumigation</td>
<td>Toxic gas</td>
</tr>
<tr>
<td>Calcium hypochlorite</td>
<td>2-3% w/v</td>
<td>10-30 minutes</td>
<td></td>
<td>Presence of organic material</td>
<td>Reduced stability at temperatures above 15°C</td>
</tr>
<tr>
<td>Sodium hydoxide</td>
<td>2% w/v</td>
<td>10 minutes</td>
<td></td>
<td>Do not use in presence of Aluminium and derived alloys</td>
<td></td>
</tr>
<tr>
<td>Sodium Carbonate (washing soda)</td>
<td>10% w/v</td>
<td>30 minutes</td>
<td>In the presence of high concentration of organic material</td>
<td>Add 0.1% sodium silicate to protect aluminium equipment</td>
<td></td>
</tr>
<tr>
<td>Hydrochloric acid</td>
<td>2% w/v</td>
<td>10 minutes</td>
<td></td>
<td>highly corrosive</td>
<td>Do not use for disinfecting metals and concrete</td>
</tr>
<tr>
<td>Citric Acid</td>
<td>0.2% w/v</td>
<td>30 minutes</td>
<td>Clothing and body</td>
<td></td>
<td>check pH</td>
</tr>
<tr>
<td>Glutaraldehyde</td>
<td>2% w/v</td>
<td>10-30 minutes</td>
<td>Excellent disinfectant</td>
<td>Allergy, use protective clothing</td>
<td>Decomposes above pH 9</td>
</tr>
</tbody>
</table>

Note: Several of these compounds are licensed in EU member states and are commercially available as ready-to-use products.
9.4.3 PRECAUTIONS WHEN USING DISINFECTANTS
Chemicals usually kill microorganisms by toxic reactions; however effective disinfectants are often hazardous for animal (and human) tissues as well. Virtually all disinfectants have to be used with care to avoid occupational injuries or health problems.
PART III

10 WELFARE ASPECTS OF AI

10.1 EFFECTS OF DISEASE ON INFECTED ANIMALS

All diseases cause some degree of poor welfare in the infected animals (Broom, 1988; 2004; Broom and Corke, 2002; Broom and Kirkden, 2004). Birds die from some strains of avian influenza and welfare is very poor during the latter stages of the progression of the infection towards death. Chickens and turkeys with HPAI show a variety of signs, associated with poor welfare, which are detailed by Alexander (1996).

“Clinical signs which may be associated with high mortality are cessation of egg-laying, respiratory signs, rales, excessive lachrymation, sinusitis, oedema of the head and face, subcutaneous haemorrhage with cyanosis of the skin, particularly of the head and wattles, and diarrhoea.” As a consequence, effective measures which are taken to minimise the spread of avian influenza will have substantial beneficial effects on chicken welfare. Where a control method, such as killing for disease control purposes or vaccination, is used it will be possible to assess the risk of poor welfare in the birds which are the subject of the activity and the likely benefit to these and other birds as a consequence of the measures taken.

10.2 WELFARE ASSOCIATED WITH KILLING FOR DISEASE CONTROL PURPOSES

10.2.1 INTRODUCTION

Killing for disease control purposes can result in large scale improvement in bird welfare because birds do not become diseased. Humane killing methods are available for those birds which are to be killed. In this section, principles are described, practical experience is summarised and then effects on welfare are described with references to a recent EFSA Report (EFSA, 2004)

10.2.2 HUMANE KILLING METHODS

If birds have to be killed as a preventive measure to reduce the risk of spread of avian influenza, a method of killing is chosen. This could result in a death which involves poor welfare prior to death for a substantial time. However, other methods of killing for disease control purposes are humane in that they involve little or no poor welfare because the effects on the animal are not severe or because any period of poor welfare is very brief.

Some of the mechanisms for causing death act by firstly causing loss of consciousness, followed by cardiac or respiratory arrest, leading to complete loss of brain function. Three key mechanisms for causing death are:

- Hypoxia - causes unconsciousness and depression of the respiratory centre in the central nervous system, followed by complete loss of brain function
- Depression of neurones necessary for life functions - depression of the central nervous system

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nervous system respiratory centre leading to cardiac arrest
• Physical disruption of the brain

There is a variety of methods available causing death of the animals. Some methods are usable for small flocks or individual birds, but are not applicable for the use during avian influenza epidemics.

10.2.3 SOME PRACTICAL ISSUES IN CULLING OPERATIONS

When killing operations are carried out, the welfare of the birds is better if professional personnel handle the animals and a well-trained veterinarian monitors the process. Preparatory and specific training is important to bird welfare and operator safety. Factors which need to be considered by the person responsible for planning the operation include:
• the human risk of infection,
• the potential danger for further spreading of the disease by operating personnel, veterinarians in the field and vehicles and equipment such as trucks, cranes, trailers, forklifts and culling machines. Rapid response is crucial in bringing avian influenza under control; the less people and material involved, the better spreading can be brought under control and the smaller the risk of further spreading in flocks that are not infected.

10.2.3.1 The culling methods used during the AI crisis situation in the Netherlands and Belgium

During the recent outbreak of avian influenza in The Netherlands and Belgium (as well as one case in Germany) a variety of different methods were used to kill poultry. There was not just one method that was suitable for each situation. The choice of the culling method depended on the housing and the numbers of birds. 66% of all the birds were culled by the method of poultry-house gassing. This method was found possible only for birds in large buildings. Problems which occurred with poultry house gassings are described below. Smaller groups of chickens, geese, ducks and turkeys housed in half open houses could not be culled by poultry-house gassing. Many farms were not suitable for poultry-house gassing, due to the fact that the animals were housed in cages or the animals were housed in half-open stables. Among the widely used methods were culling the animals by electrocution and culling the birds in containers filled with at least 70% CO2. (Gerritzen et al, 2004).

10.2.4 CULLING OPTIONS AVAILABLE

10.2.4.1 Culling by hand in plastic bags

A widely used method in the South East Asian Region is culling the animals by hand by putting them in plastic bags and burning the bags. This method is not only ineffective; it is also inefficient and with high risks of further spread of the virus. This method is still in use but is very bad from an animal welfare point of view.
10.2.4.2 Gassing (inhalation of agents)

Inhalation agents require the use of a sealed chamber such as commercial waste disposal bins that can be adequately sealed. The supervising veterinarian should be able to see inside the sealed container to ensure that the poultry are killed quickly and effectively. Large containers provide the opportunity to kill large numbers of birds quickly. Whenever birds are progressively added to large sealed containers, the veterinary supervisor needs to make sure that all birds inside the container are dead before others are added. Gassing in buildings or inappropriately designed containers always has the risk that some birds will be exposed to low concentrations of gas or no gas at all. If the gas is aversive there will be poor welfare throughout the exposure. If bird carcasses are disposed of, for example by burning, but some of the birds are conscious, welfare will be very poor at that time.

Gases vary in their effects on bird welfare. Some are not detectable when inhaled whilst some are aversive. Some have effects after inhalation which are painful or which otherwise involve poor welfare. The speed with which birds are rendered unconscious varies, as does the risk of recovery before death.

Birds of different species vary in their responses to gasses. In particular, birds adapted for diving, such as ducks, are able to hold their breath for longer than birds not thus adapted so may take much longer to become unconscious. If the gas inhaled is aversive, the period of poor welfare will be much longer in ducks than it would be in chickens.

**Carbon dioxide (CO₂)**

CO₂ is a colourless, virtually odourless, non-flammable, non-explosive gas that presents minimal hazards to operators. When inhaled it has an irritant effect and produces a choking sensation. All mammals and birds which have been tested show aversion to this gas (EFSA, 2004). CO₂ is heavier than air, so it will accumulate in the lower areas of the location, building or container, where birds are placed.

CO₂ kills poultry by depression of the central nervous system leading to death by hypoxia. Two common CO₂ methods are currently in use:

**a) Poultry-house gassing:**

A commercial poultry-house is completely sealed off up to 2 metres from the ground and slowly filled with compressed and vaporised gas, which was CO₂ in 2003. The gas under high pressure is pumped into the stable, slowly filling the room. Practical experience with several operations showed that after 35 minutes the birds started to die, the operation is completed after two to three hours. Poultry-house gassing has been used for large highly advanced technique, using mobile evaporation equipment and specialised operators. Poultry-house gassing is applicable for large quantities of broilers, kept within mechanically ventilated buildings.

The advantage is that large flocks of birds could be culled within a limited amount of time. Also limited training was needed for the collection of dead birds.
One disadvantage of this method is that high additional costs are involved. A second is that the process cannot be stopped, after putting the operation into action. Thirdly, the introduction of gas to a building will always be gradual so many birds will be exposed to a low concentration of gas for a period. Whilst the delay between first contact with the gas and unconsciousness is known for poultry introduced to a container full of gas, the delays which occur in poultry-house gassing will vary greatly but are not known. If it is an aversive gas, such as CO\(_2\), the welfare of the birds will be poor for a period which may last for many minutes. As described above, the birds may be in contact with the gas for 30 minutes. Poultry-house gassing has been used mostly for housed broilers and young turkeys.

b) Wheelie-bin container gassing:

This system was developed by HKI-Wageningen and widely used during the AI outbreak in Europe, in 2003. The birds were culled in modified wheelie-bin containers. The birds were introduced into an atmosphere of 70% CO\(_2\), within the wheelie bin using compressed gas from clustered cylinders. The birds were directly exposed to the CO\(_2\), leading to stunning within 30 seconds and culling after one minute. Practical experience has shown that if CO\(_2\) is decanted from compressed gas cylinders too quickly, it will lead to freezing of the gas in the cylinders, pipelines or of the regulators. Also, considerably more CO\(_2\) is required for birds with long necks (such as ducks) to ensure a sufficient amount of CO\(_2\) in the containers to kill these birds.

An advantage of the wheelie-bin container method is that it is not limited to quantities or type of poultry (up to a weight of 20 kg each). The culling process can be stopped at any moment, in case problems within the culling process occur.

One disadvantage of this system is the high costs of operation and the limited possibilities due to availability of sufficient CO\(_2\) cylinders, packed in clusters of 12 containers each with the minimum weight of 1,200 kg per package, extra transportation of gas cylinders and forklift trucks for the transportation of the wheelie-bins and cylinder clusters. A second disadvantage is the poor welfare of birds during the time from entry to the gas to unconsciousness. There would be no such disadvantage if argon were used.

The use of both CO\(_2\) methods is limited to areas with sufficient supply of this gas and under circumstances in which systems for the transportation of gas, gas trucks, gas equipment mobile evaporators as well as forklift trucks are available. This means that the use of CO\(_2\) gas is only possible in areas within the direct delivery distance, so near major cities.

The use of carbon dioxide has found to be very slow and often ineffectual for ducks and similar problems are likely with geese. In one practical experience, most ducks were still alive after 3.5 hours in what was intended to be 40% carbon dioxide.

**Carbon monoxide (CO)**

Carbon monoxide is colourless, odourless, non-flammable and non-explosive gas. It causes a fatal hypoxemia when levels reach 4% to 6%. It can be obtained in compressed
gas cylinders. Carbon monoxide is toxic to humans, so there are significant health and safety issues that need to be considered. A well ventilated area is needed. The CO system was used during the AI outbreak in The Netherlands. The use of the CO method is limited to areas with sufficient supply of this gas. Poultry-house gassing with CO is complex and dangerous for the operating personnel.

**Nitrogen and Argon**

Nitrogen and argon are colourless, odourless, non-explosive, non-flammable gases that present minimal hazards to operators. Both are available in compressed gas cylinders and are used in some commercial poultry slaughterhouses. They cause death by hypoxia, but are only effective when oxygen levels in the sealed container/chamber are reduced to less than 2%. Argon is a heavy gas like CO₂ so can easily be introduced into a container where it will go to the bottom and displace air or nitrogen.

**Hydrogen Cyanide**

Hydrogen cyanide is a very quick and effective culling agent. It is reported that poultry may exhibit convulsions prior to death. It acts by causing paralysis of the respiratory centre. Hydrogen cyanide is very toxic to humans and is no longer commercially available.

**Gaseous anaesthetics**

Gaseous anaesthetics can rapidly induce anaesthesia leading to unconsciousness and, when applied at overdose levels, effectively kill birds by fatal depression of the central nervous system respiratory centre. Examples of gaseous anaesthetics include halothane, methoxyfluorane and isofluorane. They are far more costly than the agents described above. In most situations there are legal considerations regarding their access and use. These agents can induce anaesthesia in humans. They are not generally considered suitable for culling large numbers of poultry.

**10.2.4.3 Injectable anaesthetics**

Injectable anaesthetics depress the central nervous system leading to loss of consciousness, anaesthesia, apnoea, depression of the respiratory centre and terminal cardiac arrest. As is the case with the gaseous anaesthetics described above, there are legal considerations concerning their access and use. There are a number of agents; the most commonly used are the barbiturate drugs such as sodium pentobarbital.

They are effective agents when used at overdose levels to humanely kill poultry. Intravenous application is the preferred method; however in smaller birds (without prominent veins) intraperitoneal application is satisfactory. Intracardiac application must only be attempted when the poultry are fully restrained. The use of injectable anaesthetics may be limited to a veterinarian or appropriately trained persons under the supervision of a veterinarian. The use of this method is restricted to small flocks of poultry.
10.2.4.4 Physical Methods

Electrocution

Electrocution is used in many poultry slaughterhouses (water bath stunning systems) to stun birds prior to the severing of the major blood vessels of the neck, which leads to death by exsanguination.

If electrocution is to be used as the method of culling birds in an emergency disease outbreak, they will not die unless there is sufficient, constant current (amperage) to cause instantaneous and simultaneous destruction of the central nervous system and cardiac arrest. Using equipment to electrocute birds may be hazardous to personnel.

During the recent outbreak of Avian Influenza in The Netherlands, especially designed stand alone electrocution equipment was developed. The use of this equipment proved to be extremely effective and efficient to process 2,500 to 10,000 birds per hour. The electrocution equipment is available in different sizes, suitable for different species of birds. One mobile is specially designed for the use in rural areas. It included a generator and is mobile. It can be placed directly on any spot within the culling area. The machine can process 2,500 to 4,000 birds per hour and is operated by one local operator. On site training of the operator takes approximately one day.

The use of mobile water-bath stunning equipment will be subject to the same problems for bird welfare as those which occur in poultry slaughterhouses with shackling lines and water-bath stunning. It is stressful to birds to be caught by humans. When the birds are carried to the stunning or killing apparatus, the method of carrying is usually stressful. Hanging by the legs on a shackling line is stressful. Some birds may lift their heads and are not stunned or killed. If a shackling line is used, welfare will never be very good and will usually be considerably worse than it would be if killed by an inert gas in a properly designed container.

Electrocution using properly designed metal electrodes could be carried out without causing a substantial degree of poor welfare.

Cervical dislocation

Cervical dislocation can be an effective means of humanely culling poultry, resulting in the loss of central nervous system stimulation of respiration and heart-beat. However, if the technique is not performed correctly, birds may not be killed without there being poor welfare.

This procedure may not be aesthetically pleasing. Cervical dislocation can be performed manually or using mechanical device, such as a Burdizzo castrator. Whenever a large numbers of chickens are to be destroyed, this may not be the method of choice, but may be the preferred option for long-necked birds such as ducks in small numbers.
Decapitation
Decapitation results in a rapid loss of consciousness and blood loss. It is an effective method for culling poultry, but is not aesthetically pleasing. If sharp instruments are not used, injury and pain may result.

Mechanical Maceration
Mechanical maceration results in instantaneous death. It has been applied in some sectors of the poultry industry for destruction of young birds, e.g. surplus day-old male chickens. This process is not aesthetically pleasing but with especially designed equipment in good working order it is humane for small birds such as day-old-chicks.

10.2.5 Effects of Methods of Killing for Disease Control on Poultry Welfare

Methods for killing for disease control purposes were described in an EU report (SVC, 1997).

However the most recent report and opinion are those of the EFSA Scientific Panel on Animal Health and Welfare in 2004. This report and opinion provide information both on normal methods for stunning and slaughter of poultry, which will may not be appropriate for birds which have or may have AI, and also on methods for killing for disease control purposes. There are accounts in the Report of the problems associated with the use of the following methods which were not recommended:

a) Killing without prior stunning, for example putting birds in plastic bags and burning the bags as mentioned above as occurring in some countries;

b) Injection of individual birds with any chemical except barbiturates;

c) The following methods of gassing:
   - gassing with hydrogen cyanide;
   - gassing with impure carbon monoxide;
   - gassing with high concentrations of carbon dioxide, i.e. more than 30%.

Some forms of poultry killer/captive bolt stunner were recommended but only if: death can be confirmed in each animal, there is proper training of personnel and for small batches of animals. The problems associated with electrocution using a water-bath were described and are summarised in 10.2.4.4 above. However, electrocution using a portable water-bath system is a practicable method which is better than the worst methods.
mentioned above as not recommended. If potentially infected birds are removed from a poultry house for killing, the risk of disease transmission is substantially increased.

The use of neck dislocation was recommended but only if: limited to small batches of poultry weighing less than 3.0 kg, performed in one stretch, carried out with care to ensure complete dislocation of the neck so that the vertebral column is severed from the cranium, death can be confirmed in each animal, and there is proper training of the personnel.

Decapitation was recommended but only if: limited to small batches of poultry, performed in one pull and cut involving separation of the head from the body.

The use of carbon monoxide was recommended provided that the birds are put into a chamber of pure gas, the concentration is 4 – 6 % for a duration of at least six minutes and there are proper safeguards for human operators.

The use of carbon dioxide was recommended provided that the birds are put into a chamber containing not more than 30% carbon dioxide in an inert gas and not more than 2% oxygen.

The killing or birds by placing them in chambers containing appropriate inert gas mixtures such as argon with not more than 2% oxygen was recommended.
11 REFERENCES


EC (1979). Council Decision 79/542/EEC of 21 December 1976 drawing up a list of third countries or parts of third countries, and laying down animal and public health and veterinary certification conditions, for importation into the Community of certain live animals and their fresh meat (OJ No L 146, 14.06.79 p. 15).


EC (2000a). Commission Decision 2000/585/EC of 7 September 2000 drawing up a list of third countries from which Member States authorise imports of rabbit meat and certain wildand farmed game meat, and laying down the animal and public health and the veterinary certification conditions for such imports (OJ. L 251, 6.10.2000, p.1).

which Member States authorise imports of fresh poultry meat (OJ L 258, 12.10.2000, p. 9).


http://europa.eu.int/comm/food/animal/diseases/strategy/index_en.htm


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Scientific report on animal health and welfare aspects of Avian Influenza


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van der Goot, J., Koch G., de Jong, M.C.M., van Boven, M. (2004). Quantification of the transmission characteristics of highly pathogenic avian influenza A virus (H7N7) in vaccinated and unvaccinated chickens. submitted


12 MEMBERS OF THE WORKING GROUP

Ilaria Capua (chair)
Laboratorio Virologia
Istituto Zooprofilattico Sperimentale (IZS) delle Venezie
Via Romea 14/A
I-35020 Legnaro, Padova, Italy

Dennis J. Alexander
Department of Environment, Food and Rural Affairs
Virology Department
Veterinary Laboratories Agency,
Woodham Lane, Addlestone, Surrey
KT15 3NB, United Kingdom

Donald M. Broom
Dept. of Clinical Veterinary Medicine
University of Cambridge
Madingley Road
UK- Cambridge CB3 0ES, United Kingdom

Véronique Jestin
AFSSA - Laboratoire d'études et de recherches avicoles et porcines
BP53 Rue des Fusillés
F-22440 Ploufragan, France

Poul H. Jorgensen
Department of Poultry Diseases
Danish Veterinary Laboratory
Hangoevej 2, DK -8200 Aarhus N, Denmark

Guus Koch
Central Institute for Animal Disease Control Lelystad(CIDC-Lelystad)
Department of Virology
Post-box 2004
8203 AA Lelystad, The Netherlands

Stefano Marangon
Istituto Zooprofilattico Sperimentale delle Venezie
Viale dell'Università, 10
I-35020 Legnaro (PD), Italy

Maurice Pensaert
Fonds voor Wetenschappelijk onderzoek
Ghent, Belgium
Scientific report on animal health and welfare aspects of Avian Influenza

Bjorn Olsen  
Biology and environmental Sciences  
Institute for Zoonotic Ecology and Epidemiology  
Kalmar University  
SE-391 82 Kalmar, Sweden

Albert D.M.E. Osterhaus  
Erasmus University  
Faculteit der genee Instituut voor virologie  
28 Dr Molewaterplein 3000 DR Rotterdam, The Netherlands

Alejandro Schudel  
World Organisation for Animal Health (OIE)  
12 rue de Prony  
F-75017 Paris, France

Marion Wooldridge  
Centre for Epidemiology and Risk Analysis  
Veterinary Laboratories Agency  
Weybridge, United Kingdom

13 ACKNOWLEDGEMENTS

Annemarie Bouma & Arjan J. Stegeman  
Department of Farm Animal Health, Yalelaan 7  
Postbus 80151; 3508 TD Utrecht, The Netherlands

Michiel van Boven  
Quantitative Veterinary Epidemiology, Animal Sciences Group  
Wageningen University and Research Centre  
PO Box 65, 8200AB Lelystad; the Netherlands

Harm Kiezebrink  
HKI-Wageningen bv  
P.O. box 311, 8160 AH EPE, The Netherlands

Ricardo Jorge Soares Magalhaes  
Royal Veterinary College  
University of London  
London, United Kingdom

Vincent Martin  
AGAH-FAO Animal Production and Health Service, FAO  
Viale delle Terme di Caracalla  
Rome 00100, Italy
14 ANNEXES

14.1 ANNEX I: STUDIES ON THE RISK OF INTRODUCTION OF AI BY WILD BIRDS

14.1.1 SPECIES OF MIGRATORY BIRDS AT RISK OF INTRODUCTION OF AIV

Even if in accordance with current knowledge H5/H7 viruses are those responsible for outbreaks and epizootics in poultry, it is important to remember that all known pandemics in humans had their origin in other subtypes of LPAI viruses. Since from this historical point of view, this is not an isolated animal health and agricultural problem, and although it is not directly linked to this risk assessment (focused on the risk of introduction and further spread of avian influenza), a complete inventory of the AIV detected in wild birds is given. It may further help assessing the capacity of other subtypes to cross the species barrier since there is a possibility that virtually all AIV presenting as low or even non-pathogenic in their natural reservoir may become highly pathogenic in a different host.

a) Prevalence of infection in wild birds

Influenza viruses have been shown to infect a great variety of birds (for reviews see Alexander 1982a, 2000; Hinshaw et al., 1980a; Lvov, 1978), including free-living birds, captive caged birds, domestic ducks, chickens, turkeys and other domestic poultry. Influenza viruses have been isolated from avian species representing most of the major Families of wild birds throughout the world. Reviews of these surveillance studies (Lvov, 1978; Hinshaw et al., 1980a; Stallknecht and Shane, 1988; Stallknecht, 1998) list 90 avian species, covering 22 different Families and 12 Orders, from which virus has been isolated (Table 13-2). The actual number of susceptible species is likely to be much greater, and to some extent this is demonstrated by the recorded susceptibility of a wide variety of birds in laboratory experiments or investigations of captive birds in quarantine or in ornamental collections (Alexander, 1982b; Panigrahy and Senne, 1998). However, it is important to differentiate between susceptible birds and true reservoirs. Virtually any bird may be susceptible but the main question is whether they are true reservoirs, able to spread the virus to wild or domestic birds. Virus isolations from other wild birds have been completely overshadowed by the number, variety and widespread distribution of influenza viruses in waterfowl. In the surveys listed by Stallknecht and Shane (1988) a total of 21 318 samples from a number of species resulted in the isolation of 2 317 (10.9%) viruses. However, 14 303 of these samples were from birds of the Order Anseriformes which yielded 2 173 (15.2%) isolates. The next highest isolation rates were 2.9% and 2.2% from the Passeriformes and Charadriiformes, respectively; but these compare with an overall isolation rate of 2.1% from all birds other than ducks and geese. In fact the overall isolation rate falls to 2.2% if birds of the genus Anas are excluded and 6.2% if those from mallard (Anas platyrhynchos) are ignored. One study (Sharp et al., 1993), suggests that waterfowl do not act as a reservoir for all avian influenza viruses. It seems likely that part of the influenza gene pool is maintained in shorebirds and gulls, from which the predominant number of isolated influenza viruses are of a different subtype to those isolated from ducks (Kawaoka et al., 1988).
b) Recent European studies

The former published data on the distribution of AIV in wild birds is both supportive and partly in contrast with results from North America. In a recent investigation in Europe (Fouchier et al., 2003) where more than 15 000 birds representing 252 species were sampled (Table 13-1) shows that in the group “other birds” representing mainly Passeriformes (sedentary, autumn and spring migratory passerines) and Charadriiformes (shorebirds/waders during autumn migration) none were AIV positive.

Table 14.1.1: AIV surveillance with M-protein gene RT-PCR in faecal samples from wild birds 1999-2003

<table>
<thead>
<tr>
<th>Species</th>
<th>N Tested</th>
<th>N PCR+</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ducks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mallard (Prevalence in Holland)</td>
<td>3549</td>
<td>280</td>
<td>(7.9)</td>
</tr>
<tr>
<td>Teal</td>
<td>470</td>
<td>16</td>
<td>(3.4)</td>
</tr>
<tr>
<td>Wigeon</td>
<td>835</td>
<td>4</td>
<td>(0.5)</td>
</tr>
<tr>
<td>Shoveler</td>
<td>87</td>
<td>1</td>
<td>(1.1)</td>
</tr>
<tr>
<td>Shelduck, Eider, Gadwall, Pintail, etc.</td>
<td>282</td>
<td>0</td>
<td>(0  )</td>
</tr>
<tr>
<td>Geese</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White-fronted</td>
<td>504</td>
<td>13</td>
<td>(2.6)</td>
</tr>
<tr>
<td>Greylag</td>
<td>274</td>
<td>7</td>
<td>(2.6)</td>
</tr>
<tr>
<td>Brent, Barnacle, Bean, Egyptian</td>
<td>849</td>
<td>0</td>
<td>(0  )</td>
</tr>
<tr>
<td>Gulls</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black-headed</td>
<td>457</td>
<td>10</td>
<td>(2.2)</td>
</tr>
<tr>
<td>Common, Herring, Black-backed, Kittiwake</td>
<td>1149</td>
<td>0</td>
<td>(0  )</td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guillemot</td>
<td>27</td>
<td>3</td>
<td>(11.1)</td>
</tr>
<tr>
<td>Others Other birds</td>
<td>6574</td>
<td>0</td>
<td>(0  )</td>
</tr>
<tr>
<td></td>
<td>15057</td>
<td>345</td>
<td>(2.3)</td>
</tr>
</tbody>
</table>

2004-10-15 ~ 50 % can be isolated in embryonated eggs

To investigate the prevalence and ecology of AIV in Western Palearctic migrating ducks, a duck funnel trap is operating since the autumn 2002 at the Ottenby Bird Observatory on the Swedish Baltic Sea island Öland where mainly mallards (*Anas platyrhyncos*) are caught. After ringing and collection of biometrical data every individual is sampled for AIV by faecal swabbing. From August to December 2002, 896 birds were caught and sampled. The overall AIV prevalence in these migratory ducks was approximately 20%. Thirteen of the 15 described HA-types where found in the 200 positive samples. Of those 12 were H7N7 and 16 H5N2. Data from the Netherlands show that the 2003 epizootic in poultry caused by the AIV H7N7 was most likely was of wild bird origin since the ancestors of the virus were found in mallards (Fouchier et al., 2004). It is noteworthy that the population passing Ottenby on their southwest bound migration is wintering in Western Europe, including the Netherlands (Figure 13-1) and that H7N7 viruses closely related to the H7N7 causing HPAI in the Netherlands were detected in wild duck populations passing through Ottenby three months prior to the start of the epizootic in the Netherlands.
Further, the overall AIV prevalence in Dutch mallards was 7.8% (table 1 and 2), lower than found in the migrating ducks at Ottenby. This is in line with previous data from North America (Hinshaw et al., 1980a).

The role of Western Palearctic shorebirds (Charidriiformes) in the perpetuation and dispersal of AIV in Europe is unclear. During the autumn migration 1999, 2000 shorebirds were sampled for AIV at Ottenby Bird Observatory, Sweden. None of them carried AIV. This is in contrast to studies in the North America were up to 15% of shorebirds have been found to carry all AIV subtypes (except H16) (Kawaoka et al., 1988). The reason for this discrepancy in carrier rate between closely related species in the old and new world is unknown. In an ongoing Swedish and Dutch study spring migrating shorebirds are caught and faecal samples are collected. This study will clarify if spring migrating shorebirds are of any importance in the enzootic cycle of AIV in Western Palearctic bird populations.

However, in the wader study from 1999, ten Black-headed Gulls (Larus ridibundus) (BHG) were sampled and five of them were positive for AIV. One BHG carried the previously known and gull-associated AIV H13, but in four gulls a new subtype H16 was found. The H16 was, in analogy, with the subtypes found in ducks, low pathogenic (Fouchier et al., 2005). A subsequent Swedish study in 2000 of more than 1000 BHG in colonies transecting from Malmö to Umeå did not show any AIV positive birds (Fouchier et al, 2005, submitted). Analysis of recovery data for BHG caught at Ottenby from 1-15 of August showed that their recruitment area is east from Ottenby, i.e. Southern Finland, the Baltic States and Belarus. This is an indication that certain of AIV subtypes are endemic in certain species of wild birds in Europe, whereas others are regularly introduced.

For the first time AIV has been isolated from Guillemots (Uria aalge) in the Western Palearctic (Wallensten et al., 2005). These birds belong to a population that is effectively isolated from the Pacific Guillemot population in which Sazonov et al. (1977) demonstrated AIV H3N2. The screening method in the latter study was egg inoculation, increasing the risk of cross contamination from the H3N2 Hong Kong virus circulating in the human population (and the laboratories) at that time. Guillemots banded in the auk colony on Bonden in the northern Baltic Sea in July 2000 were screened for AIV. Three out of 26 sampled birds tested positive by PCR and one of those was characterized as H2N6, a subtype not found in either poultry or humans. This indicates that AIV has a taxonomically wider reservoir range than previously known. It is unclear whether or not this eventual pelagic niche of AIV has any, endemic, epidemic or epizootological/epidemiological role to play.

c) Relevant species to be considered

Ducks

The world’s 147 species of ducks, geese and swans (order Anseriformes) are commonly lumped together under the term “waterfowl”. The family Anatidae (ducks, geese and swans) can be divided in three families; Anseranatinae (one species, the magpie goose), Anserinae (33 species, swans and geese, whistling ducks, cape barren goose and freckled duck) and the largest Anatinae with eight tribes and 113 species. The tribe Anatini in the
family *Anatinae*, or dabbling ducks is the largest of the family with 41 species, 38 of them belonging to the large cosmopolitan genus *Anas*, including many of the most abundant duck species in the world.

The Anseriformes are cosmopolitan and can be found in all regions except Antarctica and are associated with a wide variety of aquatic habitats, mostly breeding on fresh water.

Ducks are well-studied organisms in ecology since they: 1) are wide-spread, common, and serve as flagship species in wetland conservation and management, 2) can serve as bioindicators, 3) are among the most important hunted birds, in industrialised as well as in developing countries, 4) are an important source of protein to humans, especially in the eastern hemisphere, 5) continue to produce patterns of interest to ecology in general (Batt et al. 1992; Newton, 1998).

**Shorebirds**

The order *Charidriiformes* represent a big diverse group of marsh-, wading and cursorial birds. Representatives in all open habitats from the Arctic seas to the tropical deserts, usually closely related with water. This order includes many long distance migrants. Eighteen families including *Laridae* (gulls) and *Alcidae* (auks) recognised (all but 5 represented in Europe) (Cramp and Simmons, 1983).

### 14.1.2 AREAS AT RISK IN EUROPE DEPENDING ON THE RECRUITMENT AREA AND MOVEMENT OF WILD BIRDS

#### 14.1.2.1 Recruitment areas, migratory behaviour and wintering areas

Virtually all wild birds are migratory but there is no common pattern in migration routes or flyways for European bird populations. Most of the European birds (mostly *passerines*) have a migration route in a north-south direction either to the Mediterranean basin including North Africa, or even trans Sahara to tropical Africa. Shorebirds, terns and many passerines undertake transequatorial migrations every autumn to tropical and also to southern Africa. A few European passerine species as the bluethroat (*Luscinia svecica*) and the rustic bunting (*Emberiza rustica*) migrate, by several stop over sites and over a long time period, in a eastern western route to the winter quarter either in the Indian sub-continent or in south east Asia. A very schematic overview of migratory routes and their overlaps in particular for (a) waterfowl and (b) shorebirds, indicating potential mixing between avian species, is given in figure 14.1.1. below:
Figure 14.1.1. Migratory areas (‘flyways’) showing potential mixing regions for: (a) waterfowl; (b) shorebirds (Stroud et al., 2004).

The recruitment areas of migratory ducks and shorebirds are mainly in Fennoscandia, Western Russia and east to the Taimyr Peninsula. Certain shorebird species are high arctic recruited from Greenland, Spitsbergen and Arctic parts of Russia. The spring migration for shorebirds and ducks is often fast with stopping over at a few important sites, mainly in Western Europe, Fennoscandia and to some extent in wetland areas in South and East Europe. In autumn the migration is a continuum with succession in time of sexes and ages. It is more common with congregations of huge number of birds on optimal stop-over sites for a long time at resting places during their autumn migration. Many species have their wintering sites in wetlands and farmlands in western and south Western Europe. Other species, primarily shorebirds, are more long distance migratory to western and southern Africa.

Mallards, present a special challenge, as they utilize several countries and a wide range of habitats during their annual cycle. The mallard population is not uniform. Certain birds are sedentary others are short distance migratory, moving from inland to more optimal areas along the European west coast. Eastern mallard populations are long distance migratory, wintering much further south compared to European populations (Cramp and Simmons, 1977). Moreover, a degree of philopatry in the sexes creates complex patterns of gene flow within this geographically wide-spread species (Anderson et al., 1992).

The mallard can inhabit every kind of wetland within range, in fresh, brackish and salt waters, as long as they are relatively shallow and provide some cover. The mallard is very tolerant of human presence and is frequent in ornamental waters, irrigation networks and reservoirs. In the non-breeding season it can be found along the coast, on lowlands, estuaries bays and other sheltered sites. It is probably the most widespread and numerous of all ducks, partly due to its adaptability to humanised areas and to introduction. It is estimated that at least 9,000,000 mallards wintering in W. Palearctic (Europe, North Africa and the Middle East) (del Hoyo et al., 1992). The abundant and wide spread
mallard is the model organism of duck population ecology, and also a main natural reservoir of AIV (Webster et al., 2002).

There was an increase and expansion of Black headed gulls, establishing new populations during the 20th century. Most BHG breed inland throughout their European range and the species’ breeding distribution and abundance in most countries is not precisely known. They breed in colonies numbering up to 10,000 birds at certain optimal sites and the estimated population in Europe is approximately 1 million pairs (Cramp and Simmons, 1983). The migration of non breeding birds or even juveniles can start as early as June. Their feeding behaviour is opportunistic with a wide spectrum of items on their menu. Therefore, they can be found in good numbers virtually in any habitat in Western and Southern Europe during winter. It is a common bird around domestic animal farms, waste dumps, sewage ponds and in cities.

a) Temporal considerations

It is considered that the perpetuation of influenza viruses in Canadian free-living waterfowl was related to the passage of virus from adult to juvenile birds on lakes where the birds congregated before migration (Hinshaw et al., 1980a). Considerable quantities of the virus are excreted with the faeces, Webster et al. (1978) estimated up to $10^{8.7}$ mean egg infectious doses per g of faeces from infected ducks. This contaminates lake or pond water; to the extent that virus may be isolated from untreated lake water where large numbers of waterfowl are found (Hinshaw et al., 1980b). Influenza virus may remain infective in lake water for up to 4 days at 22°C and over 30 days at 0°C (Webster et al., 1978). Stallknecht et al. (1990a), estimated that from an initial concentration of $10^6$ TCID$_{50}$/ml infectivity was retained for up to 207 days at 17°C and 102 days at 28°C. Contaminated lake or drinking water may therefore result in infection by the faecal/oral route, or possibly by the faecal/cloacal route as a result of ‘cloacal drinking’. For all birds the ingestion of infective faeces appears to be the most important mode of transmission. Data from the 3-year study on ducks on lakes in Alberta, Canada showed that influenza virus isolation rates from juvenile ducks may exceed 60% (Hinshaw et al., 1980a).

14.1.2.2 Resting sites and Wintering sites

Gregariousness is frequent among the waterfowl and is particularly pronounced in geese and ducks. The flocks may vary in size depending on species composition, time of the year and locality. In geese and swans, but also in some ducks, the typical flock is composed of one or a few family parties, whereas in many duck species large flocks often mostly contain birds of one sex, as males and females commonly have separate distribution for much of the non breeding season. This sociability facilitates the location of the best feeding sites and the learning of migration routes and reduces the individual risk for predation. Dense flocks, numbering tens or even hundreds of thousands of roosting and/or over wintering shorebirds, with a mix of species, sex and ages are often seen in optimal spots.

Inventory of sites: Characteristics: Wetland, marshland, river deltas with estuaries, natural ponds, eutrophic lakes, tidal creeks, agricultural areas, and man made ponds and waste dumps. For information about sites of particular interest in respective country
Mingling with other species: At certain roosting or wintering sites the number of birds can be hundreds of thousands of individuals of gulls, shorebirds, swans, geese and ducks. The intra and interspecies mix of birds and eventually transmission of AIV between species on these sites can be extensive.

Proximity of poultry population: No information is available at the moment. It is important to make an inventory of areas with high density of poultry farms in proximity to roosting or wintering sites for waterfowl as soon as data on poultry dense areas will become available.
Table 14-1.2.: Summary of selected AIV surveillance studies in wild birds

<table>
<thead>
<tr>
<th>Species</th>
<th>Location</th>
<th>Time and year</th>
<th>Prevalence</th>
<th>%</th>
<th>Method of detection</th>
<th>Sample</th>
<th>Subtypes</th>
<th>Reference</th>
<th>Comments</th>
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<tr>
<td><strong>Procellariformes</strong></td>
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<tr>
<td>Wedge-tailed Shearwater (Puffinus pacificus chlororynchos)</td>
<td>Australia Great Barrier reef</td>
<td>1971 Dec</td>
<td>1/201</td>
<td>0,5</td>
<td>Virus isolation</td>
<td>Tracheal</td>
<td>H5N5</td>
<td>Downie and Laver (1973)</td>
<td>No antibodies found newly introduced?</td>
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<td><strong>Phalacrocoracidae</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Cormorant (Phalacrocorax carbo)</td>
<td>East Germany</td>
<td>1977-89</td>
<td>18/4500</td>
<td>0,4</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td>H6N1</td>
<td>Süss et al (1994)</td>
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<tr>
<td><strong>Ciconiiformes</strong></td>
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<tr>
<td>Great-Blue herons (Ardea Herodias)</td>
<td>USA, Virginia, Watts Island</td>
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<td>Fecal/Cloacal</td>
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<td>Graves 1992</td>
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<td>1979</td>
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<td></td>
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<td></td>
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<tr>
<td>Mute swan (Cygnus olor)</td>
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<td>1979</td>
<td>0/126</td>
<td>0</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td>H2N2, H1N2</td>
<td>Sinnecker et al (1982)</td>
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<tr>
<td></td>
<td></td>
<td>1980</td>
<td>0/69</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1981</td>
<td>6/308</td>
<td>0</td>
<td></td>
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<td></td>
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<tr>
<td>Whistling swan or Bewick’s swan (Cygnus columbianus, columbianus)</td>
<td>USA/North Carolina Mattqamus ket and Pungo refuges</td>
<td>1977-1979</td>
<td>0/452</td>
<td>0</td>
<td>Isolation</td>
<td>Fecal/Cloacal</td>
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<td>Graves 1992</td>
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<tr>
<td>Tundra swan (Cygnus columbianus)</td>
<td>Japan, Hokkaido Lake</td>
<td>1995-98 Oct</td>
<td>0/345</td>
<td>0</td>
<td>Virus isolation</td>
<td>Fresh fecal samples</td>
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Table 14-1.2.: Summary of selected AIV surveillance studies in wild birds

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<th>Species</th>
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<th>Time and year</th>
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<th>Subtypes</th>
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<td>1977-89</td>
<td>4/611</td>
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<td>Breeding and wintering birds</td>
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### Table 14-1.2.: Summary of selected AIV surveillance studies in wild birds

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Table 14-1.2.: Summary of selected AIV surveillance studies in wild birds

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## Table 14-1.2: Summary of selected AIV surveillance studies in wild birds

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### Table 14-1.2.: Summary of selected AIV surveillance studies in wild birds

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<td>H3N8, H4N6, H4N9, H11N1 H11N9 H11N9 H13N6</td>
<td>Okazaki et al 2000</td>
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<td>0/74 0/206 0/22 0/239</td>
<td>0 0 0 0</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td>Sinnecker et al 1982</td>
<td>No facts about date time of year or catching method</td>
<td></td>
</tr>
<tr>
<td>Russia Western Siberia</td>
<td>1995-98 Aug</td>
<td>0/44</td>
<td>0</td>
<td>Virus isolation</td>
<td>Fresh fecal samples</td>
<td>Okazaki et al. 2000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Franklins Gull (Larus pipixcan)</td>
<td>USA Minnesota</td>
<td>1974 Sept</td>
<td>0/60</td>
<td>0</td>
<td>Virus isolation</td>
<td>Tracheal</td>
<td>Bahl et al 1977</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ringbilled Gull (Larus delawarensis)</td>
<td>USA Delmar Peninsula</td>
<td>1985-87</td>
<td>0/234</td>
<td>0</td>
<td>Virus isolation</td>
<td>Fresh droppings</td>
<td>Kawaoka et al 1988</td>
<td>Date not specified</td>
<td></td>
</tr>
<tr>
<td>USA, Maryland, Baltimore</td>
<td>1977-79</td>
<td>70/3403</td>
<td>2</td>
<td>Isolation</td>
<td>Fecal/Cloacal</td>
<td>H2,5,6,9,11,13 N2-6,8-9</td>
<td>Graves 1992</td>
<td>Prevalence 0.26% winter 3% summer</td>
<td></td>
</tr>
<tr>
<td>USA, Ohio</td>
<td>1986-1988</td>
<td>0/116</td>
<td>0</td>
<td>Isolation</td>
<td>Cloacal</td>
<td>Slemons et al 1991</td>
<td>Juveniles &lt; 3 weeks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gulls (Larus ridibundus, L. canus).</td>
<td>East Germany</td>
<td>1977-89</td>
<td>13/2182</td>
<td>1</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td>Prevalent subtypes H7N3, H11N6</td>
<td>Dates not specified</td>
<td></td>
</tr>
<tr>
<td>Larger gulls (Larus argentatus, L. fuscus)</td>
<td>The Netherlands</td>
<td>1999-2000</td>
<td>0/1116</td>
<td>0</td>
<td>RT/PCR and</td>
<td>Cloacal/ droppings</td>
<td>Fouchier et al 2003</td>
<td>Both breeding,</td>
<td></td>
</tr>
<tr>
<td>Species</td>
<td>Location</td>
<td>Time and year</td>
<td>Prevalence</td>
<td>%</td>
<td>Method of detection</td>
<td>Sample</td>
<td>Subtypes</td>
<td>Reference</td>
<td>Comments</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>---------------------------</td>
<td>----------------------------</td>
<td>-----------------</td>
<td>-----</td>
<td>---------------------</td>
<td>--------</td>
<td>---------------</td>
<td>-----------</td>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Royal Tern (Thalasseus maximus)</td>
<td>USA, Virginia, Metomkin island</td>
<td>1978 summer</td>
<td>0/36</td>
<td>0</td>
<td>Isolation</td>
<td>Fecal/Cloacal</td>
<td>Graves 1992</td>
<td>5-6 weeks old</td>
<td></td>
</tr>
<tr>
<td>Sandwich tern (Sterna sandvicensis)</td>
<td>East Germany</td>
<td>1979 1980 1981</td>
<td>1/75 0/87 0/189</td>
<td>1</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td>?N2</td>
<td>Sinnecker et al 1982</td>
<td></td>
</tr>
<tr>
<td>Arctic tern (Sterna paradisea)</td>
<td>East Germany</td>
<td>1981</td>
<td>2/28</td>
<td>7</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td>H1N1</td>
<td>Sinnecker et al 1982</td>
<td></td>
</tr>
<tr>
<td>Common tern (Sterna hirundo)</td>
<td>East Germany</td>
<td>1980 1981</td>
<td>0/50 0/13</td>
<td>0</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td></td>
<td>Sinnecker et al 1982</td>
<td></td>
</tr>
<tr>
<td>South Africa</td>
<td></td>
<td>1961</td>
<td></td>
<td></td>
<td>Virus isolation</td>
<td></td>
<td>H5N3</td>
<td>Becker 1966</td>
<td></td>
</tr>
<tr>
<td>East Germany</td>
<td></td>
<td>1977-89</td>
<td>13/812</td>
<td>2</td>
<td>Virus isolation</td>
<td>Cloacal and tracheal</td>
<td>Prevalent subtypes H3N3, H7N3, H10N8</td>
<td>Süss et al 1994</td>
<td></td>
</tr>
<tr>
<td>Knots (Calidris canuta)</td>
<td>USA Delmara Peninsula</td>
<td>1985-87 may-oct</td>
<td>2/37</td>
<td>5</td>
<td>Virus isolation</td>
<td>Cloacal</td>
<td>H1, H2, H4, H6, H7, H9, H10, H11, H12, H13 and N1- N7, N9</td>
<td>Kawaoka et al 1988</td>
<td></td>
</tr>
<tr>
<td>Ruddy turnstone (Arenaria interpres)</td>
<td>USA Delmara Peninsula</td>
<td>1985 May Sept Oct 1986 Feb-Apr May June Jul-Aug Sept Nov-April 1987 May</td>
<td>17/85 0/125 2/50 0/230 10/412 6/165 0/165 5/141 0/260</td>
<td>20 0 8 0 2 4 0 4 0</td>
<td>Virus isolation</td>
<td>Fresh droppings</td>
<td>Species not specified.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shorebirds and Gulls (Otidiae sp. Laridae sp.)</td>
<td>USA Delmara Peninsula</td>
<td>1985 May Sept Oct 1986 Feb-Apr May June Jul-Aug Sept Nov-April 1987 May</td>
<td>17/85 0/125 2/50 0/230 10/412 6/165 0/165 5/141 0/260</td>
<td>20 0 8 0 2 4 0 4 0</td>
<td>Virus isolation</td>
<td>Fresh droppings</td>
<td>Species not specified.</td>
<td></td>
<td></td>
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</tbody>
</table>
### Table 14-1.2.: Summary of selected AIV surveillance studies in wild birds

<table>
<thead>
<tr>
<th>Species</th>
<th>Location</th>
<th>Time and year</th>
<th>Prevalence</th>
<th>%</th>
<th>Method of detection</th>
<th>Sample</th>
<th>Subtypes</th>
<th>Reference</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shorebirds</td>
<td>Sweden, Ottenby</td>
<td>1999 Autumn</td>
<td>0/1997</td>
<td>0</td>
<td>RT/PCR and subsequent virus isolation</td>
<td>Cloacal/ droppings</td>
<td></td>
<td>Fouchier et al 2003, Wallensten et al in prep.</td>
<td>Mainly from the families Charadriiforme and Scolopacidae</td>
</tr>
<tr>
<td>Shorebirds Charadriiformes not specified</td>
<td>Russia, Western Siberia</td>
<td>1995-98 aug</td>
<td>0/24</td>
<td>0</td>
<td>Virus isolation</td>
<td>Fresh fecal samples</td>
<td></td>
<td>Okazaki et al. 2000</td>
<td></td>
</tr>
<tr>
<td>Coot <em>Fulica atra</em></td>
<td>Italy, Laguna di Orbitello, Lago di Burano</td>
<td>1993-98 winter</td>
<td>4/333</td>
<td>1</td>
<td>Virus isolation</td>
<td>Cloacal</td>
<td>De Marco et al 2003</td>
<td></td>
<td></td>
</tr>
<tr>
<td>East Germany</td>
<td>1977-89</td>
<td>13/1312</td>
<td>1</td>
<td>Prevalent subtype H4N7</td>
<td></td>
<td>Scientific and common name not in accordance. European knots assumed to be coots</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The Netherlands/ Sweden</td>
<td>1999-2000</td>
<td>0/95</td>
<td>0</td>
<td>RT/PCR and subsequent virus isolation</td>
<td>Cloacal/droppings</td>
<td>Fouchier et al 2003</td>
<td>Mix of migrating and wintering birds</td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Coot (<em>Fulica Americana</em>)</td>
<td>USA, Ohio</td>
<td>1986-1988</td>
<td>0/11</td>
<td>0</td>
<td>Isolation</td>
<td>Cloacal</td>
<td>Slemons et al 1991</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guillemot (<em>Uria aalge</em>)</td>
<td>Russia, Sakhalin</td>
<td>1974 aug</td>
<td>1/100</td>
<td>1</td>
<td>Virus isolation</td>
<td>Cloacal swab</td>
<td>H3N2</td>
<td>Sazanov et al 1977</td>
<td>Contamination or “spill back” from human strain circulating at the time?</td>
</tr>
<tr>
<td>Sweden Bonden,</td>
<td>2000</td>
<td>A 0/10</td>
<td>0</td>
<td>19</td>
<td>RT/PCR and subsequent virus isolation</td>
<td>Cloacal/droppings</td>
<td>H6N2</td>
<td>Wallensten et al. 2005</td>
<td></td>
</tr>
</tbody>
</table>
### Table 14-1.2.: Summary of selected AIV surveillance studies in wild birds

<table>
<thead>
<tr>
<th>Species</th>
<th>Location</th>
<th>Time and year</th>
<th>Prevalence</th>
<th>%</th>
<th>Method of detection</th>
<th>Sample</th>
<th>Subtypes</th>
<th>Reference</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Galliformes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pheasant (Phasianus colchicus)</td>
<td>Italy Po Valley</td>
<td>1992 feb</td>
<td>0/379</td>
<td>0</td>
<td>Serology</td>
<td>Blood</td>
<td></td>
<td>De Marco et al 2003</td>
<td></td>
</tr>
<tr>
<td></td>
<td>USA, Pennsylvania</td>
<td>1984 June-Nov</td>
<td>0/84</td>
<td>0</td>
<td>Virus Isolation</td>
<td>Tracheal and cloacal swabs</td>
<td></td>
<td>Hinshaw et al 1985a,b</td>
<td></td>
</tr>
<tr>
<td>Quail (Coturnix coturnix)</td>
<td>Italy Pesaro province</td>
<td>1998 may</td>
<td>0/260</td>
<td>0</td>
<td>Serology</td>
<td>Blood</td>
<td></td>
<td>De Marco et al 2003</td>
<td>Migrating birds</td>
</tr>
<tr>
<td></td>
<td>USA, Maryland, Chestertown</td>
<td>1979</td>
<td>0/30</td>
<td>0</td>
<td>Isolation</td>
<td>Fecal/Cloacal</td>
<td></td>
<td>Graves 1992</td>
<td></td>
</tr>
<tr>
<td><strong>Passeriformes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reed warbler (Acrocephalus scirpaceus)</td>
<td>Italy Lago di Massaciucoli</td>
<td>1993 aug-sept</td>
<td>0/83</td>
<td>0</td>
<td>Virus isolation</td>
<td>Cloacal</td>
<td></td>
<td>De Marco et al 2003</td>
<td></td>
</tr>
<tr>
<td>Other birds mainly</td>
<td>Sweden, the Netherlands</td>
<td>1999-2000</td>
<td>0/2007</td>
<td>0</td>
<td>RT/PCR and subsequent virus isolation</td>
<td>Cloacal</td>
<td></td>
<td>Fouchier et al 2003</td>
<td>Approximately 200 species</td>
</tr>
<tr>
<td>Passeriformes</td>
<td>Nigeria</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>
14.2 ANNEX II

14.2.1 ILLEGAL IMPORTS: GB AS A CASE STUDY

In April 2001, DEFRA set up the Illegal Animal Products Seizures (ILAPS) Database, to allow all those concerned with detecting illegal imports into GB to record seizures following attempted illegal importation of animal products. Data has been collected on meat (including poultry), fish, dairy products, and other good, for example honey. The database has a number of limitations; in particular the initial recording of data was poor. For a fuller description of the database, its contents and its limitations see VLA Report (2004). The complete database was supplied to the authors of this report.

This database has been used here in order to illustrate the potential problems of illegal importation as applied to avian diseases. Data on poultry meat and meat products has been abstracted from the database, the details of quantities being given in Table 5.2. Places from which seizure was made comprise: cargo, personal baggage, shop/warehouse, transit shed and unknown. Product description of seizures comprise: raw poultry meat, canned meat and miscellaneous (e.g. poultry powder), ground/deboned. Routes of entry include ports and airports (M. Wooldridge, pers.comm.)

Table 14-2-1. Data on poultry meat and other poultry products abstracted from the GB ILAPS database supplied to the authors of VLA Report (2004) (M. Seaman, pers. comm.); any record with poultry product in seizure description (the two periods were the subject of separate analyses, so are shown separately)

<table>
<thead>
<tr>
<th>Seizures recorded from 1st April 2001 to 10th January 2003</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of seizures</td>
</tr>
<tr>
<td>Total weight of seizures (kg)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Seizures recorded from 11th January 2003 to January 5th 2004</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of seizures</td>
</tr>
<tr>
<td>Total weight of seizures (kg)</td>
</tr>
</tbody>
</table>

The data show that in GB/UK alone, during the period 1 April 2001 to 5 January 2004 33,673 kg of items containing chicken product were detected and seized during attempts to import illegally.

Detailed analysis of the data indicates that up until 10 January 2003, the weight of seizures from Asia (an area relevant to risks from AI) comprised 39% of the total for that period. Of this, 69% of seizures were from personal baggage (R. Jones, pers. comm.).

Detailed analysis of the collection period from 11 January 2003 onwards indicated that 48% of the seizures were from Asia, and 0.007% from Eastern Europe (it includes countries that became new Member States on 1 May 2004 (S. Hall, pers. comm.).

Not all attempted illegal imports are detected. The estimate of scale factors (seizure rates) obtained by the VLA Report (2004), based on the collected opinions of those responsible for detection, indicate that only a small proportion of attempted illegal imports are detected and seized. The values estimated for the scale factors in that report are given in table 13-1. As the scale factor is based on expert opinion, it is
subject to much uncertainty, thus a distribution was used to describe the scale factor. The use of a distribution gives a minimum, mean and maximum estimate of seizure rates (and thus total illegal imports) over the period investigated (for details see VLA Report, 2004). A different scale factor was applied to each detection source.

Table 14-2-2. Estimated scale factors for detection of illegal imports at GB ports and airports (VLA Report 2004, simplified)

<table>
<thead>
<tr>
<th>Detection source</th>
<th>Mean scale factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cargo</td>
<td>0.015</td>
</tr>
<tr>
<td>Personal Baggage</td>
<td>0.002</td>
</tr>
<tr>
<td>Post</td>
<td>0.01</td>
</tr>
<tr>
<td>Transit shed</td>
<td>0.007</td>
</tr>
</tbody>
</table>