Import risk analysis: avian paramyxovirus type 1 in hens' hatching eggs

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Approved for general release

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Executive Summary

This document is an analysis of the risk posed by exotic strains of avian paramyxovirus type 1 (the causative agent of Newcastle disease) in imported hatching eggs of hens (*Gallus gallus*). Although non-pathogenic strains of avian paramyxovirus type 1 are present in New Zealand, there has never been an outbreak of Newcastle disease in this country.

This analysis concludes that there is a low risk that exotic strains of avian paramyxovirus type 1 could be introduced in hen's hatching eggs. The spread of introduced strains is considered possible by a variety of exposure pathways, although the risks of airborne spread have been greatly exaggerated over the past 30 years.

The introduction of highly virulent strains of the virus would almost certainly result in serious mortality in commercial and hobby poultry flocks. Furthermore, exposure of wild birds could lead to the virus being introduced into endangered native bird populations, with potentially devastating effects. While the introduction of field strains of low virulence might not cause severe disease in New Zealand bird populations, some strains may have significant effects on avian health and production, and since low virulence field strains may not be as stable as was thought in the past, there is a possibility that such strains could mutate to become virulent after introduction into chicken flocks.

The key safeguards recommended for importation of hatching eggs of hens are:

- Pre-export testing of layer flocks to ensure that avian paramyxovirus type 1 viruses are not circulating;
- Post-arrival quarantine to ensure that any undetected avian paramyxovirus type 1 viruses are contained.

The present avian quarantine facility standard, which includes safeguards for airborne spread, will be revised as a consequence of this risk analysis.

Introduction

This is the first time that MAF has carried out a comprehensive analysis of the risk of introduction, establishment and spread of exotic strains of avian paramyxovirus type 1 (APMV-1) in hatching eggs of hens. In the past, MAF has based its safeguards and quarantine standards largely on the conditions developed in Australia.

The **hazard identification** takes account of the fact that although a number of isolates of APMV-1 have been made from several avian species over the past three decades, New Zealand has never experienced an outbreak of Newcastle disease. The intracerebral pathogenicity index (ICPI) of the New Zealand isolates is low (less than 0.2) and the amino acid sequence of the fusion protein cleavage site is typical of viruses of low virulence (Pharo et al, 2000). The hazard identification concludes that the focus of this risk analysis should include any strain of APMV-1 with a greater pathogenicity than endemic strains.

The **release assessment** considers the likelihood of introduction of field strains or vaccine strains in or on eggs.

The **exposure assessment** considers the most likely pathways by which APMV-1 could be spread to New Zealand's bird populations if the virus were introduced through hatching eggs. Long-range airborne spread of virus is a fundamental part of the case made by Australian authorities to support stringent quarantine conditions, but this risk analysis concludes that the airborne spread hypothesis is not supported by the available evidence.

The **consequence assessment** considers the uncertainty surrounding the likely behaviour of exotic strains of APMV-1 if they were to be introduced, and concludes that any strains of the virus that are more pathogenic than the endemic strains are of potential concern.

The **risk estimation** considers that safeguards are justified, but that in view of the previous exaggeration of the risk of airborne spread, the post-arrival quarantine standards may not need to be as stringent as those used in the past.

The section on **risk management** recommends safeguards that are considered appropriate to manage the identified risk.

Appendix 1 is a detailed examination of the risk of airborne spread, and **Appendix 2** presents the post-arrival quarantine standards that are currently in place for New Zealand, and compares these to standards in place for Australia, Canada, the European Union, and the USA.

1. Hazard Identification

Newcastle disease (ND) is caused by certain strains of APMV-1 which is a member of the genus *Rubulavirus* in the family *Paramyxoviridae,* subfamily *Paramyxovirinae* (van Regenmortel et al, 2000). New Zealand has never experienced an outbreak of Newcastle disease. An avirulent strain of APMV-1 with an intracerebral pathogenicity index (ICPI) of up to 0.16 has been isolated from time to time in this country (Pharo et al, 2000). Exotic strains of APMV-1 are Notifiable Organisms under the Biosecurity Act 1993.

Field strains of low virulence may not be as stable as was thought in the past, and it is now considered possibile that some such strains could mutate to become virulent after introduction into chicken flocks (Alexander, 2001).

Therefore this risk analysis is concerned not only with strains of APMV-1 which fit the definition of ND virus (OIE, 1999a), but with all exotic strains of APMV-1 virus with an ICPI greater than 0.16. Such viruses are common in many countries, either as freely circulating field strains in wild and domestic birds, or as live vaccine strains that are administered to protect poultry flocks from highly virulent strains of APMV-1.

2. Risk Assessment

2.1 RELEASE ASSESSMENT

The following discussion on the likelihood that hatching eggs will carry APMV-1 must be considered in the context of the new OIE definition for ND[1] (OIE, 1999a). An important consequence of the new definition is that it is no longer appropriate to base international trade decisions on the classification of APMV-1 viruses into pathotype groups (i.e. lentogenic, mesogenic and velogenic). Nevertheless, as much of the available research is based on those pathotype groupings, there may be difficulties applying the conclusions of earlier research work to the new system of APMV-1 classification. Therefore, this analysis will use the old terminology when discussing most of the existing research available in the literature, but will use the new definition wherever possible.

2.1.1 Carriage on eggs

Lancaster and Alexander (1975) cited the experiments of Williams and Dillard (1968) who found that ND virus (Texas-GB strain) penetrated the cuticle and shell and occasionally through the outer shell membrane of eggs. This phenomenon was considerably more common in cracked than in uncracked eggs.

Beard and Hanson (1984) considered that since ND virus is shed in large amounts in faeces of infected birds, it follows that eggs from infected hens could carry the virus, resulting in chicks becoming infected upon hatching. Alexander (1997) reached a similar conclusion.

2.1.2 Carriage in eggs

a) Natural infections

Alexander (1997) noted that true vertical transmission of ND viruses (i.e. passing of virus from parent to progeny via the embryo) remains controversial, and its significance in epidemics remains unclear. He noted further that experimental assessment of this route of transmission using virulent viruses is usually hampered by cessation of egg laying in infected birds. Beard and Hanson (1984) went further by contending that **all** embryos vertically infected with very virulent ND virus die before hatching.

However, it is unclear whether the experimental work leading to the above conclusions was carried out using immunologically naïve or immune hens.

This may be important, because as noted by Alexander (1997), where vaccination is practised or where lentogenic strains circulate, the introduction of velogenic ND virus may be masked. Recent experience in Australia confirms that virulent ND virus can circulate in chicken flocks in the absence of overt clinical disease (OIE 1999b, 1999c, 1999d, 2000a, 2000b, 2000c). In the Australian situation it appears likely that the chickens were at least partially immunised against ND with a field strain of low virulence prior to infection with the virulent strain.

[1] Newcastle disease is now defined as an infection of birds caused by a virus of APMV-1 that ***either*** has an ICPI of 0.7 or greater ***or*** has multiple basic amino acids at the cleavage site of the fusion protein.

Beard and Hanson (1984) did state that ND virus has been isolated from eggs laid by diseased hens. Three references were cited in support of this : Hofstad (1949), Bivins et al (1950), and Zargar and Pomeroy (1950).

More recently, Capua et al (1993) reported the finding of virulent ND virus from commercial embryonated hens' eggs. There were no problems of embryo mortality or reduced hatchability. The virus was also isolated from the chickens which hatched from these eggs, following episodes of acute mortality but without typical clinical signs of ND. The birds which had laid the eggs were laying normally and had no clinical signs of ND, but despite their high levels of antibody, ND virus was isolated from cloacal swabs.

b) Vaccine strains

There is good evidence that lentogenic and mesogenic vaccine strains of APMV-1 viruses can be transmitted vertically.

French et al (1967) reported that embryonated eggs experimentally infected with the Australian V4 (lentogenic) strain survived, hatched and yielded infected chicks.

According to Lancaster and Alexander (1975), Raszewska (1964) reported that La Sota vaccine was found in the reproductive tract (ovary, oviduct and uterus) of 50% of laying hens for 2-8 days after vaccination.

Lancaster and Alexander (1975) also indicated that Ahmed and El-Sisi (1965) reported the shedding of the Haifa-Komarov strain (a mesogenic vaccine virus) in the eggs of vaccinated hens. The virus was apparently shed irregularly in the yolk of eggs for a period of at least 1 month following vaccination. The greatest level of virus shedding generally occurred during the first 9 days after vaccination. It was concluded that the shedding of virus could constitute a danger following breakage of the eggs, and, in this way, contaminating chicken crates, incubators etc.

Also cited by Lancaster and Alexander (1975) was Tanwane (1971), who, using the Mukteswar strain (mesogenic), apparently recovered virus in eggs up to 35 days after vaccination.

Lancaster and Alexander (1975) also cited Coman (1963) as reporting that for almost 3 weeks following subcutaneous inoculation of hens with strain F ND virus (lentogenic), the virus could be demonstrated in the eggs. Infected chicks were apparently hatched from such eggs. Similarly, French et al (1967a) found that hatchling chicks infected with two different viruses of low pathogenicity had very high titres of virus in their tissues.

2.1.3 Release assessment conclusions

Since APMV-1 viruses have been shown to penetrate the shell of eggs, particularly if the eggs are cracked, and since the viruses are transmitted in faeces, hatching eggs derived from infected hens are likely to be contaminated.

There is good evidence that vaccine or field strains of low or moderate virulence can be vertically transmitted in eggs. True vertical transmission remains a controversial issue for highly virulent viruses, but there is a distinct possibility that vertical transmission may occur where immune or semi-immune birds are superinfected with virulent strains.

A number of outbreaks of Newcastle disease in Italy during the year 2000 have been traced back to hatcheries which had imported hatching eggs from a number of European and non-European countries, following the epidemic of avian influenza which affected Italy between 1999 and 2000 (Capua et al, 2000). Although it is not clear whether the transmission was due to "true vertical transmission" or shell contamination, these outbreaks are a timely reminder that there is a risk associated with imported hatching eggs (Ilaria Capua, personal communication with H Pharo, 18/11/2000).

It is concluded that in countries where vaccination is practised or which have field strains of APMV-1 circulating, it is possible for hatching eggs to be infected or contaminated.

2.2 EXPOSURE ASSESSMENT

If hatching eggs carrying exotic strains of APMV-1 were imported, whether the mechanism were mechanical or true vertical transmission, the most likely outcome would be the introduction of the virus into the hatchery. Exposure pathways by which infection could be introduced from hatcheries into commercial poultry flocks have rarely been explicitly stated.

Nevertheless, in order to consider the risk of exposure of birds outside the hatchery environment, potential routes of transmission of APMV-1 will be considered. In considering such spread, this assessment will assume a "ground zero" situation, that is, one where there are no biosecurity measures in place. In such an environment, the potential routes of spread from an infected hatchery would be :

- Mechanical spread, primarily by the movement of people and equipment
- Movement of infected birds from the hatchery (live or dead)
- Airborne spread

2.2.1 Mechanical spread

Utterbach (1972) considered that the importance of spread by people was second only to the movement of live birds in the 1971-1973 epidemic in California. Others have also stressed the importance of this mode of spread during epidemics (Alexander, 1988; 2000), and there is now no doubt that in outbreaks (given that live birds are not moved between poultry units) the greatest potential for spread of ND is by the actions of humans in terms of moving feedstuffs, personnel and equipment to and from poultry farms (Alexander, 1997). The reason for this is that large amounts of virus are shed in the faeces of infected birds. Vaccinated flocks have shed virulent field virus for more than 4 months (Utterbach and Schwartz, 1973) and as the virus can remain infectious for 42 to 53 days in poultry litter (Devos, 1972), there is a considerable potential for contamination of objects coming into contact with such material. Mechanical transfer of infected faeces by rodents, fleas, insects, dogs, cats or scavenging animals may occur in some circumstances (Kouwenhoven, 1993; Alexander, 1995).

2.2.2 Movement of birds

The movement of birds (live or dead) which are carrying ND virus is a major potential method of spread (Alexander, 1988).

2.2.3 Airborne spread

The risk of airborne spread has been considered by the Australian authorities to justify the requirement for HEPA filtration in quarantine hatcheries (AQIS, 1998). New Zealand has tended to follow the Australian lead in the past, and this is the first time that MAF has carried out an independent detailed risk assessment on the issue.

An analysis of the case made by the Australian authorities in support of airborne spread on APMV-1 is presented in Appendix 1. The Australian case is based on reports from the United Kingdom and Northern Ireland during the 1970-1972 epidemic. However, the conclusion reached in Appendix 1 of this risk analysis is that the likelihood of airborne spread has been

overstated and there is no convincing evidence that it occurs even between poultry sheds separated by a few metres.

By contrast, Lancaster (1975) has summarised a number of reports (Siccardi, 1966; Grass, 1971; Utterback and Schwartz, 1973) which indicate that airborne spread was considered to be of little importance in outbreaks in Nigeria, Southern USA, and California.

Despite the absence of evidence in support of the hypothesis of airborne spread, it may be possible for faecal dust to be blown a short distance by wind, so the potential for very short distance airborne transmission may exist under certain weather conditions.

2.2.4 Exposure assessment conclusion

The exposure pathways for APMV-1 are primarily those that rely on direct or indirect contact with faecal material. Spread by humans or human activities associated with poultry flocks is the most likely way that APMV-1 virus imported in hatching eggs would escape from the hatchery environment and result in exposure of avian species in New Zealand.

2.3 CONSEQUENCE ASSESSMENT

The effect of introduction of exotic APMV-1 viruses would depend on the virulence of the strain introduced.

The introduction of a highly virulent strain into commercial poultry flocks would result in high mortality, which would severely affect individual poultry farms. Control costs would be high, and if eradication were not possible the long term necessity to vaccinate poultry would add to costs such that poultry products would become more expensive. In addition to the direct costs of vaccination, Leslie (2000) estimated that in the United Kingdom vaccination resulted in a loss of about 2% of egg production in layers and 114 g per broiler.

Moreover, there is now good evidence that APMV-1 viruses may become virulent by mutation **after** introduction into chickens. Results obtained from viruses isolated from ND outbreaks in Ireland and Australia during the 1990s have suggested this may be how some virulent viruses emerge, perhaps requiring as few as two point mutations (Alexander, 2000).

Regardless of the pathogenicity, any introduction of exotic virus would adversely impact on the small but expanding export trade in poultry products and genetic material.

It is difficult to predict how APMV-1 virus would affect other avian species in this country but aviary or native birds could be adversely affected.

2.4 RISK ESTIMATION

The release assessment concludes that it is possible that vaccine strains or field strains of APMV-1 could be introduced in or on hens hatching eggs.

The exposure assessment concludes that in the absence of biosecurity measures there is a significant likelihood of spread from the hatchery, predominantly by the faecal-oral route, mediated by humans or human activities related to poultry management. This exposure could result in establishment of introduced APMV-1 viruses in avian species in New Zealand.

The consequence assessment concludes that there would be a significant negative impact if APMV-1 viruses (of any virulence) were introduced into avian species in New Zealand.

Thus, it is reasonable to conclude that sanitary measures are required to manage these risks.

3. Risk Management

Since hatching eggs obviously lead to live birds in quarantine, there is no reason to treat hatching eggs any differently from live birds in respect of pre-export and post-arrival quarantine for APMV-1.

3.1 GENERAL MEASURES

Measures are required to manage the risks of faecal contamination of eggs prior to shipping. The eggs must be clean, and must have been fumigated with formaldehyde using one of the methods in OIE *International Animal Health Code* Appendix 4.2.4.1 G 1

3.1 PRE-EXPORT TESTING

3.1.1 Unvaccinated flocks

Serological testing must show that unvaccinated layer flocks are seronegative to APMV-1. The required sample size is to give at least 99% confidence of detecting a prevalence of 5%. If test sensitivity were 100%, the sample size would be 90 birds, but assuming test sensitivity is around 90%, then the sample size will be 100 birds. For flocks smaller than 100 birds, all will be tested.

3.1.2 Vaccinated flocks

For vaccinated flocks, layer birds must be shown not to be circulating any APMV-1 viruses. This will be established by taking cloacal swabs for virus isolation from a representative sample of birds, to give at least 99% confidence of detecting a prevalence of 5%. Again, if test sensitivity were 100%, the sample size would be 90 birds, but assuming the sensitivity of virus isolation is around 90%, the sample size will be 100 birds[2]. For flocks smaller than 100 birds, all will be tested.

3.2 POST-ARRIVAL QUARANTINE

3.2.1 Biosecurity measures in quarantine facility

In the quarantine facility, strict biosecurity measures must be observed to prevent mechanical transfer of infected faeces from infected to susceptible birds outside the quarantine facility, either directly or indirectly through a number of routes. The specific requirements will be detailed in the MAF quarantine standard, but must include:

- Bird- and vermin-proofing of the building, including feed stores and water supply

[2] The typical commercial importation of hatching eggs results in about 7500 day-old chicks being placed in the quarantine unit, and these will be culled down to the required final numbers at around 4 weeks of age.

- Prohibition of any material of any sort to leave the quarantine facility during the period of quarantine, unless with the permission of MAF (e.g. samples to laboratory)

- Control of people movement, including shower-out

As discussed in the exposure assessment (section 2.2.3), the likelihood of airborne spread has been overstated for a number of years and there is no convincing evidence that it occurs even between poultry sheds separated by a few metres. However, it is also recognised that it is not possible to conclude that airborne spread does not occur at all, and therefore it is impossible to completely rule out the theoretical risk of short distance airborne spread via windborne particles of dried faeces[3]. This, together with the potentially severe consequences of introduction, justifies the imposition of measures to limit the spread of dust particles from quarantine hatcheries[4]. These measures will be:

- Filtration of exhaust air to achieve 90-95% arrestance of particles 3 microns or larger (EU8 filter).[5]

- In order to ensure that all air leaving the facility passes through the filters, the ventilation system will be designed to ensure that a negative pressure of at least -10 pascal is maintained at all times[6].

- In order to prevent any concentrations of avian species being exposed to accumulations of fine dust particles which are not filtered out from the exhaust air, the facility will be situated at least 100 metres from any concentrations of avian species[7].

It must be emphasised that despite the data deficiencies and uncertainty that surrounds this issue which prevents the objective determination of necessary safeguards of this sort, if a standard such as the above is finalised, then compliance testing will be required to ensure that the specifications are met

[3] It is not possible to objectively decide on a minimum particle size of concern. The only objective assessment of APMV-1 virus association with different particle sizes is that of Hugh-Jones et al (1973) where 77% of virus within an affected poultry shed was associated with a particle size larger than 3 microns.

[4] There is no agreed definition of what constitutes "dust". The smallest particle visible to humans is about 40 microns.

[5] There is no objective way of determining the degree of air filtration required. The use of the EU8 filter is recommended primarily because there is a reliable way of assessing its operational efficiency, using test dust No.1. This degree of filtering would prevent the escape of not only visible dust particles, but also considerably smaller particles.

[6] In order to ensure that the negative pressure is maintained when there are cross winds producing negative pressures on the leeward side of buildings by venturi effects, the within shed negative pressure may have to be run lower than -10 pascal. A commonly used level in international containment standards is -25 pascal.

[7] A minimum separation distance from any 'concentration of avian species' is included in post-arrival qaurantine standards for a number of countries, although what constitutes such a concentration is not specificed by any country. As can be seen in Appendix 2, there is also no agreement internationally regarding what separation distance is reasonable, which is perhaps not surprising given the mis-representation of the risk of airborne spread for a number of years. The EU appears to have deliberately avoided taking a stand on this in Commission Decision 2000/666/EC, specifying only that premises must be separated from poultry holdings and other bird holdings "by a reasonable distance when taking into account the epidemiology of Newcastle disease and avian influenza as regards airborne spread." Meanwhile, the Americans have settled on a separation of "half a mile", and the Canadians specify that the quarantine hatchery must be located on "a land mass distinctly separate from the mainland". The 100 metres recommended here is considered to be conservatively based on the 64 metres limit of virus detection downwind reported by Hugh-Jones et al (1973) plus a reasonable safety margin.

3.2.2 Quarantine period and testing

The quarantine period will be long enough to assess the health of hatchlings and to carry out post-arrival testing and investigate any significant mortalities[8]. This will normally be a minimum of 60 days post-hatching.

At 42 days, blood samples will be taken from a random sample of 100 birds. Again, this sampling is intended to give at least 99% confidence of detecting a seroprevalence of 5%. For importations comprising less than 100 birds at 42 days post-hatching, all will be tested. If any birds are seropositive to APMV-1 then no birds will be permitted to leave quarantine until further testing, as MAF considers is appropriate to the circumstances, is carried out.

[8] No attempt is made here to specify what level of mortality would be considered 'significant' over and above the normal background level.

Appendix 1: The risk of airborne spread

1. INTRODUCTION

Despite the limited evidence for airborne spread of Newcastle disease virus, for a number of years the Australian veterinary authorities have considered that the risk is sufficient to justify specific quarantine measures and contingency plans. New Zealand quarantine measures have tended to follow the Australian lead in this regard.

In this appendix the case made by the Australian authorities is examined in detail, resulting in a conclusion that the hypothesis of airborne spread is not sustainable.

2. THE CASE FOR AIRBORNE SPREAD

The Australian veterinary emergency plan for Newcastle disease, AUSVETPLAN, contains two statements which claim the airborne spread of Newcastle disease over large distances (ARMCANZ, 1996).

Firstly, it states :

> *In an outbreak in Ireland in 1973, farms were thought to have been infected by airborne spread up to 48 km from the initial case (McFerran, 1988).*

Secondly, it states:

> *Windborne spread has been reported up to an extreme of 64 km and will be influenced by the volume of generated virus (Alexander, in press).*

In 1998 the Australian Quarantine Inspection Service (AQIS) released their revised conditions for the importation of chicken hatching eggs (AQIS, 1998). In Appendix A of that document, under the heading "Scientific issues: Time and distance", the following statement appears:

> *The longest reported distance over which ND virus is considered to have spread by wind is approximately 40 kms.*

The above statement appears to be taken from a review of avian importation requirements of hatching eggs which was commissioned by AQIS in late 1996 (AQIS, 1997)[9]. On page 13 of that *AQIS Review*, under the subheading "2.3.d Wind", there are four statements in support of the notion of airborne spread of Newcastle disease:

> *Gloster (1983) provides details of calculations to show that there would be a 90% chance of birds in a 10,000 bird flock becoming infected as a result of windborne spread from a flock of 1000 infected birds under suitable conditions. An analysis of 2 outbreaks in Britain concluded that NDV had been transmitted aerially over a distance of 8km. (Hugh-Jones et al 1973). Spread over an even greater distance, up to 40 km was suggested as the means of spread of NDV in Northern Ireland in 1973. Approximately 40% of secondary cases of ND in England during the Essex 70 NDV outbreak were thought to be due to windborne spread (Lancaster 1977).*

[9] In this risk analysis the AQIS document will be referred to as the "*AQIS Review*".

3. ANALYSIS OF THE CASE FOR AIRBORNE SPREAD

3.1 Ausvetplan

3.1.1 First statement in Ausvetplan

> *In an outbreak in Ireland in 1973, farms were thought to have been infected by airborne spread up to 48 km from the initial case (McFerran, 1988).*

The above statement refers to the 1988 presentation by Professor J B McFerran at a workshop on avian exotic diseases which was held in Australia sometime in 1988, apparently organised by the NSW Department of Agriculture (McFerran, 1988).

At the outset it is important to note that McFerran (1988) is not a peer-reviewed paper. Rather, it is mostly an anecdotal account of the control methods used in Northern Ireland in the early1970s. Prof. McFerran's comments regarding possible airborne spread were brief and rather speculative. It appears that if an outbreak occurred downwind, and if investigators could see no obvious link in terms of human or other movements, then there was a strong tendency on the part of the authorities at that time to assume that the outbreak was due to windborne spread. However, by the time he came to present a partially revised version of the same paper at a workshop in Germany 4 years later (McFerran, 1992) Prof. McFerran had apparently modified his views on the probability of airborne spread.

In McFerran (1988), it is stated (p 17) :

> *In spite of the normal restrictions on movement, the number of cases rose quickly. Although in some of these direct contact could be established, in many no direct connection was evident. However, these cases did occur in an arc coinciding with the wind direction. Conditions in November were favourable for wind spread, with few hours of sunlight, moderate to light winds, a high humidity and temperatures ranging from 2° to 10°C. One surprising jump of 48 km was found. This was consistent with wind spread as was the development of disease on the farm. A second jump of 38 km was not consistent with wind spread as the wind did not blow in that direction during the period. Furthermore, the pattern of disease development was similar in this farm (68,000 birds) as on the initial farm, with a slow spread starting from one focal spot on one house.*

In 1992, Prof McFerran delivered another seminar (McFerran, 1992) with the same title as his 1988 paper. The relevant part of the 1992 paper was as follows (Pp 239 - 240) : [note that ~~strikeout~~ has been used to indicate the text that was in the 1988 paper but not in the 1992 paper, and **bold** has been used to show text that is in the 1992 paper but not in the 1988 one]

> *In spite of the normal restrictions on movement, the number of cases rose quickly. Although in some of these direct contact could be established, in many no direct connection was evident. However, these cases did occur in an arc coinciding with the wind direction. Conditions in November were favourable for wind spread, with few hours of sunlight, moderate to light winds, a high humidity and temperatures ranging from 2° to 10°C. One surprising jump of 48 km was found. This was consistent with wind spread ~~as was the development of disease on the farm~~.* ***However the distance travelled and the lack of cases in between leaves considerable doubt.*** *A second jump of 38 km was not consistent with wind spread as the wind did not blow in that direction during the period. Furthermore the pattern of disease development was similar in this*

farm (68,000 birds) as on the initial farm, with a slow spread starting from one focal spot on one house.

So as far as the text in Ausvetplan is concerned, it is true that in 1988 Prof McFerran was strongly advocating the airborne spread hypothesis to explain the so-called "jump" of 48 km.

However, even in 1988 Prof. McFerran suggested that there was an alternative hypothesis that could account for the pattern of Newcastle disease seen. He suggested that there could have been multiple primary outbreaks due to contaminated feed. The problem for this hypothesis was that the flocks belonged to several organisations, and they were supplied by different feed mills. But Prof. McFerran noted that the 'straights' (raw feed ingredients) were imported through a common point, and that low level contamination at that point may have been responsible.

Moreover, by 1992, Prof. McFerran was evidently distancing himself somewhat from the windborne spread hypothesis. He altered the 1988 text to include the words “considerable doubt” because of the distance of the jump and the absence of any other outbreaks in between, and he removed the ambiguous phrase immediately prior to that. Ausvetplan has included the former statement but not the latter. But even without such detailed examination of the differences between the 1988 and 1992 papers, it is difficult to see how the Ausvetplan authors could have accepted the assertion of a 48 km jump due to windborne spread when at the same time there was a 38km jump against the direction of the wind. Rather than accept uncritically the 'conventional wisdom' that airborne spread was the best explanation for the “unexpected jumps”, the most reasonable conclusion to reach would be that there were a number of problems which complicated tracing, a position which Prof McFerran seems to have adopted by 1992.

3.1.2 Second statement in Ausvetplan

Windborne spread has been reported up to an extreme of 64 km and will be influenced by the volume of generated virus (Alexander, in press).

The reference list in Ausvetplan shows the 'in press' article by Alexander is “Newcastle disease - means of spread”, in a book (to be) published by Kluwer Academic Press. Although it is not reflected in the 1996 Ausvetplan, the book was actually published in 1988[10]. More importantly, the above statement in Ausvetplan is a serious misquote of the reference. Alexander (1988) simply explains that Hugh-Jones et al (1973) reported finding:

“*detectable levels of virus 64* ***metres****, but not 165 metres, downwind of infected premises*” [emphasis added].

However, Ausvetplan has re-interpreted Alexander as:

"*windborne spread has been reported up to an extreme of 64 km*".

The Ausvetplan interpretation is incorrect on two counts. Firstly, Hugh-Jones et al (1973) did ***not*** report airborne spread, but only that “detectable levels of virus” were found downwind, and secondly the maximum distance that virus was found was 64 metres, not 64 kilometres.

Further attention is given to the Hugh-Jones et al (1973) paper in section 3.3 of this appendix.

[10] The title of the chapter by DJ Alexander in that book was in fact "Newcastle Disease: Methods of Spread".

3.2 AQIS Review

This analysis will focus on each of the four statements in the *AQIS Review*, and will show that arguments for airborne spread are based on misinterpretation of the literature.

(a) "Gloster (1983) provides details of calculations to show that there would be a 90% chance of a 10,000–bird flock becoming infected as a result of windborne spread from a flock of 1,000 infected birds under suitable conditions."

This analysis refers to the above paper as Gloster (1983a). Its conclusions were not as clear as the *AQIS Review* suggests. Whereas the *AQIS Review* states that Gloster's paper "provides details of calculations to show …", in fact the paper merely describes Gloster's model of airborne spread. The model was based on Gloster's earlier work modelling airborne spread of foot and mouth disease (FMD) virus, and the preliminary work by Smith (1964). In order to adapt the FMD model to ND, Gloster had to make a number of assumptions, as there were few data available on a number of important questions. These assumptions included :

- the number of virus particles emitted by an infected bird per day was assumed to be 10,000 particles per bird per day,
- the emitted virus was assumed to be mixed homogeneously in air, so that the concentration of virus particles in inhaled air could be assumed to be constant
- the target birds assumed to inhale 500 ml of air per minute for a period of 3 hours
- the probability of any individual particle resulting in infection if inhaled was assumed to be 0.1

The logic of Gloster's conclusion was as follows:

IF a 1000-bird poultry unit is infected with NDV

AND IF all of the 1000 birds are at the same stage of infection

AND IF each of the 1000 birds at the same stage of infection are excreting the same amount of virus (assumed 10,000 particles per bird per day)

AND IF there is a clear night with a slight breeze

AND IF there is a 100,000 bird unit 5 km downwind

THEN the probability of at least one of the 100,000 birds in the downwind unit becoming infected is 0.9

As is always the case with such predictive models, the assumptions are the key to determining the conclusions, and validation is obviously impossible except by natural experiments. Given the large number of assumptions, the outputs from this sort of model should be treated with caution. Gloster himself stressed that his model could "only give a very approximate estimate of the potential for airborne disease" and that there were four topics needing further investigation before the potential for airborne spread could be quantified accurately for any given outbreak.

Others have also been more circumspect in their interpretations of the Gloster (1983a) paper. In particular, Alexander (1988), in his review of the methods of spread of Newcastle disease commented as follows :

> *Gloster, reviewing the prerequisites determined for the airborne spread of other viruses and the ability of NDV to fulfil these, concluded that such spread was feasible but difficult to predict with certainty due to lack of information on several important parameters.*

Thus the words used in the *AQIS review* imply a level of precision in the Gloster (1983a) prediction which is not warranted.

(b) *"An analysis of 2 outbreaks in Britain concluded that NDV had been transmitted aerially over a distance of 8 km (Hugh-Jones et al 1973)."*

The quoted paper (Hugh-Jones et al, 1973) does not contain anything to justify such a conclusion. It appears likely that there was a referencing error in the *AQIS Review* and that the writer was actually referring to a further paper by Gloster (Gloster, 1983b). This paper was an attempt to analyse historical data from two previous ND outbreaks (East Sussex 1959/60, and Kent 1969) for evidence of airborne spread using the model presented in the earlier paper (Gloster, 1983a). In order to apply the airborne spread model, Gloster (1983b) had to make five key assumptions, including that the index case was the primary case in the area.

Gloster (1983b) presented five conclusions, the second of which reads as follows :

> *Airborne infection may have occurred up to 8 km from either a velogenic or mesogenic virus source.*

Not only is the wording of Gloster's conclusion significantly different to the words used in the *AQIS Review,* which stated :"An analysis of two outbreaks … concluded that NDV *had been* transmitted aerially over a distance of 8 km" (italics added), the *AQIS Review* neglected to mention that Gloster had expressed reservations because:

> *"…a rigorous meteorological investigation has not been possible due to the shortcomings in the data."*

and that Gloster suggested the results of his paper should only:

> *"…act as a pointer for other more rigorous investigation."*

Moreover, although the model on which Gloster based his work was a modification of the successful model of FMD spread developed by Blackall & Gloster in the early 1980s, it was never validated for Newcastle disease and no further research has been undertaken on this matter since.

(c) *"Spread over an even greater distance, up to 40 km, was suggested as the means of spread of NDV in Northern Ireland in 1973."*

Although this statement in the *AQIS Review* is not referenced, it apparently refers to the 1988 presentation by Professor J B McFerran at the NSW Department of Agriculture avian exotic diseases workshop (McFerran, 1988), which has already been discussed above with respect to the same comment appearing in Ausvetplan.

(d) *"Approximately 40% of secondary cases of ND in England during the Essex 70 NDV outbreak were thought to be due to windborne spread (Lancaster 1977)."*

A close examination of the review by Lancaster (1977) and the primary literature upon which his review is based, will show that the *AQIS Review* has again overstated the case for airborne spread. On page 161 of Lancaster's review article (Lancaster, 1977), the following statement appears:

> *Allan (1974) was of the opinion that the rapid airborne transmission in England of the Essex 70 virus precluded the success of eradication by slaughter; although perhaps less than 40% of the secondary cases were considered to be due to windborne infection (Allan and Stuart (1974)).*

Firstly, there are subtle but important differences in emphasis between the words used by Lancaster (1977) and those used in the *AQIS Review*. Secondly, in order to see why Lancaster (1977) chose those particular words it is necessary to examine the two papers cited.

At the outset, both of the papers cited (Allan, 1974, and Allan and Stuart, 1974) are non-peer-reviewed conference proceedings. In the Allan (1974) paper (which interestingly does not contain a single reference), Bill Allan gave his views on the development of the Essex outbreak of ND in 1970, which apparently resulted in rapid death of birds with predominantly CNS and respiratory signs. On the second page of his paper he explained that one of the reasons that eradication was not even attempted in the UK was that the country was in the middle of a major FMD outbreak. Resources were apparently not available to even attempt eradication of ND.

Bill Allan then added the following observation:

> *My personal opinion is that if we had tried to slaughter out the disease in Essex, the airborne transmission was such that we would not have succeeded.*

While it is difficult to evaluate Bill Allan's personal opinion so long after the event, it is easy to imagine that as the veterinary authorities in the UK were barely coping with a major FMD outbreak, the careful investigation of traces on and off poultry farms was perhaps a task that was simply too much to undertake. Under such circumstances, and given the wide acceptance of airborne spread of FMD virus from France, it is perhaps not surprising that the theory of airborne spread was attractive for those trying to explain the unexpectedly rapid spread of ND in the UK. However, careful reading of the paper by Allan and Stuart (1974) reveals why Lancaster (1977) chose the words "perhaps less than 40%" when assessing how much spread *could* have been by the airborne route. On page 410 of the Allan and Stuart (1974) paper the following statement appears:

> *Transmission from farm to farm was studied and although windborne infection could be shown to be the most probable cause of spread, it is thought that even in the most densely populated areas this did not give rise to more than 40% of the secondary cases.*

Allan & Stuart did not explain the basis for considering that windborne spread was the most probable cause of spread. However, they went on to explain that :

> *Examination of the units infected showed that the most likely units to become infected were those belonging to the same producer group and mechanical transfer was felt to be responsible for about 60% of all disease.*

All that is being said is that those outbreaks which were not identified as likely to have been due to mechanical transmission might have been due to airborne spread. It seems that there was good reason to consider that introduction of infection was by mechanical means for 60% of outbreaks, so it followed that not more than 40% could possibly have been due to airborne spread. But that does not mean that 40% of secondary cases **were** caused by airborne spread, and the authors did not suggest that was the case.

The personal views and inferences made in the 1974 papers cited do not constitute proof of airborne spread, and neither do they justify the *AQIS Review* statement attributed to Lancaster (1977). Thus, the *AQIS Review* has overstated the case for airborne spread.

3.3 Hugh-Jones et al (1973)

Since the Hugh-Jones et al (1973) paper is the only paper in the literature that presents any objective data in support of the hypothesis of airborne spread, it warrants particularly careful reading. The paper describes how the authors investigated outbreaks of ND in the UK caused by the Essex 70 virus, which apparently "affected the respiratory tract to an unusually high degree."

Farm A comprised eight poultry houses, each of which had been stocked with 8500 birds 18 days before. The houses were arranged side by side, each aligned more or less in a north-south direction, bounded by a hedge on the southern side. At the time of the visit, there were 4000 'sick birds' in House 3, which was the third in line from the west. Air was sampled at a point 55 metres downwind from the corner of House 3. Over approximately half an hour, 33 thousand litres of air were sampled, and a low titre of ND virus was found (2 $\log_{10}$ ELD_{50} per 33,000 litres of air)[11].

Two points arise from this. Firstly, 2 logs is not very much virus. The infectious dose with Herts 33/56 for 3-week-old chickens has been estimated at 4 $\log_{10}$ EID_{50} by the oral route[12], that is, for virus introduced directly into the oesophagus (Dennis Alexander, personal communication with H Pharo, 3/8/2000). Thus the 2 logs of virus found 55 m downwind comprises only one one-hundredth of an oral chicken infectious dose.

Secondly, thirty-three thousand litres is a very large volume of air. By comparison, the tidal volume of a domestic chicken is approximately 33 ml (Whittow, 2000) which means that 33,000 litres of air represents a million chicken breaths. Since the respiratory rate of a chicken is on average 23 breaths per minute (Whittow, 2000), an average chicken will inhale and exhale about 760 ml of air per minute. In other words, 33,000 litres of air represents almost 45,000 chicken minutes of breathing time. Of course we do not have any idea of the distribution of the virus in air. The distribution could be very uneven, in which case some of the air would contain very much more virus than on average. Nor do we know what the infectious dose of a chicken is by the respiratory route. So all we can say is that there was less than one hundredth of an infectious dose of virus recovered over about half an hour in one million chicken breaths of air.

[11] A virus titre of 2 $\log_{10}$ ELD50 means 100 times the dose of virus that would kill 50% of embryonated eggs.

[12] That is, the titre of virus necessary to infect 50% of chickens by the direct oesophageal route was 10000 times the titre of virus that if injected into fertile eggs would infect 50% of them. [Note: ELD50 and EID50 are about the same for virulent ND viruses].

It is not clear from Hugh-Jones et al (1973) whether there was any evidence of airborne spread either within House 3 or to any of the other seven houses on Farm A. Since five of the other houses were more or less downwind of House 3, one might have expected that if there were any potential for airborne spread, it would have manifested in other houses. The point is that although windborne virus was detected (a small amount), no airborne spread of disease was reported.

On Farm B, the five houses were stocked with a total of 51,000 day old chicks on 5 October, and 39 days later "excess deaths" were noted in two of the houses - House 3 and House 5. Sampling in and around House 5 took place 4 days after that, by which time 11,000 of the 14,000 birds in the house had died, and there were sick and moribund birds showing respiratory distress. The authors did not report what happened in House 3 where "excess deaths" had been noted in the 13,000 birds at the same time as the "excess deaths" in House 5. Again, it is not clear whether there was any evidence of airborne spread within House 5, and secondly, whether there was any disease in Houses 1, 2 & 4, where there were 7,500, 7,500 and 9,000 birds respectively. The fact that two of those houses were directly downwind from House 5 is significant.

On Farm B airborne virus was again found, this time 64 metres downwind, but much less virus than at Farm A - only 1.45 $\log_{10}$ ELD_{50} per 33,000 litres of air[13]. No virus was found 164 metres downwind from the infected house.

On both Farm A and Farm B, the air sampler was set up inside the infected house, and run with the ventilating fans off. Even so, less than 6 logs of virus were collected[14] in each case, again in 33 thousand litres of air collected over about half an hour.

Two relevant observations arise from this important piece of work. Firstly, as already mentioned, it is difficult to assess the importance of the very small amounts of virus (even inside the infected houses) that were found in the very large volume of air sampled over a relatively long period. Secondly, it might have been expected that if there was a potential for airborne spread it would have been seen in the nearby downwind houses - that is, Houses 1 and 2 on Farm B, and at least House 4 on farm A (although it could be argued that if there had been airborne virus the hedge might have channelled it from House 3 to Houses 4-9 on Farm A).

Of interest is the air sampling that was carried out at Farm C on day 37 of the outbreak, when birds were dying in large numbers (despite having had multiple vaccinations with live ND vaccines). By the end of the outbreak one week later, 40% of the birds had died. A different air sampler was used in order to estimate the particle size of the airborne virus. It was found that 77% of the virus was associated with particles above 3 microns, and the remaining 23% of virus was associated with particles less than 3 microns in size.

[13] The 1.45 logs of virus found 64 metres downwind in 33,000 litres of air is less than one-hundredth of a single chicken infectious dose.

[14] The virus titre was 5.7 $\log_{10}$ ELD50 per 33,000 litres of air

3.4 Airborne spread - summary and conclusion

This risk assessment suggests that there is little evidence for airborne spread being of significance in the epidemiology of ND.

While it might not have been palatable for veterinary services concerned, the simpler explanation for the observed spread in both the UK and in Northern Ireland is that there were some problems with quarantine and/or tracing. As Alexander (2001) has noted, for both farmers and those involved in the control of disease, airborne spread can sometimes seem an attractive proposition. If the infection arrives by such a natural method then no person can be blamed or suffer guilt for introducing the virus. Alexander also suggests a sensible and pragmatic approach for evaluating the importance of airborne spread :

> *In recent years airborne spread has not been an issue in reported outbreaks and there has nearly always been an alternative, more likely, cause, particularly the movement of poultry and the agency of man. If windborne spread over any distance was normally a significant factor in ND epizootics then there would seem little that could be done in terms of biosecurity short of maintaining poultry in HEPA-filtered environments. That this is not normally the case means that other measures can be employed to reduce the risk of introduction to a farm or flock.*

It appears from the above that the risk of airborne spread of ND has been significantly overstated, both by UK veterinarians in the 1970s, and more recently by AQIS. However, the risk of such spread has been overstated for so long that in some circles it has almost become part of the 'conventional wisdom'. Those who uncritically accept that conventional wisdom appear to believe that ND has a huge potential to spread by the airborne route over vast distances and that a range of restrictions are therefore necessary to manage that risk. That view has been used to justify requirements for HEPA filtration in poultry quarantine buildings in Australia and New Zealand. HEPA filtration typically involves 99.97% arrestance to 0.3 microns which is considerably smaller than the 3 microns which Hugh-Jones et al (1973) found to be the lower end of the range of particle sizes which carried 77% of virus.

3.5 Airborne spread - a final thought?

A major problem with modelling airborne spread is the necessary assumption that the index case is the primary case. This is vitally important to any assessment of airborne spread, as explained by Smith (1964) and Gloster (1983b). That assumption allows the plotting of airflows and estimated virus plumes from the supposed primary case, and estimating spread from it. But if the farm that was assumed to be the primary case was in fact a secondary case or if there were farms where infection was present for some days prior to detection, or indeed if a large number of farms were exposed simultaneously through contaminated feed supplies, then the assumption of the known single primary case would not hold, and attempts to plot airborne spread using models like that of Gloster (1983b) would lead to invalid conclusions.

Almost as a footnote to this saga, over 25 years after these outbreaks in the 1970s (which occurred simultaneously in a number of countries in Europe as well as in the UK, Northern Ireland, and several US states), molecular techniques have shown that the viruses involved were essentially the same, and that they were most likely transported around the world in poultry feed ingredients.

In the case of Northern Ireland, the recent evidence suggests that the virus was imported in feedstuffs from Rotterdam. There were then only two importers of such feedstuffs in Northern Ireland, who sold the imported ingredients on to the 15 feedmills which were operating at that time. The most reasonable conclusion is that at least one of the importers imported at least one contaminated batch of feed, and that this was spread via feedmills to poultry flocks in poultry feed (Martin Hugh Jones, personal communication with H Pharo, 18/7/2000). Therefore there was probably no single primary case, rather there was simultaneous introduction of infection onto a large number of farms, which means that attempts to model airborne spread on the assumption from the index case could not have been valid.

At the time of the 1984 ND outbreak in the UK, careful epidemiological investigation was able to rule out airborne spread as being of any practical importance. Rather it was shown that the origin was imported feedstuffs on the Liverpool docks which had became contaminated with faeces of pigeons carrying the so-called pigeon variant APMV-1 virus. The consequent feed-borne spread of pigeon virus resulted in 23 outbreaks in commercial poultry flocks, the majority of which were primary. There was no evidence of any airborne spread (Alexander, 2000).

Unfortunately no epidemiological assessment is available for the series of ND outbreaks which have occurred since 1998 in Australia, so it is not possible for this risk assessment to assess any role, if any, that airborne spread may have played in that country.

APPENDIX 2: Post-arrival quarantine requirements

1. NEW ZEALAND

MAF currently has two standards for quarantine of birds, known as high security and medium security. Both medium and high security standards have been developed to address the risk of ortho- and paramyxoviruses, and they have been based on standards initially developed in Australia.

General biosecurity measures common to both high and medium-security facilities are:
- all persons must shower out
- dead birds must be double bagged and placed into sample containers for shipping to lab
- liquid waste system must be vented back through the same air filter system
- liquid waste must be chlorinated or autoclaved
- solid waste is kept on the facility and if the quarantine is successful it is allowed off at the end of the period

1.1 High Security

High security quarantine was developed in the early 1990s from Australian standards, intended for the importation of live birds.

The main features of the standard relate to airflow, and are:
- construction is a building within a building
- inside the inner building, a negative pressure (-10 pascals) is maintained
- the air coming into the inner building is filtered using a HEPA[15] filter
- outlet air is filtered by two HEPA filters in series

1.2 Medium Security

This standard has been around in various forms since the 1970s, and is the one that is currently used for imported hatching eggs.
The main features are:
- Single-skinned buildings.
- Inlet air is not filtered (there is only a 1.25 mm mesh to stop large particulate matter)
- Air movement is controlled to the extent that there is mechanical assistance to propel the air towards the outlet fans, which are equipped with filters.

However, there are concerns that these facilities are vulnerable to cross winds, resulting in significant air flow out of the air inlets on the leeward side of the building.

[15] HEPA filtration gives 99.9% arrestance of particles greater than 0.3 microns Note this is an order of magnitude smaller than the particle size reported by Hugh-Jones et al (1973) to be carrying 77% of recovered virus.

There are also concerns regarding the clogging of outlet filters by the very considerable dust which is generated by the hatchlings.

There are two levels of air filtration in this standard, according to the buffer distance i.e. the distance between the facility and any (other) poultry:

a) Buffer distance 500 metres
- outlet air is filtered to 91% arrestance efficiency (for particles of 5 microns or larger)

b) Buffer distance 8 metres
- outlet air is filtered to 98% arrestance efficiency (for particles of 5 microns or larger

Other features are:
- liquid wastes can go into a municipal sewer or can be sterilised on the facility as for high security system
- solid wastes are kept on the facility as is the case for high security system

2. OTHER COUNTRIES

2.1 Australia

The AQIS current official position regarding the requirements for quarantine for imported hatching eggs is presented in the document "Import Risk Analysis: Revised Conditions; importation of hatching eggs of commercial poultry into Australia" published 24 December 1998 (AQIS, 1998). Concerns over the airborne spread of Newcastle disease have influenced the AQIS decision to require HEPA filtration of outlet air from quarantine stations where imported birds or hatching eggs are held in post-arrival quarantine. The possibility of airborne spread has also influenced the requirements for siting of approved quarantine facilities for live birds and hatching eggs. AQIS is currently involved in a number of import risk analyses in which Newcastle disease will be considered. In the course of these IRAs the evidence in relation to modes of spread of Newcastle disease will be assessed (David Banks, AQIS, personal communication with SC MacDiarmid, 25 August 2000).

2.2 Canada

High security quarantine is required for importing animals from countries for which there is insufficient knowledge of or confidence regarding veterinary infrastructure, surveillance or diagnostic capabilities. It is also required when importing animals of species with which the Canadian authorities have limited experience or a lack of confidence in the performance of diagnostic tests.

When air filtration is required it is always HEPA filtration of exhausted air. Alternatively, the quarantine facility may be located on a land mass distinctly separate from mainland Canada and without commercial livestock operations.

2.3. European Union

2.3.1 Poultry hatching eggs from the EU

Covered by Council Directive 90/539/EEC

- Safeguards are flock and area disease freedom and establishments must be 'approved by a competent authority'
- No post-arrival quarantine or testing of hatchlings is required

2.3.2 Poultry hatching eggs imported from third countries

Covered by Council Directive 96/482/EC

- Imports are permitted only from listed approved countries
- Safeguards are offical approval of establishments of origin, premise and area disease freedom (25 km, 30 days), and clinical inspection of the flock on the day of consignment
- Imported eggs are hatched in quarantine, and chicks kept in quarantine for 3 weeks. Veterinary examination at the end of the quarantine period, with more frequent examination (and sampling) 'if warranted'. Virus isolation (ND/AI) is attempted from chicks which die in quarantine
- No air filtration is required in quarantine facilities

2.3.3 Commercial consignments of captive birds from third countries

Such consignments are covered by Commission Decision 2000/666/EC.

Until the recent harmonisation of EU rules, the UK required HEPA filtration for exhaust air from post-arrival quarantine facilities for captive wild birds from outside the EU. However, those requirements have been superseded by Commission Decision 2000/666/EC, under which the birds must originate from 'registered holdings' (Article 2) and be accompanied by a health certificate (Annex A) which specifies requirements for country/area/holding freedom from clinical disease (ND, AI, psittacosis).

The imported birds must go into quarantine for at least 30 days. Article 1 and Annex B (chapter 1) contain definitions of quarantine facility and quarantine centre. In both definitions the premises must be separated from poultry holdings and other bird holdings "by a reasonable distance when taking into account the epidemiology of Newcastle disease and avian influenza as regards airborne spread." This appears to imply that the EU has carried out an assessment of the risk of airborne spread. But the distance is not specified. No HEPA or other air filtration system is required in quarantine premises.

While in quarantine the birds will be subjected to sampling and/or testing of birds or sentinel chickens according to Annex C. If there is any AI or ND infection of birds or sentinels, the entire shipment is destroyed (although it may be possible to salvage the shipment if all surviving birds are free of virus 30 days after last clinical signs - in such a case the birds will be kept in quarantine for 60 days after the last clinical signs).

2.3.4 Non-commercial consignments of captive birds from third countries

Consignments of non-commercial captive birds are subject to 35 days quarantine in a Category II quarantine (no air filtration).

2.4 United States of America

The USDA regulates the importation of all birds, poultry, and hatching eggs of poultry into the United States. Poultry hatching eggs from countries designated by USDA as free of exotic Newcastle disease are not required to be quarantined. Eggs from countries not designated as ND free must be hatched and brooded under veterinary supervision, and the chicks must remain in quarantine for 30 days after hatching. During quarantine, the hatching eggs and poultry from such eggs are subject to any inspections, disinfections, and diagnostic testing as may be required by the USDA.

Detailed standards for privately owned bird quarantine premises have been developed by the USDA (US Government, 1999). Such facilities :

> *"must be situated at least half a mile from any concentration of avian species, such as, but not limited to, poultry processing plants, poultry or bird farms, pigeon lofts, or other bird quarantine facilities. Factors such as prevailing winds, the efficiency of the air filtration system of the quarantine facility, possible exposure to poultry or birds moving in the local traffic, etc., shall be taken into consideration."*

The construction standards for such facilities specify double screening of all openings to the exterior, but the only requirement as far as ventilation is concerned is for

> *"a ventilation capacity sufficent to control moisture and odor at levels that are not injurious to the health of the birds in quarantine."*

There is no requirement for filtration of outlet air.

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