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Avian influenza reference guide Version 1.0

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Our work includes tackling flooding and pollution incidents, reducing industry's impacts on the environment, cleaning up rivers, coastal waters and contaminated land, and improving wildlife habitats.

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Steve Killeen

Head of Science

Executive summary

Avian influenza is a bird disease that may occasionally infect other animals and man. In recent years, a strain of highly pathogenic avian influenza has arisen in the Far East (Asian HPAI H5N1) and spread steadily across Asia, Africa and Europe. This report outlines our current understanding of the behaviour and risks of highly pathogenic avian influenza (HPAI) and in particular Asian HPAI H5N1. It presents the scientific information behind our decisions and our advice in the event of an outbreak in the bird population, but does not address the wider human health issues of, for example, a possible human influenza pandemic.

In response to an animal disease outbreak, we will work with and support partner organisations to minimise the environmental impact of any outbreak. Our work will include advising government, determining applications for waste disposal activities and advising on pollution prevention. The Department for Environment, Food and Rural Affairs' contingency plan sets out the roles, responsibilities and actions required in the event of an outbreak of avian flu. It includes a disposal strategy and states that commercial fixed plant incineration is preferred over rendering and, in turn, over licensed commercial landfill. A risk-based review of disposal options is being carried out by the State Veterinary Service and is likely to confirm that controlled processing options are preferred over uncontrolled processing which is, in turn, preferred over burial.

Since 1997, more than 200 million poultry and ducks have been killed by Asian HPAI H5N1 or culled in efforts to control the spread of disease. More than 140 people have died as a result of infection by the virus. There have been no outbreaks in the UK, although a dead whooper swan infected with the Asian HPAI H5N1 virus was discovered at Cellardyke in Scotland in April 2006.

The Environment Agency will have a key role to play if large numbers of farmed birds become infected with the Asian HPAI H5N1 virus and die naturally or are culled. Carcases and other infected materials (such as litter and faeces) present a range of biological and chemical hazards. These, along with chemicals used to cleanse and disinfect equipment and property, could affect the environment. The main environmental risks are likely to come from the pollution of groundwater from disposal activities.

Biological hazards include HPAI viruses and bacteria that can infect other birds and animals, including humans. In addition to the Asian H5N1 virus, the principal biological hazards of potential concern are *Campylobacter* and *Salmonella*. Chemical hazards include substances used to cull birds, decomposition products, cleansing and disinfection chemicals, veterinary medicines and substances arising from disposal processes such as incineration.

Infectious virus particles can exist in very high numbers within the flesh, droppings and other secretions of infected animals. They are released from live birds by respiratory secretions or faecal deposits and from the fluids of decomposing dead birds. HPAI viruses can persist outside their hosts and remain infectious for long periods of time. Survival in the environment is aided by low temperatures, neutral pH, high levels of moisture and high levels of organic matter. No specific data appears to exist on the persistence of Asian HPAI H5N1 in soil or air, although it is expected to persist for some time in soils rich in moisture and organic matter. Survival in air is expected to be brief.

There is evidence that avian flu viruses can survive for many days in the water environment. We tentatively estimate a half-life of 75 days in UK groundwaters. We suggest that bird infection could occur at virus concentrations between 10 – 1,000

viruses/ml water or above. It is important to note that these numbers are very uncertain, based as they are on very limited data and a number of assumptions.

Live infected birds are capable of transporting the Asian HPAI H5N1 virus. In addition, for some avian flu viruses, there is evidence that they can be transported on the clothing of poultry workers and, indeed, on vehicles or anything else that may be contaminated with infective secretions or fluids, especially faeces. Precautions should be taken to prevent inadvertent transport of viruses.

Once the Asian HPAI H5N1 virus has left host birds the most significant environmental pathways are considered to be via surface and groundwater, although there is no specific data for UK water bodies. Work is underway to develop risk models to simulate the possible fate and transport of the virus in UK groundwaters.

Although primarily an avian pathogen, the Asian HPAI H5N1 virus can infect other hosts (mainly mammals) and kill them. British wildlife, including badgers, ferrets and foxes, are potentially at risk if they come into contact with the virus (for example, by consuming infected carcases).

Precautions should be taken when working in close proximity to infected birds or those suspected of being infected.

Contents

1 Introduction

Key facts

This report outlines the Environment Agency's current understanding of scientific knowledge on avian influenza and in particular on the Asian HPAI H5N1 virus. It presents the scientific information behind our decisions and our advice to others. In response to an animal disease outbreak, we will work with and support partner organisations to minimise the environmental impact of any outbreak. Our work will include advising government, assessing applications for waste disposal activities and advising on pollution prevention.

The Department for Environment, Food and Rural Affairs' contingency plan defines roles, responsibilities and actions required in the event of an outbreak of avian flu. This includes a hierarchy of disposal options in the order of commercial fixed plant incineration, then rendering, then licensed commercial landfill. A risk-based review of a longer list of disposal options is underway and is likely to confirm that controlled processing options are preferred over uncontrolled processing which is, in turn, preferred over burial.

Worldwide, over 200 million poultry and ducks have been killed by Asian HPAI H5N1 or culled in efforts to control the spread of disease. More than 140 people have died as a result of Asian HPAI H5N1 infection. There have been no outbreaks in the UK to date, although a dead whooper swan was discovered at Cellardyke in Scotland in April 2006.

1.1 Introduction

The likelihood of an outbreak of highly pathogenic avian influenza (HPAI, commonly known as bird flu) in the UK appeared to increase during the first half of 2006, with the spread of the Asian HPAI H5N1 virus to poultry in five EU member states and to wild birds in another eight, including a single whooper swan found dead in Scotland. The Environment Agency has a key role to play both in advance and in the event of an outbreak. This role includes assessing and managing environmental risks and issuing permits for waste management/disposal activities. It is critically important that our decisions, and our advice to government and other organisations, are based on the best available scientific information.

This report presents the scientific information behind our decisions and our advice to government, poultry farmers, the general public and other affected parties. The report's primary audience is Environment Agency staff, and it reflects our current understanding of scientific knowledge at the time of writing (September 2006). The report focuses primarily on the Asian HPAI H5N1 virus, which has spread westwards from Asia since it first appeared in the late 1990s (see Section 2.2.1 for an introduction to virus classification and terminology). This strain has recently arrived in Western Europe and Africa and has infected both wild and domestic birds as well as several mammalian species including humans. Asian HPAI H5N1 has a mortality rate above 50 per cent in those humans infected to date.

It is important to recognise that animal carcases and associated materials such as faeces and litter contain a range of biological and chemical hazards, and that avian flu viruses are not the only source of risk to the environment, animal health and public health. Examples of other hazards include decomposition products such as ammonia, other organisms such as *Campylobacter* and *Salmonella species*, and chemicals such as veterinary medicines.

1.2 Spread and control

The current Asian HPAI H5N1 virus first appeared in the Far East in the late 1990s. The highest number of individual outbreaks reported have occurred in Vietnam, Indonesia and Thailand (Table 1.1). The virus spread westwards during 2005, with cases recorded in early 2006 in a number of countries in Europe and Africa. At the time of writing, over 200 million poultry and ducks have been killed by the virus or culled in efforts to control the spread of disease. By August 2006, more than 140 people had died as a result of H5N1 infection (WHO data taken from http://www.who.int/en/).

Table 1.1: Outbreaks of Avian Influenza (subtype H5N1) in poultry up to 4 September 2006 (data from the World Organisation for Animal Health website http://www.oie.int/eng/AVIAN_INFLUENZA/home.htm; select 'update on AI situation')

Vietnam	2314
Thailand	1080
Indonesia	215
Turkey	176
Romania	168
Russia	121
China	87
Nigeria	69
Egypt	32
Ukraine	24
Korea	19
Cambodia	18
Malaysia	15
Afghanistan	13

Pakistan	12
Myanmar	11
Israel, Sudan	9
Palestinian territories	8
Hungary, India, Japan	7
Burkina faso	4
Ivory Coast, Iraq, Niger	3
Albania, Azerbaijan, Laos	2
Cameroon, Denmark, Djibouti, France, Germany, Jordan, Kazakhstan	1

1.2.1 Spread

The Asian HPAI H5N1 virus has infected birds and mammals over a wide geographic area encompassing Asia, Europe and Africa in a relatively short period of time. It appears that movements of infected poultry, the transport of infectious material by man (such as faecal matter on gloves and boots) and wild bird movements may have all contributed to this spread (Butler, 2006b). Wild birds are known to be capable of flying whilst infectious and, indeed, may be infected and excreting the virus without showing any adverse effects (Webster *et al*., 2005a).

McQuiston *et al*. (2005) evaluated risk factors during an outbreak of low pathogenicity avian influenza (LPAI) in Virginia, USA in 2002. Their results suggested that an important factor contributing to rapid early spreading of the virus (a strain of H7N2) amongst commercial poultry farms was the transport of dead birds for off-farm rendering. The authors highlighted other significant risk factors which included mammalian wildlife on farm (but not domestic mammals or wild birds) and non-family caretakers. This short outbreak required five million birds to be culled.

1.2.2 Control of spread

Successful disease control in some countries has been achieved by implementing UN FAO (United Nations Food and Agriculture Organisation) guidelines (UN FAO, 2005). These require the setting up of zones of control and eradication, which can involve the slaughter of large numbers of birds if pre-emptive culling is considered necessary (for example, outbreaks in Hong Kong required the killing of over 1.5 million birds).

EU Directive 2005/94/EC (OJ, 2005) outlines the control methods to be used in EU Member States.

Different disposal options may be required for carcases and litter/faeces and, indeed, for different species (see Section 5.1 for information on the host range of Asian HPAI H5N1) as noted by the UN FAO (UN FAO, 2005).

Defra's contingency plan (Defra, 2005) defines the roles, responsibilities and actions required in the event of an outbreak in the UK. It identifies a hierarchy of disposal options in the order of:

- commercial fixed plant incineration;
- rendering;
- licensed commercial landfill.

The State Veterinary Service (SVS) and the Department for Environment, Food and Rural Affairs (Defra) are coordinating a review of the various options available for disposing of contaminated materials in the event of an outbreak of Asian HPAI H5N1 in the UK. The review has included representatives from the Environment Agency and other responsible bodies and, whilst not yet completed, is likely to show, that controlled processing is preferred over uncontrolled processing which, in turn, is preferred over burial.

1.3 The Environment Agency's role

The Environment Agency is the leading public organisation for protecting and improving the environment in England and Wales.

During animal disease outbreaks, we will work with and support partner organisations – including Defra, the SVS, local authorities and landowners – to minimise the environmental impact of any outbreak. We will:

- provide expert advice to the government, particularly on waste management, where our advice will focus on the disposal sites we regulate;
- assess applications for waste disposal activities (including carcases, manures and washwaters) where required;
- advise on pollution prevention issues such as the siting and operation of cleansing and disinfection facilities.

During outbreaks we will, where appropriate, provide liaison officers at strategic (Cabinet Office and/or Defra), tactical (National Disease Control Centre) and operational (Local Disease Control Centres) command levels. We will also, where necessary, attend Regional Civil Contingencies Committee meetings.

Our role does not include a significant lead involvement in air quality issues or health impacts on the wider population. Such matters are dealt with in partnership with local and health authorities. Advice should be sought from health professionals where impacts on public health are concerned.

1.4 Contents

The information presented in this report is intended to help the Environment Agency assess the risks to the environment, to guide our advice to government and other parties and to aid us in carrying out our regulatory responsibilities. Our approach to assessing environmental risks is described in Guidelines for Environmental Risk Assessment and Management (DETR *et al.,* 2000) and is founded on the source-pathway-receptor model. This requires a **source** of the hazard (such as live or dead birds and their faeces), a **receptor** that might be exposed and suffer adverse consequences (such as another bird) and a **pathway** that could mediate such exposure (such as direct contact with infected faeces). When assessing risk, we seek to understand the likelihood that receptors may suffer harm and the magnitude of the effects following exposure to hazards such as HPAI viruses. Such assessments may be undertaken in advance of any outbreak (such as in the review of disposal options) or to guide site-specific decisions if an outbreak occurs. The structure of this report reflects the source-pathway-receptor model:

- Section 2: Hazards
- Section 3: Sources
- Section 4: Pathways
- Section 5: Receptors
- Section 6: Bibliography
- Section 7: Glossary

Supporting information is presented in a number of appendices.

1.5 Acknowledgements

We are grateful to colleagues within the Environment Agency, the Health Protection Agency, the State Veterinary Service and the Veterinary Laboratories Agency, and to Dennis Alexander for comments on earlier drafts of this report.

2 Hazards

Key facts

Carcases and other infected materials (such as litter and faeces) are associated with a range of biological and chemical hazards.

Biological hazards include avian flu viruses and bacteria that can infect other birds and animals, including humans. Bacterial hazards of principal concern include *Campylobacter* and *Salmonella*.

Chemical hazards include the substances used to cull the birds, veterinary medicines, decomposition products and chemicals produced during the treatment and disposal of wastes (such as combustion products). In addition, chemicals used to cleanse and disinfect equipment and property could affect the environment.

2.1 Introduction

The Environment Agency's core role during an animal disease outbreak is to minimise environmental impacts by regulating the disposal of wastes and providing pollution prevention advice. In particular, we will play a significant role if large numbers of farmed birds[1] are culled, necessitating the disposal of carcases and associated materials (such as litter and faeces) and the cleansing and disinfection of equipment and property (such as chicken sheds). Deaths of small numbers of wild birds (or mammals) pose a much lower risk to the environment and are likely to require fewer resources. However, our advice and guidance on environmental protection remains equally applicable.

Bird carcases, litter and faeces contain a number of biological and chemical hazards that could pose risks to the environment, animal health and public health. These are explained below.

2.2 Biological hazards

2.2.1 Avian influenza viruses

There are three types of influenza virus - *Influenza virus* A, B and C - of which B and C occur only in humans and rarely some other mammals. Avian flu viruses belong to type A and, as well as infecting birds, can infect humans and other mammals.

Type A influenza viruses are further divided into subtypes based on the antigenic relationships in the surface glycoproteins, haemagglutinin (HA) and neuraminidase (NA). At present, 16 HA subtypes have been recognised (H1-H16) and nine NA subtypes (N1-N9). Each virus has one HA and one NA antigen, apparently in any combination. All influenza A subtypes in the majority of possible combinations have been isolated from avian species. To date, only H5 and H7 subtypes have been shown to cause HPAI in susceptible species, but not all H5 and H7 viruses are virulent. The strain of current concern is of subtype H5N1, which is highly pathogenic (and therefore known as Highly Pathogenic Avian Influenza or HPAI) meaning that it may cause severe disease.

[1] Large poultry farms may contain approximately 500,000 birds, although the average is around 90,000 birds

The focus of this report is the Asian H5N1 strain of HPAI, which is a relatively unfamiliar hazard to the Environment Agency. It is important to note that this strain is primarily an avian pathogen, and is not the same virus that normally causes influenza in humans. A human flu pandemic could arise if the H5N1 virus mutates into a form that is easily transmissible between humans. This might occur if a host is infected simultaneously with H5N1 and a human flu virus, and genetic reassortment takes place to produce a new strain that is easily transmissible between humans.

The particular strain of H5N1 currently circulating first appeared in geese in 1996 in Guangdong province, China. Its pathogenicity to ducks and geese varies a great deal. In poultry (hens and turkeys), it is usually highly pathogenic and flock mortality can approach 100 per cent.

Sections 3, 4 and 5 focus on the characteristics of the Asian HPAI H5N1 virus that are important to assessing environmental risk.

Further information about avian influenza can be obtained from the following weblinks:

- Defra

  http://www.defra.gov.uk/animalh/diseases/notifiable/disease/ai/index.htm

- Veterinary Laboratories Agency

  http://www.defra.gov.uk/corporate/vla/science/science-viral-ai.htm

- World Organisation for Animal Health (Office Internationale Epizooties)

  http://www.oie.int/eng/AVIAN_INFLUENZA/home.htm

- United Nations Food and Agriculture Organisation

  http://www.fao.org/ag/againfo/subjects/en/health/diseases-cards/special_avian.html

2.2.2 Other biological hazards

A number of biological hazards are associated with birds. Organisms responsible for diseases in humans (zoonoses) that are also associated with poultry (or their litter and faeces) include:

Campylobacter coli
Campylobacter jejuni
Chlamydophila psittaci
Clostridium botulinum
Clostridium perfringens
Clostridium tetani
Cryptosporidium spp
Erysipelothrix spp
Escherichia coli including 0157
Giardia intestinalis
Listeria monocytogenes
Leptospira spp
Mycobacterium spp
Salmonella spp
Yersinia enterocolitica

Appendix A summarises the influence of environmental conditions such as extremes of pH, temperature and oxygen on each of these pathogens.

Further information about animal disease surveillance and control is available from the Defra website (http://www.defra.gov.uk/animalh/diseases/default.htm).

The SVS/Defra-led review of disposal options considered which biological hazards could be of serious concern in the event of an outbreak. The review filtered out those that do not cause serious harm, those that would be destroyed by treatment and those that would not be present at sufficient quantities to cause harm. Avian influenza

viruses, *Campylobacter* and *Salmonella* are the only biological hazards considered further in the disposal options review.

All risk assessments (such as those for proposals for on-farm carcase disposal or for health and safety assessments) should consider the prevalence and nature of all the hazards listed above and should not focus solely on avian influenza viruses.

2.2.3 Mammalian species

Brief consideration has been given to biological hazards associated with pigs and cows, as Defra's generic contingency plan (Defra, 2005) encompasses diseases of these animals. There is some evidence that pigs can become infected with avian flu viruses and it is possible that they might become infected without being adversely affected (Webster *et al.,* 2005b). However, there is currently no evidence of cattle being infected or affected by avian influenza viruses. Preliminary data regarding hazards associated with pigs and cows is given in Appendix D.

2.3 Chemical hazards

Domestic poultry, particularly those reared in high density operations, may present a variety of chemical risks to the environment at disposal, including:

- substances used in the culling process;
- ammonia and other products of decomposition;
- substances used in cleansing and disinfection operations;
- substances such as veterinary medicines that may be present in the bird at the time of death;
- substances arising from disposal processes such as incineration.

The SVS/Defra-led review of disposal options applied the same criteria as those used for biological hazards and concluded that the following chemicals could be of potential concern:

- cleansing and disinfection chemicals;
- veterinary medicines;
- ammonia;
- methane;
- hydrogen chloride;
- hydrogen sulphide;
- carbon monoxide;
- benzene, toluene, ethylbenzene and xylenes;
- wood resins;
- dioxins, polychlorinated biphenyls (PCBs) and furans;
- polynuclear aromatic hydrocarbons (PAHs);
- nitrogen oxides;
- sulphur oxides.

Our focus herein is on cleansing and disinfection and veterinary medicines. Data on the other chemical hazards is available from a number of sources including:

- http://cfpub.epa.gov/ecotox/ [aquatic and terrestrial toxicity data for individual chemicals]
- http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB [range of information on individual chemicals including use, physico-chemical properties, fate and toxicity]

Care should be taken when retrieving information from these sources as the data are subject to certain limitations in common with all databases. Professional advice should be sought if in doubt. Environment Agency staff should consult the Chemicals Science Team in the first instance.

Data on cleansing and disinfection chemicals has been collated in order to advise Defra and SVS on environmental protection (Section 2.3.1). A list of veterinary medicines considered likely to be present in the carcases of farmed birds is included in Section 2.3.2. Supporting information can be found in Appendices B and C.

2.3.1 Cleansing and disinfection

Information on disinfectants approved by Defra for use in England under the Diseases of Animals (Approved DISINFECTANTS) (Amendment) (England) Order 2005 can be accessed at http://www.opsi.gov.uk/si/si2005/20051908.htm.

SVS plans to use Virkon S and FAM 30 during an outbreak. Appendix B contains detailed data on these products and also includes data on a further disinfectant, citric acid, which appears to be of low to moderate acute toxicity to aquatic organisms and is likely to biodegrade rapidly in aquatic systems.

Virkon S is a highly acidic solution. Potential impacts are therefore likely to be related to the effect of the product on pH. The limited data available suggests that Virkon S is of moderate toxicity to aquatic organisms. The Material Safety Data Sheet (MSDS) classifies the product as biodegradable.

FAM 30 is a highly acidic solution. As with Virkon S, potential impacts are likely to relate to the effect of the product on pH. No data was found on the ecotoxicity of the product and its potential fate in aquatic environments.

Groundwater Regulations use the following definition to identify biocides:

“Active substances and preparations containing one or more active substances, put in the form in which they are supplied to the user, intended to destroy, deter, render harmless, prevent the action of, or otherwise exert a controlling influence on any harmful organism by chemical or biological means.”

Biocides are determined as List II under these regulations. However, if any of the ingredients of the products are List I substances, then discharge or disposal of the whole product would be treated as discharge or disposal of a List I product.

Virkon S and FAM 30 are confirmed[2] List II biocides under the Groundwater Regulations.

2.3.2 Veterinary medicines

There are approximately 60 veterinary products, containing over 40 different active ingredients, currently approved for use in the UK on poultry and other birds such as

[2] Joint Agency Groundwater Directive Advisory Group (JAGDAG) has determined the appropriate status for a large number of substances including Virkon S and FAM 30.

pigeons and game birds. These may be applied topically, orally or by injection. As a result of their use, traces of the products' components may be present on the birds, within their tissues or in manure and slurry from excretion after treatment. Disposal of carcases and manure/slurry in the event of an outbreak of avian flu could therefore result in the release of such substances into the environment.

Due to the large number of active ingredients, we have focused on those that are most commonly used. Based on communication with the Veterinary Medicines Directorate (VMD), the following 15 active ingredients are considered here:

Lasalocid sodium
Semduramicin sodium
Diclazuril
Robenidine
Maduramicin ammonium
Decoquinate
Monensin sodium
Narasin
Nicarbazin
Salinomycin
Halofuginone
Sulphadiazine
Trimethoprim
Neomycin
Chlortetracycline hydrochloride

Information has been collated on the fate and toxicity of these active ingredients in both the terrestrial and aquatic environment. Data has been obtained, where available, from sources including reviews by the European Food and Safety Authority (EFSA) and the European Agency for the Evaluation of Medicinal Products (EMEA).

A summary of the available aquatic and terrestrial toxicity data is provided below in Table 2.1. The data indicates that the active ingredients range from high to low acute toxicity to the species studied. Further details are provided in Appendix C.

Table 2.1: Summary of aquatic and terrestrial toxicity for 15 active ingredients

Substance	**Water** [1]	**Soil**
Lasalocid sodium	1 - 8 mg/l	71.8 - >100 mg/kg
Semduramicin sodium	19 - 39 mg/l	0.77 – 1,000 mg/kg
Diclazuril	No data	No data
Robenidine	0.036 - 0.56 mg/l	No effects at 0.36 - > 1,000 mg/kg
Maduramicin ammonium	No data	0.25 mg/kg (NOEC)[2]
Decoquinate	No data	No effects at 1.28 - > 1,000 mg/kg
Monensin sodium	0.98 -16.6 mg/l	4 - > 1,000 mg/kg
Narasin	0.77 – 20.56 mg/l	17.9 – 46.4 mg/kg
Nicarbazin	No data	No data
Salinomycin	1.14 - 32.2 mg/l	1.3 - >100 mg/kg
Halofuginone	No data	200 mg/kg
Sulphadiazine	0.135 – 403 mg/l	100 – 1,200 mg/kg
Trimethoprim	16 –130 mg/l	No data
Neomycin	> 800 and 2,829 mg/l	No data
Chlortetracycline hydrochloride	0.05 - > 128 mg/kg	No data

Notes

1. Data show ranges of concentrations at which adverse effects have been observed on test organisms in laboratory studies. Data relate to short-term (acute) studies and generally indicate the concentrations at which effects (mainly mortality) have been observed on 50% of the test populations.
2. NOEC: No Observable Effect Concentration. Data included in the absence of any effect data for this substance.

Data on the fate of the various active substances in soil, water and manure has been collated, along with information on metabolism within the treated organism and the potential for bioaccumulation. Available data is summarised in Appendix C and indicates that the majority of half-lives are in the order of days to months. Furthermore, although some actives have the potential to bioaccumulate, they are generally excreted unchanged or as metabolites. They therefore have the potential to be present in carcases, manure and slurry with the levels being dependent on elapsed time.

None of these active ingredients have so far been considered for classification under the Groundwater Regulations.

3 Sources

Key facts

The Asian HPAI H5N1 virus can exist in very high numbers within the flesh and faeces of infected animals. It is released from live birds by respiratory secretions or faecal deposits and will be released from dead birds as they decompose.

The Asian HPAI H5N1 virus can persist and remain infectious for long periods of time. Low temperature, neutral pH, high levels of moisture and high levels of organic matter promote survival in the environment.

There does not appear to be any specific data on the strain's persistence in soil or air. It is expected that the virus could persist for some time in soils containing moisture and organic matter. Survival in air is expected to be brief.

There is evidence that avian influenza viruses can survive for many days in the water environment. We tentatively estimate a half-life of 75 days in UK groundwaters.

3.1 Introduction

Sources of biological and chemical hazards in an outbreak of Asian HPAI H5N1 may be both diffuse and localised. Infected wild birds, should they arrive, will shed virus as they move around and where they fall when they die. Deaths of wild birds in nearby countries such as France and Germany have generally involved single birds or small numbers only. Farmed poultry, in contrast, are generally housed in buildings containing many thousands of birds. Infection or culling of such birds in line with Defra's contingency plan (Defra, 2005) will consequently involve large numbers of carcases located in specific areas.

3.2 Virus numbers

3.2.1 Terminology

One way of expressing virus numbers is to describe the number of plaque forming units (pfu). Here, a dilution series of a solution of virus is spread over cultured cells and the number of plaques (where host cells have lysed) is noted. The number of infectious particles in the original solution can then be calculated. Differences in plaque sizes (plaque heterogeneity) have been used to determine if a mixture of viruses is present in a particular host (Hulse-Post *et al*., 2005).

In poultry faeces Utterback (1984) found up to 10^7 (10 million) infectious particles (or pfu) per gram of faeces (not H5N1).

Infectivity is also commonly expressed in terms of the virus numbers required to infect 50 per cent of exposed eggs (median Egg Infectious Dose, EID_{50}). Webster *et al*. (1978) reported up to $10^{7.8}$ EID_{50} per gram of faeces (approximately 500 million egg infectious doses per gram of duck faeces) for a LPAI virus. Another study (Anon, 2004) showed that ducks could excrete virus for more than 17 days after infection.

3.2.2 Virus numbers in faeces

The main transmission route for Asian HPAI H5N1 in poultry is likely to be through contact with infected faeces. The numbers present in such faeces will vary. Webster *et al.* (1978) noted for a LPAI virus around $10^{7.8}$ EID_{50} per gram of duck faeces. For chickens, 10^{7} EID_{50} per gram is usually taken as a representative working figure (D. Alexander, pers. comm. 2006). Broiler hens produce around 60 g faeces per day and laying hens produce around 110 g faeces per day (A Holdsworth, pers. comm.).

Ducks produce wetter faeces than chickens and Webster *et al*. (1978) noted around 6.4 g of faeces produced per hour (154 g per day) by muscovy ducks (*Cairina moschata*). This would equate to up to 10^{10} EID_{50} per day (assuming $10^{8.7}$ EID_{50} per gram of faeces – see above).

3.2.3 Virus numbers in birds

The main environmental issue is likely to be groundwater protection. It is assumed that the main risk to groundwater would be through burial of infected stock and in particular any on-farm disposal of dead poultry and waste, should such practices be employed. Farm sites will not have been engineered in the same way as licensed commercial landfill sites.

Reports cited above suggest approximately 10^{7} EID_{50} per gram of poultry faeces. Swayne and Beck (2005) found H5N1 at $10^{7.3}$ EID_{50} per gram in breast and thigh meat. Mase *et al*. (2005) found viral titres up to $10^{7.5}$ per gram of tissue up to four days post-inoculation. In ducks, Tumpey *et al*. (2002, 2003) measured up to $10^{6.8}$ EID_{50} per gram of tissue. Perkins and Swayne (2005) found up to $10^{6.7}$ EID_{50} per gram of tissue. The virus affects tissues throughout the body in infected birds (Suarez *et al.,* 2004; Tumpey *et al.,* 2002, 2003) including skeletal muscle, so we can assume that the virus is present in all soft tissues.

If we assume that there are 10^{8} EID_{50} per gram of tissue and that the mass of an individual bird is 2.2 kg (and ignoring the fact that there should be no viable virus present in the cartilage and skeleton)[3], then the number of virus per carcase can be calculated as ($2{,}200 \times 10^{8}$) = 2.2×10^{11} EID_{50} per bird. This equates to approximately 10^{14} EID_{50}/pfu per tonne of disposed infected stock.

H5N1 is distributed throughout the soft tissues and any waste products. Under the anaerobic conditions that will prevail in a burial, we suggest that the readily degradable soft tissue mass will degrade over a period of 10 years at a linear rate (Hart and Casper, 2004). This is different to the assumptions used in guidance for the foot-and-mouth burials (Marsland *et al.*, 2003), and reflects the constant rates governing supply and removal of substances such as recharge water and terminal electron acceptors.

This suggests that up to 10^{13} EID_{50} per tonne per year could be released into the environment. This estimate is subject to considerable uncertainty and, in practice, rates of release maybe lower as much of this virus may itself be inactivated or degraded in the process.

[3] Some typical values for poultry carcase weights are: chicken (broiler or breeder) – 2.2 kg; turkey (male) – 13.5 kg; turkey (female) – 6.5 kg (A. Holdsworth, pers comm)

3.2.4 Release mechanisms and speed

Infected poultry release virus from respiratory secretions and in their faeces. The Asian HPAI H5N1 virus in poultry has an average infection to death period of around three days. Ducks may either succumb quickly (as for poultry), or survive whilst infected (Webster *et al*., 2005a), shedding viruses mainly in respiratory secretions instead of in their faeces. Webster *et al*. (2005a) show that of 23 recently isolated strains of H5N1, some were of low pathogenicity to ducks whilst others remained highly pathogenic. The experiments lasted 14 days, by which time some strains of virus had killed their hosts, with other hosts fully recovering from infection with other strains of H5N1. These findings suggest that shedding from ducks is H5N1 strain-dependent, with lower viral titres in the faeces than in respiratory secretions. Virus isolates from the strains that remained highly pathogenic still replicated to high titres in the trachea of surviving ducks at five days post-infection.

Webster also combined datasets from other experiments to show that in deliberately-infected ducks, the median value for tracheal shedding fell from Day 3 to Day 5 (days after infection) from $10^{2.5}$ (range 10^{0} to $10^{6.5}$) to $10^{0.5}$ (range 10^{0} to $10^{5.5}$). The median value for cloacal shedding (from the digestive tract) also fell from Day 3 to Day 5, from $10^{0.5}$ (range 10^{0} to $10^{5.5}$) down to 10^{0} (range 10^{0} to $10^{3.75}$). All figures are in EID_{50} ml^{-1}.

3.3 Infectivity

Definitive infectious dose information is not currently available, although research is being undertaken here in the UK at the VLA and also in the USA (http://www.ars.usda.gov/research/projects/projects.htm?ACCN_NO=409883). Studies using Asian HPAI H5N1 have used 10^{6} EID_{50} ml^{-1} (one million median egg infectious doses (Govorkova *et al*., 2005; Webster *et al*., 2005a)). In these studies, this quantity of virus initiated infection in most, if not all, subjects regardless of species. D. Alexander (pers. comm. 2006) notes that the infectious dose size can be specific to both the viral strain and the host species. Spackman *et al*. (2006), using an H7N3 virus isolated from wild cinnamon teal (a species of duck), established that the infectious dose size for chickens was 10^{5} higher than that for turkeys.

In undertaking a risk assessment for the Asian HPAI H5N1 strain, Schijven *et al*. (2005) assumed a range of infectivity between 1 and 100,000 (units not provided) for individual chickens, but noted that where poultry were housed together the risk increased dramatically because once infection occurs in one bird then any infection is likely to spread rapidly. These authors calculated risks via drinking water assuming that one duck was responsible for infecting a water body and that homogeneous distribution followed infection. They acknowledged that these assumptions may not be realistic – ducks are usually present in flocks and contamination is likely to be surface borne or restricted to the edges of the pond / water body. Clearly, for each log increase above 1 in the number of poultry then there needs to be an equivalent log increase in the efficiency of the treatment system to keep the risk per chicken equal.

There are no data available to suggest the infective dose of avian flu viruses for humans. The infectious dose for human flu viruses (Influenza A2) is thought to be around 790 pfu (Barkley 2004), although 200-300 pfu's was suggested by Zambon (pers.comm. M. Zambon, 2006). Relating pfu to EID_{50} can be complex depending upon cell types and host organism. The Veterinary Laboratory Agency (VLA) is currently undertaking research to identify source term data more positively.

3.4 Persistence in the environment

In general, many different kinds of viruses can persist in and on environmental media, including liquid and solid media and in the airborne state, with half-lives of hours, days, weeks or even months. Viruses will not replicate without a host in any environment.

The extent of persistence depends on the type of virus, its physical state (dispersed, aggregated, cell-associated, membrane-bound or adsorbed to other solids), the medium in which it is present (faeces, respiratory secretions, tissues, other liquids or solids, air) and the prevailing environmental conditions. These generally include temperature, pH and other physical and chemical properties of the medium in which the virus is present, such as moisture content, organic matter, particulates, salt concentration, protective ions, and antiviral chemicals such as proteolytic enzymes, antiviral microbial activity, and light.

On environmental surfaces and in aerosols, additional environmental factors also influence virus survival, such as relative humidity and physico-chemical forces at air-water and air-water-solid interfaces (Sobsey and Meschke, 2003).

Whilst respiratory secretions and excretions are considered infectious materials, these are not exclusive, and the virus can survive in the environment for extended periods dependent upon environmental conditions. The virus will remain infective longer on surfaces of hard or non-porous materials. Bean *et al.* (1982) found that human influenza virus persisted on plastic or steel surfaces (up to 48 hours at 28ºC, relative humidity 35 – 40 per cent), as opposed to cloth tissues (up to 15 minutes) under the same conditions.

For the Asian HPAI H5N1 virus to become non-infective, some change must occur that prevents the virus particle binding to potential host cells. A number of chemical disinfectants as well as humidity and temperature allied to defined time periods inactivate avian flu viruses. Where no data is available for the Asian HPAI H5N1 strain, information has been used from other influenza viruses (as indicated).

3.4.1 Humidity

Human influenza virus survives best in aerosols at low relative humidity. Aerosolised avian flu is more resistant than human flu, although survival is likely to be measured in hours rather than days (Sobsey and Meschke, 2003).

3.4.2 Soil

No data has been identified on survival and persistence in soil. In general, the lower the organic matter content in soil, the less adsorption and survival of the virus (Sobsey and Meschke, 2003). Soil moisture content and temperature will also affect survival - low temperatures and high moisture content will enhance survival (Sobsey and Meschke, 2003).

3.4.3 Air

In air, virus survival will be brief. Virus inactivation is associated with elevated temperature, high UV and low humidity. Airborne transmission of influenza virus is via inhalation of infectious droplets, direct contact, fomite contact, or self-inoculation onto the upper respiratory tract (by inhalation or touch). No case of human-to-human

transmission by small particle aerosol of Asian HPAI H5N1 has yet been identified (WHO, 2005) and no specific data has yet been identified.

3.4.4 Groundwater

The survival of Asian HPAI H5N1 virus in UK groundwater is untested or unreported at present. Clearly there are a number of processes that are likely to restrict virus survival and transport in groundwater, including:

- chemical inactivation;
- sorption onto soil or aquifer matrix;
- predation by indigenous micro-organisms.

However, existing reports on general virus behaviour suggest that survival will be enhanced by factors associated with the groundwater environment such as limited predation, low solute concentrations, lower temperature and absence of sunlight. As a result, it should be anticipated that any virus released into relatively clean, uncontaminated groundwater may survive for extended periods.

Stallknecht *et al*. (1990a, b) report data on survival of viruses (H3N8, H4N6, H6N2, H12N5 and H10N7) in distilled water and provide calculated half-lives for these avian influenza viruses of:

- five days at 28 ^{o}C;
- ten days at 17 ^{o}C;
- seventy-five days at 4 ^{o}C.

This suggests that for UK groundwater (at 11^{o}C) a half-life ($t_{1/2}$) of between 10 and 75 days may be realistic, but in the absence of specific data for either H5N1 viruses or for UK groundwater it should be assumed that the $t_{1/2}$ might be at least 75 days.

It is important to note that this estimate represents virus freely suspended in water. In practice, the virus is likely to be present in complex solutions or protected within solid matrices (such as in dead tissues or sorbed to mineral surfaces). We have no data on $t_{1/2}$ for these environments, so the estimate above should only be used for virus freely suspended in the groundwater.

3.4.5 Surface water

Survival of avian flu viruses in surface water is strongly temperature-dependent and lower temperatures will promote survival. However, the presence of organic matter may also help to protect the virus envelope and increase survival. For example, it has been documented that H5N2 viruses may survive in liquid chicken manure for more than 105 days at winter temperatures; H7N2 virus survived 30 to 35 days in faeces at 4ºC and seven days at 20ºC (Lu *et al*., 2003). Stallknecht *et al*. (1990a) calculated from an initial input of a million infective virus doses (H4N6) per ml of distilled water that the water could remain infective to birds for up to 207 days at 17ºC. In freshwater, similar strains of avian influenza virus took up to 35 days at 17ºC and 17 days at 28ºC to achieve a one-log reduction in viral titre. Stallknecht *et al*. (1990a, b) undertook experiments with a number of avian influenza viruses and demonstrated that the longest time for a log order reduction was 34.5 days. They demonstrated a range from five to 34.5 days depending on virus strain (H3N8, H4N6, H6N2, H12N5 and H10N7), temperature and water type.

Phuong (unpublished data, 2005) evaluated H5N1 survival in phosphate-buffered saline (PBS); PBS plus one per cent milk, pond water, brackish water and a fast flowing stream (arroyo). At 4ºC, survival was longest at greater than four days for all waters. At 20 - 25ºC, the virus survived in the arroyo water for over 90 hours. It survived for over four days in PBS plus one per cent milk at 4ºC, 20-25ºC and 37ºC. This shows that the presence of organic material provides a protective effect. Unfortunately, the author did not provide organic matter content of the waters. Webster *et al*. (1978) conducted a simple batch experiment using H3N6, where lake water containing an initial $1 \times 10^{8.1}$ EID_{50} per ml contained $1 \times 10^{4.3}$ EID_{50} per ml after 32 days at 0ºC. This suggests a log reduction in 8.4 days.

3.4.6 Landfill environments and leachate

No information has been located on Asian HPAI H5N1 virus mobility and persistence in landfill environments and leachate. Low temperatures, moisture content and neutral pH are likely to account for some aspects of persistence. Landfills will contain sorbtive solids and the leachate from waste will also contain soluble and colloid organic matter that may well protect the virus against elimination.

Within a landfill, the temperatures will generally be over 30ºC, liquid flow rates will be low and the salinity content of leachate may be high (with conductivity of over 20,000 micro Siemens). These conditions may protect groundwater by inactivating the virus. Also, the presence of solvents and detergents, albeit at relatively low concentrations of a few micrograms per litre, would help to inactivate the virus. However, the conditions contained within landfills will be very heterogeneous and these averaged values for protective factors cannot be relied upon. The possibility of liquid shortcuts through any landfill can not be discounted. Fichtner (1984) noted that repeated attempts to isolate avian influenza (H5N2) virus from a “sanitary” landfill effluent were all negative.

3.5 Factors that influence persistence

3.5.1 Soil type and organic matter

Organic matter reduces the rate of viral inactivation by a viricide regardless of the medium. Soil or faecal matter suspended in the aquatic medium helps protect viruses from chemical viricidal activity.

However, without viricide present, LPAI H7N2 mixed with chicken manure lost infectivity more quickly than without manure (Lu *et al*., 2003) - the authors speculated that other micro-organisms or digestive enzymes were likely to be responsible.

In faeces, avian flu viruses can survive for some time. When discussing the 1983 Pennsylvania outbreak, Utterback (1984) concluded that viruses may have been present at concentrations as high as 10^7 infectious particles per gram and may have survived for longer than 44 days. Beard *et al*. (1984) looked at the same H5N2 virus and incubated wet faeces from naturally-infected hens at 4ºC, where infectivity persisted for up to 35 days (the longest time the faeces were tested); however, at 25ºC infectivity could only be detected for two days.

Lu *et al*. (2003) reported that within two days of deliberate infection (10^7 EID_{50} per gram) with an LPAI H7N2 avian influenza virus, infected hens shed virus in their faeces. The most intense shedding of virus occurred during the first week post-infection. After 16 days, virus shedding had ceased from the birds. The virus survived

in manure for over two weeks after the birds had stopped shedding virus within the cages. The younger the bird at the time of infection, the longer it took for the virus to be cleared by its immune system.

Lu *et al*. (2003) also examined LPAI H7N2 virus survival in chicken manure at various temperatures. 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 days and at 37°C, they could detect virus at 14 but not 16 days. At 15 to 20ºC, infectivity was lost after 23 days when added to manure. At 37ºC, infectivity was lost after 16 days. The same authors showed that in 'field chicken' manure held at ambient temperature (15-20°C), they could detect virus at four days but not six, and at 37°C they could detect virus at 12 hours but not 36 hours (Lu *et al*., 2003).

3.5.2 Hydrogeology

No definitive information has been identified about the effect of different hydrogeological conditions on virus survival and transmission.

3.5.3 Time

Viruses will not replicate without a host in any environment. Time will lead to a reduction in viral numbers, with the rate depending on environmental factors such as pH, salinity, temperature and levels of organic matter.

3.5.4 Temperature

Temperature greatly affects viral survival. Regardless of the environmental medium, the higher the temperature the greater the inactivation rate. In water, survival of H4N6 reduced from 207 days at 17ºC down to 80 days at 28ºC, although H10N7 survived 102 days at 28°C (Stallknecht *et al*., 1990a). Their experiments also demonstrated that the longest time for a log order reduction was 34.5 days (range from five to 34.5 days) as noted previously.

In composted birds, Senne *et al*. (1994) determined complete HPAI virus inactivation after 10 days at 56ºC. The length of time spent at a temperature is also important. In bird carcases, the core temperature needs to reach 70ºC for 30 minutes, 75ºC for five minutes or 80ºC for one minute (DPIE, 1996).

Lu *et al.* (2002) undertook heat inactivation experiments with LPAI H7N2 avian influenza virus. At 60ºC, all virus present was inactivated within 10 minutes.

3.5.5 pH

Extremes of pH inactivate viruses. Organic acids and aldehydes appear to be less effective at inactivating virus at temperatures below 20ºC (Yilmaz *et al*., 2004).

Lu *et al.* (2002) undertook pH inactivation experiments with LPAI H7N2 avian influenza virus, where pH 2 inactivated all virus present within five minutes (starting titre $10^{5.5}$ EID_{50}), while pHs 5, 7, 10 and 12 failed to inactivate virus after 15 minutes.

3.5.6 Salinity

Saline conditions appear to reduce viral persistence (Phuong, 2005). This agrees with Stallknecht *et al*. (1990b) who looked at H6N2 survival, and determined virus survival was lowest in saline conditions (20 parts sodium chloride per thousand) irrespective of temperature.

3.5.7 Treatment and disposal activity

Senne *et al.* (1994) composted poultry carcases between 38ºC and 60ºC and after 10 days, the HPAI virus could not be recovered (not known if H5N1) despite repeated testing.

An H7N2 LPAI outbreak in Virginia, USA (2002) was controlled used stamping out (culling). The first flock to be culled was buried in a lined, on-farm burial pit, with long-term monitoring wells. After several other flocks were culled in the outbreak, landfills were used for carcase disposal. Carbon dioxide gas was used for euthanasia. The litter was composted in the poultry-housing unit. The pattern of disease spread in this outbreak was consistent with transmission via fomite, people and equipment, rather than by airborne transmission. Transport of daily mortality off-farm to a rendering facility was the management practice with the highest association with infection.

4 Pathways

Key facts

Live infected birds are capable of transporting the Asian HPAI H5N1 virus. Additionally, there is evidence for transport on the clothing of poultry workers and on vehicles for other avian influenza viruses.

Once the virus has left host birds (or their secretions and excretions), the most significant environmental pathway is considered to be via surface and groundwater, although there is no specific data for UK water bodies. Additional work is underway to simulate the likely fate and transport of the Asian HPAI H5N1 virus in UK groundwaters.

Using very limited data and making several assumptions, we suggest that bird infection could occur at virus concentrations between 10 – 1,000 viruses per ml water or above.

4.1 Where might shed virus go?

Wild birds such as ducks mainly shed faeces into water (see Section 3.4 for persistence information). Their respiratory secretions will also mainly fall into water. This is not the case for poultry, where all shedding will occur on land. It is less likely that the virus will be shed as an aerosol from ducks, and is unlikely to remain airborne when shed either by ducks or poultry.

4.2 Fate and transport of the virus in waters

Viruses may enter water through a variety of routes, including:

- excretions from live birds directly into surface water;
- sneezes, coughs and so on from live birds directly into surface water;
- decomposition of dead wild birds in, or adjacent to, surface water;
- loss from farmed bird manure into surface or groundwater;
- leaching from buried remains into groundwater (this may also affect surface water through springs or abstractions).

4.2.1 Sorption of virus in the unsaturated zone

A variety of one-, two- and three-dimensional transport models have been developed to simulate virus transport through unsaturated media. In essence, these models are similar to the traditional pollutant transport models used in the Environment Agency such as P20 and CONSIM (Environment Agency, 1999, 2003). All the models are simplifications, however, and require the adoption of a variety of assumptions to work. The main difficulty in using these models in planning for any future outbreak of avian influenza is the lack of specific data for viruses and avian flu viruses in particular.

The key piece of data is the partition coefficient (Kd) that describes the distribution of virus between attached and suspended phases. No reports were found of Kd for influenza virus attachment to any soil or aquifer materials.

A virus attenuation model developed by the US Environmental Protection Agency for estimating bank filtration capacity provides a table of Kd values for combinations of five viruses and three US soil types (Table 4.1).

Table 4.1: Kd values for combinations of five viruses and three US soil types

	Partition coefficient (m^3 g^{-1})		
Virus	**Clay soil**	**Silt**	**Sand**
Polio	7.2×10^{-4}	3.8×10^{-4}	2.4×10^{-4}
Hepatitis A	1.9×10^{-3}	NA	4.7×10^{-6}
Reovirus 3	1.2×10^{-3}	2.1×10^{-3}	3.0×10^{-3}
Coxsackie	8.7×10^{-5}	NA	6.2×10^{-4}
Echovirus	4.5×10^{-4}	4.4×10^{-4}	7.4×10^{-4}

None of the viruses provides a good surrogate for influenza based on biological and physico-chemical properties. For example, they all exhibit naked capsids (no host-derived membrane or envelope surrounding the protein coat), whereas influenza A viruses possess an envelope.

Sorption and filtration may also be affected by particle size. Unfortunately, influenza A is seen in a variety of forms including filamentous. However, most estimates place the virion (virus particle) size between 80 and 120 nm.

Sorption is likely influenced strongly by the surface charge of the virus. However, once again we have found little in the literature about this. A single report (Zhuang, 2004) suggests an isoelectric point of pH 6.5; no estimates of zeta potential have been found.

4.2.2 Acceptable concentration of virus at receptor

We need to consider a limit for the acceptable concentration of virus at a groundwater receptor (spring, borehole and surface water). This will allow us to use existing tools to estimate the acceptable pollutant loading for groundwater and to set limits at compliance points. For an infectious agent this relates to the infectious dose which, in turn, depends on a wide range of factors including the sensitivity of the host and the route by which the host is exposed to the pathogen.

We can find no reports for the Asian HPAI H5N1 virus, but Swayne and Beck (2005) estimated that one chick mean infectious dose (CID_{50}) by the intranasal route for the HPAI virus H5N2 was 10^3 EID_{50}. We assume that the likely route for infection by groundwater is ingestion of contaminated water, and that a bird or small animal may consume up to 100 ml of water. We conclude that bird infection could occur at virus concentrations between 10 and 1,000 viruses per ml or above. It is important to note that these numbers are very uncertain, based as they are on very limited data and a number of assumptions.

Work is underway to prepare bespoke editions of regulatory models such as P20 for use in the assessment of disposal sites, should this be needed.

5 Receptors

Key facts

Although primarily an avian pathogen, the Asian HPAI H5N1 virus has repeatedly shown that it can infect other hosts – mainly mammals – and kill them. More than 140 people have died from H5N1 infection to date.

Precautions should be taken when working in close proximity to infected birds or those suspected of being infected.

British wildlife, including badgers, ferrets and foxes, are potentially at risk if they come into contact with the virus (for example, by consuming infected carcasses).

5.1 Host range for H5N1

This section includes information on host range, briefly describing where the virus replicates within the host, how quickly the host is killed, symptoms, and what material might be infective. Any animal carcase suspected to have died from HPAI must be treated as potentially infective material. More detailed information can be found in Appendix E.

The host range includes:

- **Poultry** (including chickens and turkeys). Poultry excrete virus in the short time between infection, incubation and death (two to three days), usually via respiratory secretions and faeces.
- **Ducks and geese.** The current Asian HPAI H5N1 strain arose in geese in China. It is quite possible that whilst the strain is highly pathogenic to poultry, the same strain demonstrates low (or non) pathogenicity to ducks and geese (Hulse-Post *et al*., 2005). Thus, ducks and possibly geese may carry and excrete virus without showing signs of disease.
- **Cats** (all felids). The virus replicates in the respiratory tract: however, the virus is excreted at low titre in faeces (Kuiken *et al.,* 2004). This was a small experiment using high doses of virus, with a mortality rate of 33 per cent (death within six days of infection).
- **Mice and rats**. The virus replicates in the respiratory tract, and shedding of virus in faeces can not be ruled out. Therefore, rodents should not be permitted to come into contact with infected material, where rodents may shed virus into water that birds use, and these birds could then become infected (Dybing *et al*., 2000).
- **Ferret.** Nasally-inoculated H5N1 produces systemic disease in ferrets. Until other information becomes available, this should be applied to all Mustelidae which include badgers, stoats, otters, mink, polecats and pine martens.
- **Pigs.** The virus reproduces in the respiratory tract, not in the alimentary canal – no data currently exists to suggest that H5N1 virus is shed in pig faeces (Webster *et al*., 2005b). It is not currently thought to transmit readily between pigs.

- **Humans.** The virus replicates in the respiratory tract (and gastrointestinal tract). Incubation period is two to 17 days. Occasionally systemic. The H5N1 virus does not readily transmit between humans.
- **Dogs.** Over 25 per cent of 629 dogs examined in Thailand have recently been noted as carrying antibodies to the H5N1 virus (Butler, 2006a). No evidence yet noted that dogs could spread the H5N1 virus. The VLA advises that this information should be treated with caution until more is known.
- **Cattle and horses.** These animals are not currently thought to be hosts for HPAI H5N1.

5.2 Worker health and safety

General information is available from Defra's website (http://www.defra.gov.uk/animalh/diseases/notifiable/disease/ai/index.htm).

Environment Agency staff should read our generic risk assessment (http://intranet.ea.gov/ams_document_library/04/4_07_health_and_safety/hs_risk_assessments/469_05.doc) and our health advice for staff (http://intranet.ea.gov/people_matters/national_health_and_safety/guidance/Avian_Influenza_Health_advice_for_staff.pdf).

5.3 The wider environment

Even if there is no potable use of groundwater, care should be taken to prevent entry into farm ditches and streams potentially used for stock watering or public access. Virus-contaminated materials must not be allowed to penetrate groundwater or enter standing water (ponds, ditches, troughs). As we know that the Asian HPAI H5N1 virus can survive for several months (depending on environmental conditions – see Section 3), it is possible that it could survive to cause another outbreak (and could become endemic).

6 Glossary

ABPR	Animal By Products Regulations
Aerosol	Microscopic airborne particles
Anseriformes	Bird order including ducks, geese and swans
Antigen	Substance that stimulates the production of antibodies when introduced into the body
Charadriformes	Bird order including gulls and shorebirds such as lapwings and plovers
CID_{50}	Chick Infectious Dose 50, the amount of virus required to infect 50 % of inoculated chicks
Composting	Oxygen-requiring biodegradation process
Conjunctiva	Eye-associated membrane
Defra	Department for Environment, Food and Rural Affairs
DETR	Department for the Environment, Transport and the Regions
EFSA	European Food and Safety Authority
EID_{50}	Egg Infectious Dose = dose required to infect 50% of inoculated eggs
EMEA	European Agency for the Evaluation of Medicinal Products
Endemic	A disease constantly present at a low incidence within a particular geographic region
Envelope	Lipid (fatty) coating of avian influenza virus
Fomite	An article (such as clothing or books) in sufficient close contact with infectious agent to retain infectivity upon its surface
Galliformes	Bird order, including poultry (hens and turkeys)
Groundwater	All water below the surface of the ground in the saturation zone and in direct contact with the ground or subsoil
Haemagglutinin	Viral protein that agglutinates blood cells
Hazard	A property or situation that in particular circumstances could cause harm
HPAI	Highly Pathogenic Avian Influenza
Incineration	Destruction by heating
Log reduction	Reduction by one order of magnitude (by 90%)
LPAI	Low Pathogenicity Avian Influenza
Lysed	A host cell is lysed when its membrane has been disrupted by virus activity (see lysis)
Lysis	The leaking of cell contents when a virus particle punctures its host cell membrane as it exits the cell
MSDS	Material Safety Data Sheet
Mustelidae	Mammal family, including badgers, otters, weasels, ferrets, mink, polecats
Neuraminidase	A viral enzyme permitting the escape of new infectious particles from a previously infected host cell
OJ	Official Journal of the European Union
PBS	Phosphate-buffered saline
Pfu	Plaque forming unit - measurement of viral concentration. Dilution series of solution containing virus is spread over 'lawn' of cells;

	a plaque (or hole) appears where a virus particle has grown and ruptured cells.
Ratites	Flightless birds (emu, cassowary, ostrich)
Rendering	Use of animal by-products for producing tallow, grease and high-protein meat and bone meal
Source term	Property, substance, activity or event that represents a hazard
SPF	Specific pathogen free
SVS	State Veterinary Service
Systemic	Something that affects the whole body
$TCID_{50}$	Tissue Cell Infectious Dose = dose required to infect 50% of exposed tissue cultures
Titre	Strength of solution, or amount of virus present in a solution
UNFAO	United Nations Food and Agriculture Organisation
Viricide	Chemical agent that inactivates virus
Virus	Nucleic acid-based, parasitic self-replicating infectious agent
VMD	Veterinary Medicines Directorate
WHO	World Health Organisation
Zoonosis	An animal disease transmissible to humans

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Appendix A: Biological hazards

	Growth range		
Zoonotic pathogen	**pH**	**Temperature**	**Aerobic / anaerobic**
Campylobacter coli	4.9 - 9	30ºC - 45ºC	Aerobic (prefers low oxygen)
Campylobacter jejuni	4.9 - 9	30ºC - 45ºC	Aerobic (prefers low oxygen)
Chlamydophila psittaci	Not available	Not available	Aerobic
Clostridium botulinum	4.5 - 9	10ºC – 48ºC	Anaerobic
Clostridium perfringens	5.5 - 9	12ºC - 50ºC	Anaerobic
Clostridium tetani	5.5 - 9	12ºC - 50ºC	Anaerobic
Cryptosporidium spp	4	Optimal at 37ºC	Aerobic
Erysipelothrix spp	4	Optimal at 37ºC	Aerobic
Escherichia coli including 0157	4 – 9	6.5ºC – 49ºC	Grows aerobically and anaerobically
Giardia intestinalis	4 – 9	Optimal at 37ºC	Aerobic
Leptospira interrogans	Optimal at 7.2	20°C-37°C	Aerobic
Listeria monocytogenes	4.5 – 9.5	-0.5º - 45º	Grows aerobically and anaerobically
Mycobacterium spp	5.5 – 8.5	Optimal at 37ºC	Aerobic (prefers low oxygen)
Salmonella spp	4 – 9	5ºC – 46ºC	Grows aerobically and anaerobically
Yersinia enterocolitica	4.2 - 10	-1ºC – 42ºC	Grows aerobically and anaerobically

Appendix B: Cleansing and disinfection

This appendix presents a summary, along with more detailed supporting information, of the ecotoxicity and potential fate of three disinfectants (Virkon S, FAM 30 and citric acid) in freshwater systems. It is our understanding at the time of writing (September 2006) that SVS intend to use the first two only in the event of an outbreak of avian influenza in the UK. Information on each product and its main ingredients has been obtained from the Material Safety Data Sheets (MSDS). Table B1 summarises ecotoxicity data available for freshwater organisms.

Table B1: Acute ecotoxicity of disinfectants to aquatic organisms[1]

Product/Main ingredients	**Acute ecotoxicity to aquatic organisms** (concentration range for acute toxicity endpoints)
• **Virkon S**	• Highly acidic solution (pH 2.6) – potential impact on pH Moderate acute and chronic toxicity (limited data: 6 – 25 mg/l)
• Potassium peroxomonosulphate	• Moderate acute toxicity (limited data: 5.3 – 32 mg/l)
• Sulphamic acid	• Acid – potential impact on pH Moderate acute toxicity (limited data:14.2 – 70 mg/l)
• Sodium dodecylbenzene sulphonate	• Moderate to high acute toxicity (0.23 - 38.5 mg/l)
• **FAM 30**	• Highly acidic solution (pH 0) – potential impact on pH
• Sulphuric acid	• Acid – potential impact on pH Low to high acute toxicity (0.1 – 120 mg/l)
• Phosphoric acid	• Data too limited (138 mg/l/)
• Non-ionic surfactants	• Moderate to high acute toxicity (0.11 – 50 mg/l)
• Iodine	• Moderate to high acute toxicity (0.01 – 4.2 mg/l)
Citric acid	• Low to moderate acute toxicity (1 – 2,600 mg/l)

[1] Concentration ranges that have a significant effect on growth or mortality of a variety of aquatic organisms.

B1 Summary information

B1.1 Virkon S

Virkon S is a highly acidic solution. Potential impacts may therefore be related to the effect of the product on pH. The limited data available suggests that Virkon S is of moderate toxicity to aquatic organisms. The MSDS classifies the product as biodegradable.

The major components of Virkon S are potassium peroxomonosulphate, sulphamic acid and sodium dodecylbenzene sulphonate. The limited data suggest that potassium peroxomonosulphate and sulphamic acid are of moderate acute toxicity to aquatic organisms, whilst the surfactant sodium dodecylbenzene sulphonate is of moderate to high acute toxicity. The potential impact of sulphamic acid is likely to be in relation to its effect on pH. The main routes of degradation are likely to be via hydrolysis for potassium peroxomonosulphate, and biodegradation for sodium dodecylbenzene sulphonate, but data on degradation rates is limited. Sulphamic acid however may be regarded as persistent.

B1.2 FAM 30

FAM 30 is a highly acidic solution. As with Virkon S, potential impacts may therefore be in relation to the effect of the product on pH. There was no data on the ecotoxicity of the product and its potential fate in aquatic environments.

The major components of FAM 30 are sulphuric and phosphoric acids, non-ionic surfactants and iodine. Sulphuric acid is harmful to aquatic life primarily due to its acidity, with available data ranging from low to high acute toxicity. The non-ionic surfactants and iodine are of moderate to high acute toxicity. Information on the ecotoxicity of phosphoric acid was limited, with only one data point. It is a weaker acid than sulphuric acid, but could potentially impact on the pH of the receiving water if present in sufficient quantities. Once released, both sulphuric and phosphoric acids may be neutralised over time. Iodine is an elemental inorganic compound and will therefore not degrade. The potential fate of the non-ionic surfactants is difficult to assess, as the specific substance(s) contained in the product are unknown.

B1.3. Citric acid

Citric acid appears of low to moderate acute toxicity to aquatic organisms. It is likely to biodegrade rapidly in aquatic systems.

B2 Further information

B2.1 Antec Virkon S

B2.1.1 Product toxicity

Virkon S is a highly acidic solution (pH 2.6). Potential impacts may therefore be in relation to the effect of the product on pH. An EQS for pH is available for the protection of freshwater life. The 95th percentile of pH values should remain within pH range 6 to 9.

Toxicity data for the whole product suggests that Virkon S is of moderate toxicity to aquatic life, however only very limited aquatic toxicity data is available for the whole product.

A document produced by WRc for Antec reports a *Daphnia magna* 48 hour EC_{50} (immobilisation) of 6.5 mg/l (at the end of a 48 hour test, 50 per cent of the animals were immobilised at 6.5 mg/l). An acute 96-hour LC_{50} for Atlantic salmon is reported as 25 mg/l (after 96 hours, 50 per cent of the salmon was killed by 6 mg/l). Two chronic studies are reported which state that continuous exposure of rainbow trout and Atlantic

salmon to 6 mg/l for four months caused no loss of feeding or growth. A second chronic study on rainbow trout reports that at the unfed fry stage, fish were completely tolerant of Virkon S for aquaculture at concentrations of zero to 8 mg/l. Once the fish began to feed, toxic effects were seen. For the first feeding fry, an LC_{50} of 6 mg/l was determined. However, after this first feeding period (approximately day 14-21) fish exposed to concentrations up to 8 mg/l showed no differences in mortality to the controls until the study ended after 88 days. This study concluded that 6 mg/l was the LC_{50}.

B2.1.2 Product fate

According to the MSDS, the product is expected to be biodegradable. Virkon S is a peroxygen-based disinfectant. The biocidal activity of this product results from the oxidising potential of the potassium peroxymonosulphate. The oxidising potential is directly biocidal but may also act on the sodium chloride to form hypochlorous acid. This proposed mode of action involves the oxidation of the sodium chloride to form chlorine, which then reacts with the sulphamic acid to form a temporary intermediate, chlorosulphamic acid. This then hydrolyses to form hypochlorous acid, a biocide. Although there is therefore the potential for chlorinated byproducts to form from the use of this product, the formation of organochlorines is thought to be limited.

B2.1.3 Toxicity and potential fate of individual components of Virkon S

The major components of Virkon S are set out in Table B2:

Table B2: Major components of Virkon S

Product	Active ingredient (Concentration in product)	CAS number
Virkon S supplied by Antec	Potassium peroxomonosulphate 50% Sulphamic acid 5% Sodium dodecylbenzene sulphonate 15%	70693-62-8 5329-14-6 25155-30-0

B2.1.3.1 Potassium peroxomonosulphate

Toxicity

Potassium peroxomonosulphate is a strong oxidising agent. The limited ecotoxicological information indicates that this compound is of moderate acute toxicity to two aquatic species, with a 96-hour LC_{50} >32 mg/l for the zebrafish *Brachydanio rerio* and a 24-hour EC_{50} (immobilisation) of 5.3 mg/l for the waterflea *Daphnia magna*.

Potential fate

The main route of degradation is via hydrolysis forming potassium sulphate and oxygen (potassium sulphate is thought to be of moderate to low toxicity to aquatic organisms). Limited information is available on the rate of degradation. The rate appears to be in the order of hours to weeks depending on the conditions. Degradation is accelerated by metal ions and by particles of different matter present in sewage, surface waters and soil.

B2.1.3.2 Sulphamic acid

Toxicity

The only data obtained for sulphamic acid relates to acute effects on the fathead minnow, with effects observed in the range of 14.2 – 70 mg/l. This indicates moderate toxicity to this species. However, as it is an acid, the potential impact of sulphamic acid is likely to be in relation to its effect on pH.

Potential fate

Limited information is available on the degradation of sulphamic acid. Abiotic degradation occurs due to the hydrolysation of the acid to ammonium hydrogen sulphate. However, the available data indicates that this is a slow process with a half-life of greater than 12 months at 25º C in waters with pH > 3. Therefore, sulphamic acid can be regarded as persistent.

B2.1.3.3 Sodium dodecylbenzene sulphonate

Toxicity

Sodium dodecylbenzene sulphonate is an anionic surfactant, of moderate to high toxicity to aquatic life (according to available data). Reported effect concentrations range from 3 to 29 mg/l for algae, 0.23 to 38.5 mg/l for invertebrates and from 1 to 36 mg/l for fish.

Potential fate

In water, sodium dodecylbenzene sulphonate is expected to be essentially non-volatile. Bioconcentration, adsorption to sediment, and hydrolysis are not expected to be important in aquatic systems. Sodium dodecylbenzene sulphonate is expected to biodegrade in aerobic soils and aquatic conditions, based on a variety of biodegradation studies. However, no information is available on the rate of degradation in the environment. In sewage treatment works, biodegradation rates were reported in the order of days to weeks.

B2.2 FAM 30 supplied by Evans Vanodine

B2.2.1 Product toxicity

FAM 30 is a highly acidic solution (pH 0). As with Virkon S, potential impacts are therefore likely to be in relation to the effect of the product on pH. In addition, direct discharge into aquatic systems will be harmful to fish and other aquatic species due to the iodine, which is very toxic to aquatic organisms.

B2.2.2. Potential fate

No information specific to water was found. However, when discharged to soil, the sulphuric and phosphoric acids are rapidly converted to inorganic sulphates and phosphates by reaction with alkali metal salts. The resultant rise in pH will result in precipitation of residual available iodine as iodide. It should be noted that the level of available iodine will be significantly reduced by interaction with organic matter, both during the disinfection process and after discharge.

B2.2.3. Toxicity and potential fate of individual components of FAM 30

According to the MSDS, the major components of FAM 30 are sulphuric and phosphoric acids, non-ionic surfactants and iodine (Table B3).

Table B3: Major components of FAM 30

Product	Active ingredient Concentration in product)	CAS number
FAM 30 supplied by Evans Vanodine	Sulphuric acid 5-10%	7664-93-9
	Phosphoric acid 5-10%	7664-38-2
	Non-ionic surfactant 20-30%	-
	Available iodine < 3%	7553-56-2

B2.2.3.1. Sulphuric acid

Toxicity

Sulphuric acid is primarily harmful to aquatic life due to its acidity and is generally less toxic in hard or highly buffered water. Aquatic toxicity data have been reported in the range of 0.1 – 120 mg/l for freshwater organisms (Table B4).

Potential fate

Sulphuric acid is miscible with water. It will ultimately react with calcium and magnesium to form sulphate salts. Sulphuric acid does not chemically or biologically degrade naturally. However, it is neutralised with time.

Table B4: Effects of sulphuric acid on freshwater organisms

Species	Exposure duration	Effect/Endpoint	Concentration	Hardness
Invertebrates				
Water flea (*Daphnia magna)*	1-3 hrs	Lethal	50 mg/L	Soft
	24-72 hrs	Lethal	29 mg/L	Soft
	168 hrs	Lethal	0.1 mg/L	Soft
Fish				
Bluegill (*Lepomis macrochirus)*	24 hr	Lethal	24.5 mg/L	-
	60 hr	Lethal	7.36 mg/L	Distilled
	1 month	NOEC	3.68 mg/L	Distilled
Goldfish (*Carrasius auratus)*	4 days	NOEC	17 mg/L	Soft
	100 hrs	NOEC	100 mg/L	Hard
Trout species	24 hr	Lethal	6.25 mg/L	-
Minnow species	24 hr	NOEC	100 mg/L	-
	6 hr	Lethal	6 - 8 mg/L	Distilled
	6 hr	Lethal	110 – 120 mg/L	Hard

B2.2.3.2. Phosphoric acid

Toxicity

Only one toxicity datum was found at this time: effects on the mosquito fish were seen at 138 mg/l/ (24-96 hours) in turbid water at 22-24º C. No further information was available on the test performed.

Potential fate

As with sulphuric acid, acidity may be reduced by natural water hardness minerals. However, the phosphate may persist indefinitely.

B2.2.3.3 Non-ionic surfactants

Toxicity

Non-ionic surfactants are a generic group of compounds. Generally, they are of moderate acute toxicity to fish, algae and molluscs, with reported effect concentrations in the range of 1.6 - 8.5, 2.0 – 50, and 11 – 23 mg/l, respectively. Crustaceans appear to be the most sensitive aquatic organisms with reported effect concentrations in the range of 0.11 – 9.2 mg/l. This indicates that these surfactants are of moderate to high acute toxicity to these organisms.

Potential fate

According to the MSDS, the surfactant is readily biodegradable during biological effluent treatment. However, it is difficult to assess potential environmental fate, as the specific substance(s) contained in FAM 30 are unknown. More details are needed from the manufacturer.

B2.2.3.4. Available iodine

Toxicity

Available data indicate that iodine is of moderate to high acute toxicity to aquatic organisms with 24-96 hour LC_{50} ranging from 0.01 – 4.2 mg/l. Iodine is a powerful oxidising agent, which is only slightly soluble in water.

Potential fate

Iodine is an elemental inorganic compound and will therefore not degrade. The potential for the formation of organoiodine compounds is low under environmental conditions

B2.3. Citric acid

Toxicity

This is a weak organic acid and is of low to moderate acute toxicity to aquatic organisms. Reported LC_{50s} and EC_{50s} were above 120 mg/l citric acid for *Daphnia magna*. Overall, toxicity ranged between 1 and 2,600 mg/l for fish and crustacean.

Potential fate

Study of biodegradation in the presence of sewage showed 98 per cent degradation in 48 hours (rapid). A study of degradation in natural waters indicated that it was also rapid. In river water spiked with citrate at concentrations of 15 mg/l, the time of disappearance of half the citrate was reported to range from 0.2 to 3.5 hours. Within 8 to 24 hours, citrate concentrations were reported to have dropped to 0.15 mg/l, a 99 per cent reduction. The available data thus indicate that citric acid is rapidly degraded.

References

Information from the Environment Agency's ETAS enquiry service.

Appendix C: Veterinary medicines

There are approximately 60 veterinary products, containing over 40 different active ingredients, currently approved for use in the UK on poultry and other birds such as pigeons and game birds. Data on the toxicity and fate of the 15 most commonly used active ingredients in the terrestrial and aquatic environment and manure have been collated, along with information on metabolism within the treated organism and the potential for bioaccumulation. The available data is summarised in Tables C1 and C2.

Table C1: Summary of aquatic and terrestrial toxicity data for the actives of interest including an indication of the extent of the dataset available

Substance	Water		Soil	
Lasalocid sodium	1 – 8 mg/l	9 data points Algae, inverts, fish	71.8 - > 100 mg/kg	4 data points Plants, earthworms
Semduramicin sodium	19 – 39 mg/l	4 data points Algae, inverts, fish	0.77 – 1,000 mg/kg	7 data points Plants, earthworms, microbes
Diclazuril	No data		No data	
Robenidine	0.036 - 0.56 mg/l	8 data points Algae, inverts, fish	No effects at 0.36 - > 1,000 mg/kg	3 data points Microbes, plants, earthworms
Maduramicin ammonium	No data		0.25 mg/kg (NOEC)	2 studies Microbe, plants
Decoquinate	No data		No effects at 1.28 - > 1,000 mg/kg	4 data points Microbes, plants and earthworms
Monensin sodium	0.98 - 16.6 mg/l	2 data points Algae, inverts and fish	4 - > 1,000 mg/kg	10 data points Plants, earthworms
Narasin	0.77 – 20.56 mg/l	5 data points Algae, inverts and fish	17.9 – 46.4 mg/kg	3 data points Earthworms
Nicarbazin	No data		No data	
Salinomycin	1.14 - 32.2 mg/l	11 data points Algae, inverts and fish	1.3 - > 100 mg/kg	19 data points Plants, worms
Halofuginone	No data		200 mg/kg	2 data points Microbes and plants
Sulphadiazine	0.135 – 403 mg/l	8 data points Algae, inverts	100 – 1,200 mg/kg	3 data points
Trimethoprim	16 – 130 mg/l	3 data points Algae	No data	
Neomycin	> 800 and 2,829 mg/l	2 data points	No data	
Chlortetracycline hydrochloride	0.05 - > 128 mg/kg	6 data points Algae, inverts	No data	

This table shows the size and extent of the available datasets by indicating both the number of data points available and also the range of data available in terms of types of organisms covered, such as algae, invertebrates and fish for the aquatic environment and microbes, plants and earthworms for the terrestrial environment.

Table C2: Summary of available information on the fate of the 15 actives of interest

Substance	Fate (soil and water)	Bioaccumulation/Metabolism
Lasalocid sodium	Limited data available on fate. Soluble in water (1.06 g/l). Log Koc of 2.9 - 3.2 indicates limited potential to adsorb to soil and sediment. Half-lives in soil reported in the order of days to weeks (0.6 - 14.2 days). No degradation data available for water – hydrolysis study showed 32 % degraded at pH 9	No data on bioaccumulation available. Reported log Kows of 1.4 and 2.3 indicate risk of bioaccumulation is low.
Semduramicin sodium	Undergoes photolysis in water with half-lives in the order of a few days reported. Hydrolysis occurs with half-lives in the order of 11 - 115 days reported depending on pH. Half-lives in soil reported in the order of 42 – 104 days. Log Kocs ranged from 2.18 – 3.2 with potential for absorption greater in clay than sandy soils	Reported log Kows of 2.2 – 2.6 indicate it does not have significant potential to bioaccumulate. Following dosing, levels in tissues were found to deplete readily after withdrawal. Following treatment, ingested drug was found to be broken down into a number of metabolites.
Diclazuril	No data available	No data available on bioaccumulation. Noted that following treatment the majority of administered dose excreted within 10 days of which the majority is the parent compound.
Robenidine	No degradation data available for water. Low solubility in water reported (<1 mg/l) Half-lives in soil reported in the range of 6 – 162 days. Log Koc of > 5.6 reported where pH greater than 6.	No specific data on bioaccumulation is reported, however a log Kow of 3.3 has been reported for robenidine. Rapid excretion observed following treatment. with unchanged robenidine being the main compound excreted.
Maduramicin ammonium	No data available	No data on bioaccumulation is available. Data on metabolism showed that following dosing, 6 – 86 % of the applied dose was excreted after 3 days withdrawal. Excreted products included the parent compound and metabolites.
Decoquinate	Reported to be poorly soluble in water (0.06 mg/l). No data available on fate in water or manure. Log Koc of > 5.6 determined – indicates potential to adsorb to soil or sediment. Half-lives in soil reported in the range of 96 – 140 days.	No info on bioaccumulation available. Log Kow of 5.2 - 5.5 reported indicating potential to bioaccumulate. Metabolism studies noted that 90 - 100 % of dose excreted within 24 - 48 hrs with most being in the form of unchanged decoquinate.
Monensin sodium	Half-lives in soil and manure have been reported in the order of 2 – 18 days. Degradation is thought to be slower in anaerobic conditions. Limited info related to fate in water, however a study suggests photolysis with a decline of 40 % in the concn of monensin after 30 days exposed to	No data on bioaccumulation was located. Log Kow in the range of 2.8 - 5.4 which indicates potential to bioaccumulate. Metabolism studies show excretion via the faeces with both unchanged monensin and metabolites.

	natural summer sunlight. Kocs in the order of 61 - 308 were reported, which suggested limited potential to adsorb to soil or sediment. Low solubility in water reported (4.8 - 8.9 mg/l).	
Narasin	Degradation and dissipation studies in soil suggest half-lives in the order of 4 - 22 days, although half-lives up to 49 days have been reported. With respect to water, no information is available on biodegradation, however photolysis half-lives of 1.5 - 3.5 days have been reported. A log Koc of 6 - 6.88 is reported for narasin, which suggests potential to adsorb to soil and sediment	Log Kows of 3.84 - > 6.2 have been reported ,which suggest the potential for bioaccumulation. Excretion via the faeces for both unchanged narasin and metabolites has been reported.
Nicarbazin	No data available	No data available on bioaccumulation. Limited data showed moderate to high potential for metabolism for this group of chemicals.
Salinomycin sodium	Half-lives in soil have been reported as 8 – 16 days. Log Kocs in the range 2.2 – 2.8 suggest adsorption to sediment and soil is not significant. No data on fate in water was available.	A log Kow of 2.4 has been reported – doesn't indicate significant potential to bioaccumulate. Following treatment, excreted rapidly in the form of both unchanged salinomycin and metabolites.
Halofuginone	No data available	No bioaccumulation data available. Metabolism data indicated that following treatment, majority of absorbed dose excreted within 12 - 14 hrs with majority as unchanged halofuginone.
Sulphadiazine	No data available	No data available
Trimethoprim	A half-life of 110 days was reported in soil. Log Kocs of 3.2 – 3.6 were reported, indicating potential to adsorb to soil and sediment. Half-life in marine sediment of 75 -100 days was reported. Limited data on fate in water – no photolysis observed after 42 days and volatilisation and hydrolysis not thought to be important processes.	No bioaccumulation data available. Measured log Kow of 0.91 suggests unlikely to bioaccumulate.
Neomycin	Half-life in manure and slurry reported as approx 30 days. No other fate data available.	No data on bioaccumulation. Metabolism studies note that 90 % of applied dose is excreted in the faeces, with the majority in the form of unchanged parent compound.
Chlortetracycline hydrochloride	Limited data available on fate. Half-life in soil reported as > 30 days. No Koc data, however data for other tetracyclines indicated low mobility in soils.	No data available on bioaccumulation. Detected in manure, urine and faeces. In a study, showed 46 % of applied dose was excreted in urine and faeces after 8 hrs, 22 % of which was unchanged trimethoprim.

Appendix D: Pigs and cows

It is possible that because pigs are susceptible to certain influenza viruses, they may also become infected with the Asian HPAI H5N1 virus even if it is not pathogenic to pigs. If pigs turn out to be carriers, then pig carcases and pig slurry will need to be considered in any disposal option. As with any animal, the potential for the presence of zoonoses exists. Table D1 is a non-exhaustive list identifying zoonotic agents and their presence or absence in the animal or its slurry. Appendix A lists the environmental conditions that may eliminate or cause these organisms to proliferate (such as extremes of pH, temperature, oxygen).

Table D1: The presence of zoonotic agents in pig and cattle material

Hazard	**Carcasses**		**Infected litter / manure / slurry**	
	Pigs	**Cattle**	**Pigs**	**Cattle**
Zoonoses				
Avian influenza	Yes	Possible ?	Yes	Possible?
Salmonella	Yes	Yes	Yes	Yes
Campylobacter coli / jejuni	Yes	Yes	No	No
Escherichia coli 0157	No	Yes	No	Yes
Leptospirosis	Yes	Yes	Yes	Yes
Cryptosporidiosis	No	Yes - calves	No	Yes
Bovine tuberculosis	Yes	Yes	No	No
Streptococcus suis	Yes	No	No	No
Psittacosis (*Chlamydophila psittaci*)	No	No	No	No
Q fever (*Coxiella burnettii*)	No	Yes	No (afterbirth yes)	No (afterbirth yes)
Ringworm	Yes	Yes	No	No
Bovine spongiform encephalopathy	No	Yes	No	No
Foot and Mouth	Yes	Yes	Yes	Yes
Listeria monocytogenes	Yes	Yes	No	No
Anthrax (*Bacillus anthracis*)	Yes	Yes	No	No
Clostridium botulinum	Yes	Yes	Yes	Yes
Giardia duodenalis	Yes	Yes	Yes	Yes
Yersinia enterocolitica	Yes	Yes	Yes	Yes
Toxoplasma spp	Yes - rare	Yes	No	No
Erysipelothrix spp	Yes	No	Yes	No

Appendix E: Host range

This appendix expands on Section 5 and includes information on the host range of Asian HPAI H5N1, where the virus replicates within the host, how quickly the host is killed, symptoms, and what material might be infective. Any carcase of an animal suspected to have died from HPAI should be treated as potentially infective material.

The host range includes:

- **Poultry** (including chickens and turkeys). Poultry excrete virus in the short time between infection, incubation and death (two to three days), usually via the respiratory secretions and faeces. Transmission is usually by contact with respiratory secretions, conjunctiva or faecal material, rarely by airborne particles (UN FAO 2004). Flock mortality up to 100 per cent. Virus should be treated as systemic, that is, present throughout the body of the host animal, so all tissues should be treated as infective. DPIE AusVetPlan (1996). Disease spreads rapidly within flock by direct contact, while indirect spread occurs via contaminated people, articles, feed and vehicles. HPAIs airborne spread between flocks is thought to be insignificant. Virus can be isolated from eggs laid by infected poultry; the virus can penetrate intact eggshell. (UN FAO, 2005). Therefore, all products and waste material should be considered as infective and require appropriate disposal.
- **Ducks and geese.** As described in the introduction, the current H5N1 strain arose in ducks or geese. It is quite possible that whilst the strain is highly pathogenic to poultry, the same strain demonstrates low (or non) pathogenicity to ducks and geese (Hulse-Post *et al*., 2005). Thus, ducks and possibly geese may carry and excrete virus without showing signs of disease.
- **Cats** (all felids). In cats, the virus replicates in the respiratory tract; however, the virus is excreted at low titre in faeces (Kuiken, 2004). This was a small study with mortality rate of 33 per cent (death within six days of infection). Infection noted horizontally, from infected to previously non-infected animals. Whilst this study used a high titre of infectious material, there are non-peer reviewed reports of cats in Thailand dying of H5N1 infection after eating infected chickens. Infection and death observed in experimental domestic cats and inadvertently infected tigers and leopards. Butler (2006) comments that a previously unreported survey showed the cats excreted virus in faeces and in coughed-out droplets
- **Mice and rats**. The virus replicates in the respiratory tract, but shedding of virus in faeces can not be ruled out. Therefore, rodents should not be permitted to come into contact with infected material; the possibility exists that rodents may shed virus into water that birds use, and that these birds then become infected (Dybing *et al*., 2000). Disease was systemic, with mortality up to 100 per cent (dependent upon virus strain).
- **Ferrets** (reads across to all mustelidae such as badgers, stoats, otters, mink, polecats, pine martens). Nasally inoculated HPAI H5N1 produces systemic (through out the body) disease in ferrets (Govorkova *et al*., 2005; Zitzow *et al*., 2002). Where the virus used in an experiment caused severe disease in humans, it caused more than 50 per cent mortality in infected ferrets (Govorkova *et al*., 2005).
- **Pigs** (presumably including wild boar). The virus reproduces in the respiratory tract, not in the alimentary canal – currently no data exists to suggest that H5N1 virus is shed in pig faeces (Webster *et al*., 2005). It is not currently thought to transmit readily between pigs.

- **Humans.** The virus replicates in respiratory tract (and gastrointestinal tract). Incubation two to 17 days. Occasionally systemic. Available evidence in August 2006 suggests that the HPAI H5N1 virus does not readily transmit between humans. Human influenza is transmitted by inhalation of infectious droplets and droplet nuclei, by direct contact, and perhaps, by indirect (fomite) contact, with self-inoculation onto the upper respiratory tract or conjunctival mucosa. The relative efficiency of the different routes of transmission has not been defined. For human influenza A (H5N1) infections, evidence is consistent with bird-to-human, possibly environment-to-human, and limited, non-sustained human-to-human transmission to date. (WHO, 2005). Where disease has been detected, the mortality rate is approximately 50 per cent. Bean *et al*., (1982) point out that self-inoculation with human influenza, for example via hand contact with contaminated surfaces, may result in transmission. There is no reason to believe that this situation is different with HPAI H5N1.
- **Dogs**. Over 25 per cent of 629 dogs in Thailand have recently been noted as carrying antibodies to the H5N1 virus. No evidence yet noted that the dogs can spread H5N1.
- **Cattle and horses**. Cattle and horses are not currently thought to be hosts for HPAI H5N1.

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