AUSTRALIAN VETERINARY EMERGENCY PLAN

AUSVETPLAN

Response strategy

Avian influenza

Version 5.0

AUSVETPLAN is a series of technical response plans that describe the proposed Australian approach to an emergency animal disease incident. The documents provide guidance based on sound analysis, linking policy, strategies, implementation, coordination and emergency-management plans.

National Biosecurity Committee

ISBN 0 642 24506 1 (printed version)

ISBN 1 876 71438 7 (electronic version)

Licence

Moral rights

Disclaimer and warranty

Text under development

In this manual, text placed in square brackets [xxx] indicates that that aspect of the manual remains unresolved or is under development; such text is not part of the official manual. The issues will be further worked on by experts and relevant text included at a future date.

Contact information

If you have any requests or inquiries concerning reproduction and rights, or suggestions or recommendations, you should address these to:

AUSVETPLAN – Animal Health Australia
Executive Manager, Emergency Preparedness and Response
PO Box 5116
Braddon ACT 2612
Tel: 02 6232 5522
email: aha@animalhealthaustralia.com.au

Approved citation

Animal Health Australia (2021). *Response strategy: Avian influenza* (version 5.0). Australian Veterinary Emergency Plan (AUSVETPLAN), edition 5, Canberra, ACT.

DISEASE WATCH HOTLINE: 1800 675 888

The Disease Watch Hotline is a toll-free telephone number that connects callers to the relevant state or territory officer to report concerns about any potential emergency disease situation. Anyone suspecting an emergency disease outbreak should use this number to get immediate advice and assistance.

Edition 1
1991

Edition 2
Version 2.0, 1996 (major update)
Version 2.1, 2000 (minor amendments and updated OIE International Animal Health Code)

Edition 3
Version 3.0, 2002 (major update and inclusion of new cost-sharing arrangements)
Version 3.1, 2007 (interim update in response to epidemic of HPAI in Asia, January 2004)
Version 3.2, 2007 (further revised version to take account of terminology and policy developments)
Version 3.3, 2008 (minor revision to Section 3.1.4)
Version 3.4, 2011 (minor update and inclusion of a reference for vaccination use policy; disease summary moved to Appendix 1 and renamed 'Key features of AI', in line with other disease manuals)

Edition 5
Version 5.0, 2021 (major update)

Contents

Tables

Figures

1 Introduction

1.1 This manual

1.1.1 Purpose

As part of AUSVETPLAN (the Australian Veterinary Emergency Plan), this response strategy contains the nationally agreed approach for the response to an incident – or suspected incident – of avian influenza (AI) in poultry, or cage (aviary) or zoo birds in Australia. It has been developed to guide decision making to ensure that a fast, efficient and effective response can be implemented consistently across Australia with minimal delay.

1.1.2 Scope

This response strategy covers AI caused by avian influenza virus.

This response strategy provides information about:

- the disease (Section 2)
- the implications for Australia, including potential pathways of introduction; social, environmental, human health and economic effects; and the critical factors for a response to the disease (Section 3)
- the agreed policy and guidelines for agencies and organisations involved in a response to an outbreak (Section 4)
- declared areas and premises classifications (Section 5)
- biosecurity controls, including quarantine and movement controls (Section 6)
- response surveillance and establishing proof of freedom (Section 7).

The key features of AI are described in the **Avian influenza fact sheet** (Appendix 1).

1.1.3 Development

The strategies in this document for the diagnosis and management of an outbreak of AI are based on risk assessment. They are informed by the recommendations in the World Organisation for Animal Health (OIE) *Terrestrial animal health code* (Chapter 10.4) and the OIE *Manual of diagnostic tests and vaccines for terrestrial animals* (Chapter 3.3.4). The strategies and policy guidelines are for emergency situations and are not applicable to policies for imported animals or animal products.

This manual has been produced in accordance with the procedures described in the **AUSVETPLAN *Overview***, and in consultation with Australian national, state and territory governments; the relevant livestock industries; nongovernment agencies; and public health authorities, where relevant.

In this manual, text placed in square brackets [xxx] indicates that that aspect of the manual remains unresolved or is under development; such text is not part of the official manual. The issues will be worked on by experts and relevant text included at a future date.

1.2 Other documentation

This response strategy should be read and implemented in conjunction with:

- other AUSVETPLAN documents, including the operational, enterprise and management manuals; and any relevant guidance and resource documents. The complete series of manuals is available on the Animal Health Australia website[1]
- relevant nationally agreed standard operating procedures (NASOPs).[2] These procedures complement AUSVETPLAN and describe in detail specific actions undertaken during a response to an incident. NASOPs have been developed for use by jurisdictions during responses to emergency animal disease (EAD) incidents and emergencies
- relevant jurisdictional or industry policies, response plans, standard operating procedures and work instructions
- relevant Commonwealth and jurisdictional legislation and legal agreements (such as the Emergency Animal Disease Response Agreement – EADRA[3]), where applicable.

1.3 Training resources

EAD preparedness and response arrangements in Australia

The EAD Foundation online course[4] provides livestock producers, veterinarians, veterinary students, government personnel and emergency workers with foundation knowledge for further training in EAD preparedness and response in Australia.

[1] www.animalhealthaustralia.com.au/our-publications/ausvetplan-manuals-and-documents
[2] www.animalhealthaustralia.com.au/what-we-do/emergency-animal-disease/nationally-agreed-standard-operating-procedures
[3] https://animalhealthaustralia.com.au/what-we-do/emergency-animal-disease/ead-response-agreement
[4] www.animalhealthaustralia.com.au/emergency-animal-disease-training-program

2 Nature of the disease

Avian influenza (AI) is a highly contagious viral infection, primarily of avian species.

AI virus infections vary in pathogenicity. Infections are classed as being with low pathogenicity AI (LPAI) or highly pathogenic AI (HPAI) (see Section 2.1). Clinical manifestations vary with the subtype and strain of virus, the avian species infected, and the presence of other diseases.

From 1955, the disease was known as either 'virulent avian influenza' or 'fowl plague'. An international meeting on AI in 1984 recommended that the name 'highly pathogenic avian influenza' be used to describe the most pathogenic form of infection. The current nomenclature, as of 2020, endorsed by the World Organisation for Animal Health (OIE) is 'high pathogenicity avian influenza'.

Changes in understanding of the epidemiology of AI virus infections, particularly the capacity of some AI viruses to infect humans and the potential risk this poses for the emergence of human-to-human transfer, have led to a reassessment of the management of AI virus infections in poultry.

This manual considers primarily the consequences of infection of poultry in Australia with any AI virus. It distinguishes between infection with HPAI, LPAI of the H5 and H7 subtypes (LPAI (H5/H7)), and LPAI of other subtypes (LPAI (not H5/H7)) (see Section 2.1).

For the purposes of this manual, 'poultry' means chickens, turkeys, guineafowl, ducks, geese, quail, pigeons, pheasants, partridges, emus and ostriches reared or kept in captivity, including commercial and backyard. This is different from the OIE definition:[5]

Poultry means all domesticated birds, including backyard poultry, used for the production of meat or eggs for consumption, for the production of other commercial products, for restocking supplies of game, or for breeding these categories of birds, as well as fighting cocks used for any purpose.

Birds that are kept in captivity for any reason other than those reasons referred to in the preceding paragraph, including those that are kept for shows, races, exhibitions, competitions or for breeding or selling these categories of birds as well as pet birds, are not considered to be poultry.

2.1 Aetiology

AI virus classification

All influenza viruses are members of the family *Orthomyxoviridae*. Influenza viruses are enveloped RNA viruses that are categorised into four types – A, B, C and D – based on the antigenic character of the internal nucleoprotein (NP) and matrix (M1) protein. All AI viruses belong to the influenza A type, and only influenza A viruses have been isolated from avian species; influenza viruses of the B, C and D types have never been isolated from birds.

Influenza A viruses are further divided into subtypes based on the antigenic characteristics of the haemagglutinin (HA) and neuraminidase (NA) surface glycoproteins. To date, 18 HA subtypes (H1–H18) and 11 NA subtypes (N1–N11) have been described. HA subtypes 17 and 18, and NA subtypes 10 and 11 have only been identified in bats (Wu et al 2014). Each influenza virus contains one of each of these surface glycoprotein variants.

[5] www.oie.int/fileadmin/Home/eng/Health_standards/tahc/current/glossaire.pdf

Influenza B viruses and subtypes of influenza A virus are further characterised into many different strains. New strains of influenza viruses appear and replace older strains through antigenic drift or shift.

Pathotypes

AI viruses are classified[6] into two pathotypes – HPAI and LPAI – based on either the lethality of the virus in experimentally inoculated chickens or molecular characteristics. The pathogenicity of AI virus infection in one species is not predictive of the pathogenicity in other species.

HPAI

HPAI viruses either have an intravenous pathogenicity index (IVPI) in 6-week-old chickens greater than 1.2 or cause at least 75% mortality in 4–8-week-old chickens infected intravenously.

Alternatively, the AI virus can be sequenced to determine the sequence of amino acids present at the cleavage site (HA0) of the HA molecule. The sequence is compared with those of other HPAI isolates; if the amino acid motif is similar to that of other HPAI isolates, the isolate being tested should be considered to be HPAI.[7]

LPAI

LPAI viruses are all influenza A virus subtypes that are not HPAI viruses (as defined above).

Pathogenicity

Pathogenicity depends on both the genetic properties of the virus and the host species. In poultry, HPAI virus can cause severe clinical disease. Even LPAI subtypes can be associated with severe clinical disease in the presence of other infectious agents (eg infectious bronchitis virus, infectious laryngotracheitis virus).

The HA gene, specifically the amino acid sequence at the cleavage site (HA0), is the primary determinant of pathogenicity of AI viruses in chickens. The sequence of amino acids at the HA cleavage site determines the HA protein structure and the ability of protease enzymes to cleave the HA protein into two proteins (HA1 and HA2); this cleavage is essential for the virus to replicate and be infectious.

The HA cleavage site in LPAI viruses can only be cleaved by trypsin-like proteases produced by the epithelial cells of the respiratory and intestinal tracts. Infection with, and replication of, LPAI viruses are limited to these regions.

HPAI viruses have an HA cleavage site that is recognised by both trypsin-like and ubiquitous (eg furin) proteases.

Other factors such as the binding between the HA and the host cell receptor are important for host specificity, and cell or tissue tropism.

Antigenic variation – drift and shift

Influenza viruses have a high frequency of genetic changes in the HA and NA regions, which is known as antigenic drift or shift.

Antigenic drift is associated with point mutations in the HA and/or NA genes, resulting in minor antigenic changes in the encoded proteins. Immune pressure, usually associated with natural infection or vaccination in susceptible hosts, has an important role in selecting antigenic variants in mammalian

[6] https://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/3.03.04_AI.pdf
[7] www.cdc.gov/flu/avianflu/index.htm

influenza viruses, but the relative importance of immune pressure in antigenic change of AI viruses is less well documented and understood.

Antigenic shift arises from genetic reassortment between the gene segments of two influenza viruses that infect the same cell, which results in the acquisition of new HA and/or NA antigens in a virus population. This mechanism may cause major genetic changes in the encoded proteins.

Either of these mechanisms, but primarily antigenic drift (Suarez 2008), is thought to lead to acquisition of virulence by an LPAI virus to create an HPAI virus. H5, H7 and H9 subtypes shown to be HPAI have been isolated from diseased poultry. Although not all H5 and H7 subtypes are highly virulent, these two subtypes are considered to include high-risk strains for antigenic drift towards HPAI, even if the initial clinical picture seen in poultry is of low pathogenicity. The change in virulence of the virus is associated with the insertion of additional basic amino acids at the cleavage site of the HA protein.

Antigenic shift in HA or NA proteins has been documented in live poultry markets, and genetic reassortment of genes other than HA and NA has also been reported (Suarez 2000).

Although AI viruses mainly infect birds, they have the potential to adapt and infect other species, including pigs and humans, because they undergo frequent antigenic alterations. Further antigenic adaptations can enable an AI virus to cause sustained human-to-human transmission. Human pandemics are caused by viruses that contain an HA to which human populations are immunologically naive. Such an HA can be introduced into the human population through reassortment among human, porcine and AI virus strains, or through the direct transfer of an AI virus to humans. However, to become a pandemic, the virus must become adapted to humans and highly transmissible from human to human.

2.2 Susceptible species

Birds

Influenza A viruses have been isolated from most major bird families – so far, at least 105 bird species from 26 families have been reported. Experimentally, AI virus can infect almost all commercial, domestic and wild avian species.

Nearly all of the possible HA and NA subtype combinations have been detected in wild birds. In wild waterbirds, the HA and NA glycoproteins appear to be stable (Reid et al 2003). Species in the orders Anseriformes (ducks, geese, swans) and, to a lesser extent, Charadriiformes (shorebirds, waders, gulls) are regarded as important reservoir hosts and disseminators of influenza A viruses, but rarely (with some notable exceptions) display clinical signs of infection. Avian species other than those identified as reservoirs of LPAI virus may have a higher clinical susceptibility to AI virus.

When chickens and turkeys are infected with viruses with HA and NA combinations that have not occurred previously in these species, a proportion of the viruses may mutate and produce strains that cause severe disease. Epidemics largely occur when an H5 or H7 LPAI virus has been introduced to a naive poultry population and then mutates to HPAI. AI circulation in waterfowl and shorebirds is largely LPAI.

Some of the bird orders in which AI viruses have been detected (EFSA Scientific Panel 2005) are:

- Anseriformes (eg ducks, teals, shovelers, geese, swans, shelducks)
- Charadriiformes (eg gulls, curlews, sandpipers)
- Galliformes (eg gallinaceous birds – chickens, turkeys, pheasants, partridge, quail)

- Ciconiiformes (eg egrets, storks, herons)
- Columbiformes (eg pigeons, doves)
- Falconiformes (eg eagles, falcons, buzzards, goshawks, kestrels)
- Strigiformes (eg owls)
- Gruiformes (eg crakes, moorhens, coots, cranes)
- Pelecaniformes (eg cormorants, pelicans, spoonbills)
- Struthioniformes (eg emus)
- Podicipediformes (eg grebes)
- Passeriformes (eg zebra finches, swallows, mynahs, thrushes, orioles, magpies)
- Psitticiformes (eg budgerigars).

Other susceptible mammals

The ecology of AI viruses is complex; it involves not only wild bird reservoir hosts but also interactions between animals and humans, or between wild and domesticated animals.

AI viruses can give rise to host-adapted viruses – such as swine influenza virus, equine influenza virus, canine influenza virus and human influenza virus – that can become endemic.

Sporadic infections of humans with LPAI viruses of wild bird or poultry origin, or with HPAI viruses, can also occur (Swayne 2008).

Nonhuman mammals

Natural AI virus infections have been recorded in horses, mink, cats, dogs, rats, mice, donkeys, ferrets, rabbits, leopards, pigs, foxes, stone martens, palm civets, wild racoons, marine mammals (including seals and whales) and bats. Endemic infection with H1N1 and H3N2 influenza virus containing AI virus genetic components occurs in pigs; H7N7 and H3N8 infection occurs endemically in horses; H3N8 influenza is a common cause of respiratory infection in dogs in the United States; and H3N2 infection has occurred in dogs in Asia and the United States, and in cats in Asia. H17N10 and H18N11 viruses have recently been detected in New World bats, though have not yet been detected in birds (Tong et al 2013); preliminary studies suggest that these may represent an independent ancient reservoir of influenza viruses in bats.

Sporadic infections identified as HPAI in poultry have been reported in nonhuman mammals. H5N1 virus has been isolated from village donkeys in Egypt exhibiting signs of respiratory distress (Abdel-Moneim et al 2010). Fatal infection has been reported in captive civets in Vietnam (ProMED-mail 2005), a stone marten in Europe (ProMED-mail 2006), a lion in China (Chen et al 2016) and a dog in Thailand (Songserm et al 2006). A large number of tigers became fatally infected with H5N1 virus after being fed infected chicken carcasses in Thailand (Keawcharoen et al 2004, ProMED-mail 2004). Inoculated cats and cats fed infected chicken meat became infected with H5N1 virus and died with severe alveolar disease; in-contact cats also became infected (Kuiken et al 2004). Pigs were infected with H7N7 virus during the Netherlands outbreak in 2003 (ProMED-mail 2003, Loeffen et al 2004), and H5N1 viruses have been isolated from pigs in Indonesia (Nidom et al 2010).

AI viruses can experimentally infect dogs, foxes, cattle, pigs, ferrets, rats, rabbits, guinea pigs, mice, cats, mink, nonhuman primates and humans (Swayne & Halvorson 2003, Rimmelzwaan et al 2006, Kalthoff et al 2008).

A referenced list of species from which H5N1 virus has been isolated is maintained by the United States Geological Survey National Wildlife Centre.[8]

[8] www.usgs.gov/media/files/list-species-affected-h5n1-avian-influenza

2.2.1 Zoonotic potential

Humans are susceptible to infection with AI viruses. Natural exposure to H5, H6, H7, H9 and H10 subtypes has caused zoonotic disease in various forms in humans, ranging from mild or inapparent infection to death (Mostafa et al 2018).

Circulating antibodies to H4, H5, H6, H7, H9 and H11 antigens were detected in people in southern China in the late 1970s and 1980s, as well as more recently, indicating probable exposure to LPAI viruses (Zhou et al 1996, Chen et al 2008, Wang et al 2009).

In Australia, poultry workers were infected with H10N7 virus when they were exposed to clinically healthy birds from an infected flock during routine processing (Arzey et al 2012). Experimental exposure of people to H3N8, H3N2, H5N2, H6N1, H9N2, H4N8 and H10N7 isolates indicated that these isolates could infect humans, although they were generally ineffective in virus transmission (Peiris 2009).

Human infection with H5 and H7 AI viruses has led the World Health Organization (WHO) to consider whether a new pandemic human influenza virus could be derived directly from birds. Fortunately, there have been no cases of sustained human-to-human transmission of AI viruses to date. Further information on human infection with AI viruses can be found on the WHO website.[9]

2.3 World distribution

For the latest information on the distribution of AI, refer to the World Organisation for Animal Health (OIE) World Animal Health Information System.[10]

2.3.1 Distribution outside Australia

Serotypes of AI virus occur on all continents where research has been carried out.

Outbreaks of HPAI in North America (H5N2 in the United States in 1983 and Mexico in 1994, and H7N3 in Canada in 2004 and 2007), Italy (H7N1 in 1999), the Netherlands (H7N7 in 2003) and Chile (H7N3 in 2002) all initially involved LPAI. In each case, HPAI virus emerged by mutation of existing LPAI viruses, and AI virus infection became widespread. In 1997, a novel H5N1 virus caused fatal disease in poultry and humans in Hong Kong; disease subsequently appeared in other birds and poultry throughout Asia, and spread across Eurasia and into Africa. Before the emergence of this strain, AI was considered a sporadic high-mortality poultry disease.

In December 2014 and January 2015, the Animal and Plant Health Inspection Service of the United States Department of Agriculture reported the presence of HPAI (H5N2) and HPAI (H5N8) viruses in wild birds in a few states. In January 2015, an HPAI (H5N1) virus was detected in a wild duck in the United States. The H5N1 virus isolated from the wild duck was a new mixed virus (a reassortant) that is genetically different from the Asian avian H5N1 viruses that have caused human infections with high mortality in several other countries (notably in Asia and Africa). No human infections with this new reassortant H5N1 virus have been reported.

The largest outbreak of HPAI recorded in countries of the European Union occurred in 2016–17 (Brown et al 2017).

[9] www.who.int/influenza/human_animal_interface/en
[10] https://wahis.oie.int/#/home

H5N1 influenza virus infection is now considered to be endemic in poultry in some countries, including Bangladesh, China, Egypt, India, Indonesia and Vietnam.[11]

For the latest information on AI outbreaks, refer to the websites of the OIE and the Food and Agriculture Organization of the United Nations.[12]

2.3.2 Occurrence in Australia

Commercial poultry

HPAI

Eight outbreaks of HPAI occurred in Australia between 1976 and 2020, most likely due to LPAI infection being passed from wild birds to commercial poultry, followed by mutation to HPAI. HPAI viruses caused clinical disease in commercial poultry in Victoria in 1976 (H7N7), 1985 (H7N7), 1992 (H7N3) and 2020 (H7N7); in Queensland in 1994 (H7N3); and in New South Wales in 1997 (H7N4), 2012 (H7N7) and 2013 (H7N2) (Swayne & Suarez 2000, Agriculture Victoria 2020,[13] Scott et al 2020). Each time, there was severe disease in affected chicken flocks. All instances had obvious or circumstantial evidence of contact with wild waterfowl or surface water contaminated by wild waterfowl, or an association with free-range farmed ducks (Westbury 1998, Scott et al 2020). There is some evidence that, initially, LPAI may have been involved in the outbreaks in 1976 (Westbury 1998), 1992 (Victorian Department of Agriculture, pers comm) and 1997 (Selleck et al 2003).

Full genome sequencing of 11 Australian H7 isolates suggested that Australian H7 isolates form a monophyletic clade when compared with H7 subtypes worldwide (Bulach et al 2010).

In August 2020, three different strains of AI virus were identified across seven infected properties in Victoria – three egg farms and one composting property (HPAI (H7N7)), two turkey farms (LPAI (H5N2)) and one emu farm (LPAI (H7N7)). This was the largest recorded outbreak response to AI in Australia to date.

LPAI (H5/H7)

Seven LPAI (H5/H7) virus strains have been detected in Australian domestic poultry (Agriculture Victoria 2020, QDAF 2020, Scott et al 2020):

- An LPAI (H7N7) virus was isolated on a duck farm during investigation of an HPAI (H7N7) outbreak in chickens in Victoria in 1976. The ducks showed no signs of clinical disease.
- Antibodies to H5, H7 and other subtypes of AI viruses were detected in commercial domestic ducks during investigation of an HPAI (H7N3) outbreak in chickens in Victoria in 1992.
- LPAI (H5) antibodies were detected on a Tasmanian noncommercial, multispecies smallholding in 2006.
- An LPAI (H5N3) virus was detected in a free-range duck flock in Victoria during routine surveillance in 2012. The source of the virus could not be determined, but it is speculated that the primary source may have been wild birds, which had free access to the range area.
- An LPAI (H5N3) virus was isolated from one duck on a noncommercial holding in Western Australia in 2013. The positive test was an incidental finding during routine surveillance. There was no evidence of infection of in-contact birds or birds in the surrounding area.
- An LPAI (H5N2) virus was detected on a turkey farm near Lethbridge, Victoria, during routine surveillance in 2020.

[11] www.fao.org/news/story/en/item/66118/icode
[12] www.oie.int/downld/AVIAN%20INFLUENZA/A_AI-Asia.htm; www.fao.org/avianflu/en/index.html
[13] https://agriculture.vic.gov.au/biosecurity/animal-diseases/poultry-diseases/avian-influenza-bird-flu

- An LPAI (H7N6) virus was detected on an emu farm near Kerang, Victoria, during routine surveillance in 2020.

LPAI (not H5/H7)

The following LPAI (not H5/H7) detections have been made (QDAF 2020):

- Antibodies to LPAI H1, H4, H5, H7 and H9 subtypes were detected in ducks on a farm in Victoria in 1992.
- LPAI (H3N8) virus was detected on a multi-age commercial duck farm in Victoria in 1992.
- An LPAI (H6N4) virus was isolated from a single duck on a property in Queensland in 2006.
- Chickens in several sheds from a property in New South Wales tested seropositive to LPAI (H6N4) in 2006.
- LPAI (H10N7) virus was detected in 2010 on a chicken farm in New South Wales, where transmission to abattoir workers during processing of the poultry was documented. Phylogenetic analysis of the full HA sequence of the virus involved in this event showed a higher degree of homology with North American H10 viruses (Arzey et al 2012) than with Australian H5, H7 and H9 isolates (Bulach et al 2010, Hansbro et al 2010).
- In April 2012, LPAI (H9N2) virus was confirmed on a turkey farm housing about 26 500 turkeys in three sheds near the Hunter Valley in New South Wales; the source of the infection is unknown.
- In 2012, an LPAI (H4N6) virus was found in ducks of several age groups on a multi-age farm of 2400 ducks on the north coast of New South Wales.
- In 2012, an LPAI (H10N7) virus was detected in a Queensland poultry flock; the source of the infection is unknown, but it is postulated that the primary source may have been wild birds.
- In 2018, an LPAI (H1N2) virus was detected in Queensland in free-range chickens, ducks and guinea fowl (Scott et al 2020).

Wild bird surveillance[14]

HPAI viruses have never been detected in birds sampled as part of the National Avian Influenza Wild Bird Surveillance Program, which is run by Wildlife Health Australia. LPAI viruses of many subtypes – including H1–H13, and H15 – have been isolated from, or demonstrated in, a wide range of wild aquatic birds, including resident and migratory species, in well-separated locations in Australia (Arzey 2004, Haynes et al 2009, Hansbro et al 2010, Grillo et al 2015, WHA 2018). The subtypes detected in wild birds vary between species, locations and years.

LPAI viruses have been detected in:

- Anseriformes (including mallard hybrids, Pacific black duck, Australian shelduck, Australasian shoveler, grey teal, chestnut teal, Australian wood duck, pink-eared duck, magpie goose, hardhead, plumed whistling duck, black swan)
- Charadriiformes (including red-necked stint, red knot, black-tailed godwit, bar-tailed godwit, curlew sandpiper, eastern curlew, lesser noddy, sooty tern, sanderling, silver gull, white-capped noddy, whimbrel, whiskered tern)
- Gruiformes (Australian coot)
- Pelecaniformes (Australian white ibis)
- Procellariiformes (shearwater).

Since 2005, LPAI viruses of H5 subtypes have been detected in Queensland, New South Wales, Victoria, Tasmania, South Australia and Western Australia (Pacific black duck, chestnut teal, grey teal,

[14] https://wildlifehealthaustralia.com.au/ProgramsProjects/WildBirdSurveillance.aspx

Australasian shoveler, red-necked stint, plumed whistling duck and magpie goose, and samples from mixed species, which included these species plus pink-eared duck), and LPAI viruses of H7 subtypes have been detected in Queensland, New South Wales, Victoria, Tasmania and South Australia (Pacific black duck and grey teal, and samples from mixed species, which included these species plus Australasian shoveler and pink-eared duck).

Serological surveillance has detected antibodies to AI virus using a competition enzyme-linked immunosorbent assay (c-ELISA) to the nucleoprotein in (Curran 2012):

- Charadriiformes – black-fronted dotterel, greater sand plover, grey plover, oriental plover, Australian pied oystercatcher, sooty oystercatcher, Caspian tern, common noddy, gull-billed tern, little tern, silver gull, whiskered tern, black-winged stilt, red-necked avocet, bar-tailed godwit, black-tailed godwit, curlew sandpiper, eastern curlew, great knot, grey-tailed tattler, little curlew, red knot, red-necked stint, ruddy turnstone, sanderling, sharp-tailed sandpiper, terek sandpiper, whimbrel, wood sandpiper
- Anseriformes – mallard hybrids, Pacific black duck, Australian shelduck, Australasian shoveler, grey teal, chestnut teal, Australian wood duck, pink-eared duck, magpie goose, hardhead, plumed whistling duck, black swan.

In general, the rate of detection[15] via polymerase chain reaction (PCR) and virus isolation from Australian wild birds is low (1.4–3.2%) compared with other global studies of wild birds (Hansbro et al 2010, Tracey 2010). Internationally, the prevalence of AI virus infection and detection in waterfowl ranges from 0.6% to 26% (Alfonso et al 1995); however, surveillance differs greatly between countries.

Recent studies demonstrate higher rates of detection in certain species (eg 0.45–0.55% in shorebirds versus 2.0–3.6% in waterfowl); this is consistent with findings in other geographic regions (Hansbro et al 2010). Detection rates can be highly variable between sampling periods and locations, and significantly higher in dabbling ducks than in non-waterfowl species (Hansbro et al 2010, Tracey 2010).

Data from outbreaks of HPAI in poultry in southeastern Australia suggest that outbreaks occur during periods of drought following a period of high rainfall (Klaassen et al 2011, Ferenczi et al 2016). Environmental conditions such as drought and flood affect waterbird abundance in local areas and increase the number of serologically naive juveniles entering the population (Roshier et al 2002, 2008; Tracey et al 2004; Klaassen et al 2011). For example, increased rainfall may lead to increased breeding season success for many wild bird species, increased mixing of wild birds on water bodies, and increased movement as birds follow food and move into or out of flooded areas. Flooding may also lead to increased opportunities for wild birds to come into closer contact with poultry. In contrast, periods of drought may lead to increased densities in localised areas (such as around water sources). Therefore, it is likely that environmental conditions will affect the abundance of Anseriformes species and thus the apparent prevalence of AI viruses.

[15] Comparing rates of detection is difficult because surveillance methodologies and strategies differ between studies.

2.4 Epidemiology

2.4.1 Incubation period

The incubation period and infectious period depend on the virus strain, the dose and route of exposure, the species exposed, the age of individuals, and the environment.

Incubation periods are extremely variable, from a few hours to 24 hours in intranasally inoculated birds and 2–3 days in naturally infected birds. Up to 16 days has been reported for layers in cages (Mutinelli et al 2003).

OIE incubation period

For the purposes of the OIE *Terrestrial animal health code,* the incubation period[16] for AI is 21 days.

2.4.2 Persistence of agent and modes of transmission

Appendix 2 provides information on infective dose and virus shedding.

The principal means by which AI viruses initiate outbreaks is thought to be via wild birds contaminating water or food supplies for poultry, or directly contaminating range areas with faeces on free-range farms, and the infection subsequently spreading through the movements of infected live birds, or faecally contaminated eggs, feed, equipment, materials, clothing and footwear. Infected backyard poultry and live bird markets can be a source of AI virus for commercial poultry.

In past outbreaks, dissemination of AI virus between flocks has been primarily attributed to poor biosecurity, involving:

- movement of infected birds (including vaccinated birds)
- live bird markets (movement of birds, contaminated crates and vehicles)
- human-associated movements, such as transporting food, personnel, equipment and vehicles out of premises that are contaminated with infected faeces or respiratory secretions
- centralised egg handling facilities and equipment, particularly shared use of egg trays and fillers
- depopulation activities that infect nearby properties (Henzler et al 2003)
- use of dead bird pick-up or waste collection centres by people from different premises (McQuinston et al 2005).

[16] In the OIE Terrestrial animal health code, 'incubation period' means the longest period that elapses between the introduction of the pathogenic agent into the animal and the occurrence of the first clinical signs of the disease (see www.oie.int/en/what-we-do/standards/codes-and-manuals/terrestrial-code-online-access/?id=169&L=1&htmfile=glossaire.htm).

General properties

Influenza viruses are lipid-enveloped RNA viruses. Stability of AI viruses varies by subtype. In general, influenza viruses are fragile; however, some serotypes can persist longer under optimal conditions. The virus is stable over a pH range of 5.0–8.2.

Environment (including windborne spread)

Environmental conditions have a marked effect on AI virus viability outside the bird. AI viruses are readily inactivated by heating, and remain viable for longer periods in cold and humid environments. They are sensitive to ultraviolet light; however, a long exposure period (>14 hours) is required for inactivation (Sutton et al 2013). In aerosols, viability is prolonged by high relative humidity and low temperature. Low temperature and high moisture levels also prolong virus viability in faeces. Viability of virus in dust in poultry houses has been reported for 2–5 weeks after depopulation (Webster et al 1978), and virus was recoverable from wet manure after 105 days (Fichtner 2003). AI virus was detected in leachates from landfill for nearly 2 years under conditions of neutral pH and low temperature (Graiver et al 2009).

Water

In water, AI viruses appear most stable at a pH of 7.4–8.2, low temperatures (4–17 °C) and fresh to brackish salinities (salinity range of 0–20 000 ppm). Increasing water acidity, temperature or salinity shortens viability times for AI virus (Brown et al 2008a). However, there is significant variation between virus strains.

AI virus can be isolated from lake water and on-farm water where waterfowl are present (Hinshaw et al 1979, Markwell & Shortridge 1982). Virus may remain infective in lake water for up to 4 days at 22 °C and for more than 30 days at 0 °C (Webster et al 1978). However, in some cases, water bodies have virus persisting for more than 100 days (Stallknecht et al 1990, Brown et al 2007).

Influenza viruses have been demonstrated to remain viable in ice, and it is speculated that frozen ponds may act as a reservoir of virus from year to year (Lang et al 2008).

Windborne and aerosol spread

Windborne contamination has not been regarded as important in the spread of infection between properties (Swayne & Suarez 2000). However, more recent research suggests that it may have a role to play. During the outbreaks of HPAI in the United States in 2015, there was evidence of HPAI virus in air samples taken up to 70 m from infected facilities (Torremorell et al 2016). In China, airborne AI virus was detected as far as 100 m downwind of a live poultry market (Wei et al 2018).

Aerosols may have caused some secondary spread during the New South Wales outbreak in 1997. Virus was detected in aerosol samples within 8 m of exhaust fans in buildings housing birds with clinical signs during the 1983–84 Pennsylvania outbreak (Pearson et al 2003), and viral RNA was detected in one aerosol sample during the Canadian outbreak in 2004 (Power 2005a). Close proximity of sheds (<100 m) was considered very important for spread during control activities in the Canadian outbreak (Power 2005b).

Henzler et al (2003) believed that depopulating infected premises early in the infection cycle led to spread of infection to nearby properties; this spread was reduced by orderly marketing of the birds in the normal production cycle after infection had died down. It is thought that any disturbance increases the likelihood and size of the viral plume.

Live animals

Live domestic animals

Poultry

Viruses with the potential to be highly pathogenic for chickens and turkeys can be carried by birds and shed in faeces and from the respiratory tract from 14 to 30 days after the birds recover from the disease. Cloacal shedding can continue for longer than 30 days after infection in the presence of immunosuppressive diseases or other physical stresses.

The importance of spread by live poultry became apparent in the 2004 epidemic in eastern Asia (Tiensin et al 2005).

Cage birds, including psittacines and canaries

AI viruses have been found in aviary birds (Easterday et al 1997), particularly psittacines (parrots, cockatoos and parakeets); however, these birds were possibly infected after capture. Aviary birds have not been determined to be the cause of infection in chickens or turkeys.

Mexico reported to the OIE in February 2014 that LPAI (H7N3) virus had been identified in monk parakeets originating from South America (ProMED-mail 2014). The birds were for the pet trade, and it is unclear whether they were wild caught.

AI viruses isolated from wild birds have not yet been isolated from cage birds.

Live wild (including feral) animals

AI virus is infective for almost all wild waterfowl species, which are an important reservoir for the virus. A virus that is highly pathogenic for poultry could emerge from the pool of viruses in wild birds at any time.

Waterfowl

Wild aquatic birds, such as waterfowl and seabirds, are important reservoirs and can shed AI virus for up to 1 month (Latorre-Margalef et al 2009).

Wild birds other than waterfowl

AI virus has been isolated from other native species such as from the Charadriiformes, but the duration of virus excretion is not known. Crows were reported dead in the repeated outbreaks in Japan (ProMED-mail 2004, Haynes et al 2009).

Game birds

AI virus was recovered from pheasants, partridges and guineafowl for up to 10 days after infection during the outbreak in the United States in 1983–84 (Swayne 2008).

Japanese quail have been reported to have severe clinical signs, including respiratory problems, prostration and diarrhoea. Before death, gasping and nervous signs have also been observed. In experimental studies, infection has been detected in quail faster than in chickens (Yee et al 2009).

Carcasses

Tissues

Tissues of experimentally infected chickens contain infective virus for long periods at various temperatures. AI virus has been detected in blood, brain, heart, lung, liver, spleen, kidney and skeletal muscle of infected chickens for 5–6 days after experimental inoculation with HPAI viruses (Becker & Uys 1967). HPAI viruses have also been detected in the respiratory tract, gastrointestinal tract and bone marrow. Titres varied with subtype but were $10^{2.7}$–$10^{7.3}$ egg infectious dose $(EID)_{50}$/g in breast and thigh meat, $10^{6.0}$ EID_{50}/g in lung tissue and $10^{1.4}$–$10^{8.0}$ EID_{50}/mL in blood (Mase et al 2005). The highest titres are found approximately 2–4 days post-infection. Refer to Appendix 2 for more information on infective dose and virus shedding.

Blood

Detectable titres of AI virus in chicken, turkey and duck blood have been reported. The viraemic phase of HPAI virus varies, depending on the virus strain and the species infected. Blood collected from acutely infected birds or birds showing no clinical signs at slaughter may present some risk (Beato & Capua 2011).

Animal products

All animal products and byproducts from birds and flocks that are infected with AI virus, and farms or other sites contaminated with AI virus should be considered as high risk for the transfer of infection.

Meat, meat products and casings, including use as animal feed

Viral titres in meat and organs vary with virus strain, bird species, tissue type and clinical stage of infection.

AI virus has been recovered from autolysed carcasses of wild birds (other than waterfowl) after 23 days at 4 °C. Virus was re-isolated from the skeletal muscle, bone marrow and other organs of inoculated chickens that had died and were allowed to decompose at room temperature for more than 40 days (Vrtiak & Kapitancik 1967).

Skeletal muscle and some other organs remained infective for 30–40 days at room temperature, and bone marrow remained infective for 60 days at room temperature (Vrtiak & Kapitancik 1967). HPAI virus, protected within chicken meat scraps, is likely to remain viable in the environment at ambient temperatures of 10–35 °C for several days.

Virus was recovered for more than 280 days from chilled muscle and bone marrow of chickens inoculated and killed before exhibiting clinical signs of infection (Purchase 1931).

The rate of detection of HPAI viruses in poultry meat appears to be higher during the early stages of infection. Birds processed during the viraemic stage may contaminate other carcasses with blood or faecal material containing virus. Packaging and the drips that develop during storage are also important, as both can be contaminated with virus from infected carcasses.

Eggs and egg products

During systemic infection with AI virus, the virus replicates within the oviduct. Infected birds will stop laying; however, both LPAI and HPAI viruses have been recovered from yolk, albumen and the shell surface of chicken eggs during natural exposure (Cappucci et al 1985).

The most likely route of viral infection of eggs is faecal contamination of the external surface of the egg during passage through the cloaca or in the nest. Vertical transmission may also play a role. Since AI virus can penetrate cracked or intact shells, nonpasteurised or uncooked egg pulp and pulp products could be a source of virus.

Persistence during embryo development and incubation is most likely through shell contamination (Swayne & Halvorson 2008). Limited studies indicate that embryonated turkey eggs infected with influenza virus survived infection (Samadieh & Bankowski 1971), and poults developed haemagglutination inhibition (HI) titres. An experimental study demonstrated that incubation temperatures (37 °C and 39 °C) significantly promoted AI virus growth (Lang et al 2011).

In naturally infected birds, H5N2 virus could be recovered from eggs (both albumen and yolk contents) laid by broiler breeders and commercial layers up to 18 days after the onset of clinical signs. AI virus can remain viable for at least several (up to 8) days in albumen and yolk of eggs stored at 10–18 °C (Cappucci et al 1985). Virus recovery was higher from albumen than from yolk. Virus was also recovered, at a lower rate, in flocks not showing clinical signs.

The egg contents (albumen and allantoic fluid) and oviduct of naturally infected Japanese quail (*Coturnix japonica*) contained H5N1 virus titres of $10^{4.6}$–$10^{6.2}$ EID_{50}/mL (Promkuntod et al 2006).

Commercial breeder duck eggs are laid on the floor and are often heavily soiled with faeces, and multiple hens will lay eggs in the same nest. External surface contamination with faeces is the major risk, but systemic circulating virus could be shed into the internal contents during egg development in the oviduct.

Animal byproducts

Hides, skin, wool and other fibres

Feathers may be contaminated via faecal material or dust. Systemic infection has been demonstrated to localise in the feather follicle epithelium. AI virus has been detected in feathers from chickens, Japanese quail, turkeys, guineafowl, call ducks (a bantam breed of domesticated duck raised primarily for decoration or as pets) and geese (Beato & Capua 2011).

HPAI (H5N1) virus has been shown to replicate in swan feather epithelial cells and could be detected on detached feathers for up to 160 days when the feathers were kept at 4 °C. When feathers were stored at 20 °C, virus remained infective for 15 days. Virus titres as high as $10^{4.3}$ EID_{50}/mL were detected at 120 days (Yamamoto et al 2010).

Feathers are important byproducts that are used in bedding (quilts) and down jackets. Duck feathers are exported from Australia.

Swill and meatmeal

Rendered meals produced from frames (boned-out skeletons), viscera, blood, feathers, feet, heads, necks, offcuts, birds dead in trucks and discarded live birds are added to poultry feed as poultry offal meal and tallow. They may also be added to pet foods.

Heat-treated poultry offal meal and pet foods are usually cooked at temperatures above 100 °C for several minutes to more than 1 hour, which is sufficient to inactivate AI virus. However, not all pet foods or pet meat (including swill) are heat treated or pasteurised, and uncooked pet foods and pet meats containing poultry products will require treatment to kill AI viruses. If the procedure is not carried out properly or cooked product is subsequently contaminated by unprocessed product, AI virus could persist in the byproduct for several weeks.

Under Queensland legislation (*Biosecurity Act 2014*[17]), it is illegal to feed swill (prohibited feed) – which includes any feed that contains bird materials – to either pigs or poultry.

Waste products and effluent

Waste can be any of the unwanted byproducts of processing. Products that are used in the production of rendered meals may also be discarded as waste. In addition, there will be wastes from hatcheries, laboratories (cultures and specimens, dead birds), farms and egg marketing establishments (unsaleable eggs, eggshells after pulping, solid egg fillers), as well as chicken manure and litter. AI virus has the potential to persist in these products and could be spread by vehicles that transport them, unless the products are treated before movement.

AI virus can remain viable in faeces for up to 35 days at 4 °C and for 6 days at 37 °C. H7N2 virus remained viable for less than 1 week at 15–20 °C (Halvorson 2008).

In liquid manure, AI virus was recoverable after 105 days in freezing conditions, 30–25 days at 4 °C, 7 days at 20 °C and 4 days at 25–32 °C when kept out of direct sunlight, and only 30 minutes at 32–35 °C in direct sunlight (Swayne & Halvorson 2003, Songserm et al 2006).

Experimental inoculation of poultry manure demonstrated no recoverable virus after 24 hours at 25 °C or 15 minutes at 40 °C (Chumpolbanchorn et al 2006).

Nonsusceptible animals

Nonsusceptible animals can act as fomites in transferring virus from infected flocks to uninfected flocks.

It is generally concluded that mice and rats do not play significant roles in the spread of AI virus, but that insects may (Achenbach & Bowen 2011, Nielsen et al 2011). In a study where a large number (*n* = 516) of samples were taken from rodents exposed to HPAI virus, no positive virus isolations were obtained (Nettles et al 1985). Similarly, in a study where 12 rodents were inoculated with LPAI virus, no positive virus isolations were identified (Achenbach & Bowen 2011). Feeding flies with LPAI and HPAI viruses resulted in positive virus isolations; flies are therefore an important pathway to consider for AI spread (Sawabe et al 2006, Nielsen et al 2011).

[17] www.legislation.qld.gov.au/view/html/inforce/current/act-2014-007

People

Transmission from birds to humans can occur through handling of infected live or dead birds, or through close contact with:

- the faeces of infected birds
- respiratory secretions or saliva
- uncooked infected birds, eggs or wild game bird products (via handling or consumption)
- contaminated products, such as litter
- contaminated dust and aerosol during destruction of infected premises.

People can act as fomites in transferring virus (on contaminated shoes and clothing) from infected flocks to uninfected flocks.

Crops, grains, hay, silage and mixed feeds

These commodities have not been identified as being involved in the transfer of AI virus to susceptible birds. However, they could theoretically act as fomites, although the virus is susceptible to desiccation. Heat treatment during the manufacture of commercially prepared pellet poultry feed is usually sufficient to destroy AI virus.

Vehicles, including empty livestock transport vehicles

Vehicles, including empty livestock transport vehicles, can play an important role in transfer of AI virus from farm to farm. This is particularly the case for vehicles involved in transporting dead infected birds, if there is inadequate containment and decontamination between premises (Bowes et al 2004).

Before the initial case is identified, it is possible that vehicles will play a role in disease spread from one premises to another if routine biosecurity controls are not in place. Following confirmation of AI on a premises, biosecurity controls will be more stringent, reducing the likelihood of vehicles contributing to disease spread.

Equipment, including personal items

Persistence of AI virus in faeces and respiratory secretions is important in the spread of the virus via fomites. The moisture content of these components facilitates survival and thus spread of the virus over a wide geographical area on footwear, clothing, equipment and other fomites. This is considered to be the main route of transmission of infection between premises.

Overseas experience has shown that AI virus can spread very rapidly and over long distances by movement of contaminated materials such as bird cages, pallets, egg filler flats, manure, feed, clothing, equipment and vehicles. The time the virus remains viable on eggs and fillers is sufficient to allow wide dissemination. Overall, access of immunologically naive birds to fomites contaminated with infected faecal material poses the greatest risk of spreading infection.

High surface titres (around 10^5 tissue culture infective dose $(TCID)_{50}$/mL) are required for transfer via fomites. Hard, nonporous surfaces (steel and plastic) at 28 °C and 35–40% humidity were able to transfer virus for 24–48 hours; porous surfaces for 24 hours (to hands); and cloth, paper and tissues for less than 8–12 hours (WHO 2007a).

Arthropod vectors

The poultry red mite, a common ectoparasite of poultry worldwide, has been identified as a potential mechanical vector in the spread of AI virus. In a study in 2016, the mite ingested AI virus while feeding on infected chickens and was then able to transmit the virus to specific pathogen–free chickens. (Sommer et al 2016). Mechanical transmission by either invertebrate or vertebrate vectors through contact with infected faeces is possible, but such transmission would be infrequent (Sawabe et al 2011).

Mosquitoes collected at poultry farms during an outbreak of HPAI (H5N1) in central Thailand in 2005 tested positive for the virus (Barbazan et al 2008). A study has also demonstrated that H5N1 virus remained in the gastrointestinal tracts of house flies for at least 24 hours post-exposure (Tyasasmaya et al 2016).

2.5 Diagnostic criteria

2.5.1 Clinical signs

Animals

The clinical signs of AI virus infection are variable and influenced by the virulence of the virus, the species infected, the age of the infected individual, concurrent infection with other agents (eg infectious bronchitis virus, infectious laryngotracheitis virus) and environmental factors such as temperature. Pathogenicity in chickens can vary during an outbreak.

Clinical signs are commonly due to damage associated with:

- intracellular viral replication in tissues
- indirect effects of cell cytokine production (eg cytokine storm)
- ischaemia from vascular thrombosis
- coagulopathy or disseminated intravascular coagulation.

Infection with HPAI viruses

Clinical signs of infection with HPAI viruses result from replication of the virus in the respiratory tract, and subsequent systemic replication in the visceral organs and brain.

Chickens and turkeys

In peracute cases involving sudden death, clinical signs may not be seen. Mortalities occur as early as 24 hours after the first signs of the disease and frequently within 48 hours. Mortality rates of nearly 100% have been reported for peracute and acute cases. In other cases, more diverse visible signs occur, and mortalities can be delayed for as long as a week.

Clinical signs in chickens and turkeys may include severe respiratory signs with excessively watery eyes and sinusitis; cyanosis of the combs, wattle and shanks; oedema of the head; ruffled feathers; loss of appetite; diarrhoea; nervous signs; and sudden death.

A decrease in water and feed consumption may be noticeable, and birds may suffer from egg drop syndrome or misshapen eggs. The last eggs laid after the onset of illness frequently have no shells. Some severely affected hens may recover, but rarely come back into lay.

Disease may spread very slowly in birds reared in cages, such as chickens and quail. Infection may be seen in a restricted area of a house, in single birds, but slowly spread to adjacent cages (Capua et al 2000a, Savill et al 2008).

The disease in turkeys is similar to that in chickens, but is often complicated by secondary infections such as fowl cholera, turkey coryza and colibacillosis.

Ducks

Of five species of North American duck experimentally infected with H5N1 virus, wood duck (*Aix sponsa*) was the only species to exhibit illness or death after inoculation with either of the HPAI viruses A/whooper swan/Mongolia/244/05 (H5N1) (Mongolia/05) and A/duck meat/Anyang/01 (H5N1) (Anyang/01) (Brown et al 2006). Clinical signs were characterised by cloudy eyes, ruffled feathers, rhythmic dilation and constriction of the pupils, severe weakness, incoordination, tremors, and seizures.

Black swans

In experimentally infected black swans (*Cygnus atratus*), which are native to Australia, clinical signs included severe listlessness and neurologic dysfunction, consisting of seizures, tremors and marked incoordination. Death occurred within 3 days post-exposure (Brown et al 2008b).

Ratites

An HPAI (H7N1) virus caused anorexia, depression, enteric signs (brilliant green urine, urates, severe haemorrhagic enteritis, haemorrhagic faeces), swollen throat and neck, nervous signs (including incoordination, wing paralysis, and head and neck tremors) and 30% mortality in young ostrich poults (Capua et al 2000a, Mutinelli et al 2003). An HPAI (H5N2) virus isolated from an emu in Texas in 1993 was isolated widely from internal organs, and from cloacal and tracheal swabs for 2–12 days post-infection (Clavijo et al 2001). The virus did not cause clinical signs when inoculated into ostriches.

Wild birds

A list of target wild bird species with known associations with HPAI is provided in European Union legislation.[18]

For further information on clinical signs in wild birds, see the Wildlife Health Australia fact sheet *Avian influenza in wild birds in Australia*.[19]

Infection with LPAI viruses

Chickens and turkeys

Clinical signs of infection with LPAI viruses in chickens and turkeys range from inapparent to mild or severe respiratory disease. The signs can be confused with other respiratory tract infections.

Layer and broiler breeder chickens may show loss of appetite, decreased water consumption, depression, drops in egg production by up to 45% and misshapen eggs. Recovery of egg production to normal or 2–3% below normal typically occurs in 2–4 weeks.

Mortality ranges from 3% in caged layers to 15% in meat chickens; in free-range layers, mortality has increased by 0.20% in 1 week (Germeraad et al 2020). Generally, mortality is expected to be less than 5% (Swayne et al 2013).

[18] https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:32010D0367&from=EN

[19] www.wildlifehealthaustralia.com.au/Portals/0/Documents/FactSheets/Avian/Avian_Influenza_in_Wild_Birds_in_Australia.pdf

The severity of infection in turkeys varies with the virus subtype, the age of the birds and the presence of secondary infections. Common signs include respiratory distress, rales and snicking, progressing to severe dyspnoea. Signs may also include swelling of the infraorbital sinuses, conjunctivitis, anorexia, fever, ruffled feathers and depression. Egg production may drop by 30–80%, with misshapen, fragile and whitish eggs produced during the acute phase. Mortality rates vary from 5% to 97%, depending on age, and morbidity may be up to 100% (Mutinelli et al 2003). Turkeys are generally more severely affected by reproductive disease than chickens (USAHA 2008).

Ratites

In ostriches, clinical signs are highly variable and may be absent (with only serological evidence of infection) (Panigrahy & Senne 1998), but may include green urine, respiratory distress, enteritis, weakness and death. Clinical signs are more severe in young poults (Allwright et al 1993), in which mortality may be as high as 30% (Jorgensen et al 1998).

Respiratory signs were reported in rheas and emus (in the United States) infected with LPAI H5N2 and H7N1 viruses (Panigrahy et al 1995). No clinical signs were detected in infected emus during the 1997 outbreak in New South Wales (OCVO 2010).

Guineafowl, pheasant, partridge and quail

Severe conjunctivitis was consistently detected in guineafowl breeders infected with LPAI (H7N1) virus (Mutinelli et al 2003). Signs in guinea broilers included respiratory signs, nervous signs (including opisthotonus), torticollis and paralysis of the wings; mortality was up to 30%.

Pheasant and partridge are susceptible to infection and clinical disease.

Quail may show no clinical signs of infection. Infection appears to spread slowly in quail, and they do not always produce an antibody response to infection. Therefore, surveillance programs using serology and virus isolation may require higher levels of testing than for chickens to detect infection.

Ducks

Infection with LPAI virus in domestic ducks and mallards typically results in subclinical infection; however, tufted ducks have been reported to present with neurologic signs, including head tilt, circling, loss of balance and drooping wings (Gavier-Widen et al 2012).

2.5.2 Pathology

Gross lesions

In LPAI virus infections, lesions may be seen in the sinuses, characterised by catarrhal, serofibrinous, mucopurulent or caseous inflammation. The tracheal mucosa may be oedematous, with an exudate varying from serous to caseous. The air sacs may be thickened and have a fibrinous to caseous exudate. Catarrhal to fibrinous peritonitis and egg yolk peritonitis may be seen. Catarrhal to fibrinous enteritis may be seen in the caecum and/or intestine, particularly in turkeys. Exudates may be seen in the oviducts of laying birds (Easterday et al 1997).

In HPAI virus infections, including the peracute form of the disease, chickens may not show any gross lesions; such chickens die 1–2 days post-infection. In Hong Kong and Italy in 1997, following acute infections in chickens, severe lung congestion, haemorrhage and oedema were observed in dead chickens; other organs and tissues appeared normal.

With the acute form of HPAI virus infection, more diverse visible lesions are evident, beginning 3–5 days after infection. Chickens have ruffled feathers, congestion and/or cyanosis of the comb and wattles, and swollen heads. The changes in the comb and wattles progress to dark red, then to blue, depressed areas of ischaemic necrosis. Internally, acute infections with HPAI viruses cause haemorrhagic, necrotic, congestive and transudative changes. The oviducts and intestines often have severe haemorrhagic changes. As the disease progresses, the pancreas, liver, spleen, kidney and lungs can display yellowish necrotic foci. Pancreatitis was a common lesion in chickens and turkeys infected with HPAI (H7N1) virus (Mutinelli et al 2003).

Petechiae and ecchymoses cover the abdominal fat, serosal surfaces and peritoneum. The peritoneal cavity is frequently filled with yolk from ruptured ova, associated with severe inflammation of the air sacs and peritoneum in birds that survive 7–10 days. Haemorrhages may be present in the proventriculus, particularly at the junction with the gizzard (Swayne & Suarez 2000).

In ostriches, gross lesions include severe haemorrhagic enteritis, and liver degeneration and necrosis (Capua et al 2000a, Mutinelli et al 2003).

Microscopic lesions

The histological lesions associated with the gross pathological changes described above are not definitive for HPAI, although vasculitis in the brain and other organs may be highly suggestive of the disease.

Pathogenesis

Gallinaceous birds

Pathogenesis begins by inhalation or ingestion of infectious AI virions. In both HPAI and LPAI virus infections, the initial replication site is usually the nasal epithelium. The viral HA adsorbs to host endothelial cell receptors containing sialic acid–bound glycoproteins. Proteolytic cleavage of the HA into H1 and H2 is essential for fusion and initiating receptor-mediated endocytosis. For all AI pathotypes, trypsin-like enzymes on the surface of epithelial cells of the respiratory and intestinal tract allow cleavage of the surface HA and entry of virus particles into the cell, where multiple replication cycles occur with the release of infectious virions.

For LPAI viruses, replication is limited to tissues with trypsin-like enzymes – usually the respiratory and intestinal tracts – but may spread systemically in some species to kidney tubules, pancreatic acinar epithelium, oviduct and other epithelial cells that have trypsin-like enzymes (Swayne & Suarez 2000).

HPAI viruses can be cleaved by protease (furin-like) enzymes in addition to trypsin-like enzymes; this allows them to enter and replicate in other tissues. After initial replication in the respiratory or intestinal epithelium, virions may invade the submucosa, replicating in endothelial cells, and spread via the vascular or lymphatic system to infect other cell types in visceral organs, brain and skin. Alternatively, they may become systemic before extensive replication in vascular endothelial cells. Macrophages and heterophils play a key role in systemic virus spread (Swayne & Halvorson 2003). Initial visceral replication may be seen as early as 24 hours after intranasal infection, with high titres present by 48 hours.

Nongallinaceous birds

In nongallinaceous birds, the pathogenesis is less well understood.

2.5.3 Differential diagnosis

HPAI in birds should be suspected whenever there is high or escalating mortality and sudden death (with no known cause apparent) with severe depression, loss of appetite, nervous signs, watery diarrhoea, severe respiratory signs and/or a drastic drop in egg production. The likelihood of HPAI is increased by the presence of facial subcutaneous oedema, swollen and cyanotic combs and wattles, and petechiae on serosal surfaces.

LPAI may not cause marked disease, but any drop in food or water consumption, decrease in egg production, increase in egg abnormalities or increase in mortalities should be investigated as a potential AI incident.

AI in chickens is frequently indistinguishable on clinical and postmortem examination from:

- viral diseases
    - Newcastle disease
    - infectious laryngotracheitis
- bacterial diseases
    - erysipelas
    - fowl cholera
    - *Escherichia coli* cellulitis of the head
    - acute pasteurellosis
    - mycoplasmosis
    - ornithobacteriosis
    - infectious coryza
    - avian chlamydiosis
- fungal diseases
    - aspergillosis
- other causes
    - acute poisoning
    - heat exhaustion
    - severe water deprivation
    - misadventure associated with high mortality (eg smothering, heat stress, dehydration).
    - botulism.

2.5.4 Laboratory tests

Because pathological changes are not definitive for AI, diagnosis needs to be confirmed by isolation and characterisation of the causative virus. Relevant laboratory tests should be performed to exclude Newcastle disease and bacterial septicaemias from the differential diagnosis, particularly to identify mixed infections with less pathogenic forms of AI.

If an outbreak is confirmed to be caused by an HPAI virus, this agent may also be classified as a security sensitive biological agent (SSBA), to which regulatory requirements (eg for handling and reporting) may apply. However, emergency situations, including emergency animal disease (EAD) outbreaks, can be exempted from some SSBA regulatory requirements. Clarification should be sought from the SSBA responsible officer at the facility concerned.

Samples required

Samples should be taken both from live, clinically affected birds and from recently dead birds.

Cloacal, oropharyngeal or tracheal swabs; and/or fresh faeces, brain and/or lung tissue; and clotted blood/serum may be taken from live birds.

Alimentary tract tissues (proventriculus, pancreas, intestine, caecal tonsil); respiratory tract tissues (trachea, lung); cloacal, oropharyngeal and tracheal swabs; and fresh faeces should be collected from recently dead or euthanased birds.

See also Section 2.5.5.

Transport of specimens

Specimens should be submitted in accordance with agreed state or territory protocols. Specimens should initially be forwarded to the state or territory laboratory for appropriate analysis, and assessment of whether further analysis will be required by the CSIRO Australian Centre for Disease Preparedness (CSIRO-ACDP), Geelong.

If the state or territory laboratory deems it necessary, duplicate samples of the specimens should be forwarded to CSIRO-ACDP for emergency disease testing, after the necessary clearance has been obtained from the chief veterinary officer (CVO) of the state or territory of the suspect case, and after the CVOs of Victoria and Australia have been informed about the case and the transport of the specimens to Geelong (for the first case). Sample packaging and consignment for delivery to CSIRO-ACDP should be coordinated by the relevant state or territory laboratory.

For further information, see the **AUSVETPLAN management manual *Laboratory preparedness***.

Packing specimens for transport

Unpreserved tissue, swab and blood specimens should be forwarded with water ice or frozen gel packs (dry ice or liquid nitrogen if a delay of more than 48 hours is expected) in an International Air Transport Association–approved specimen transport container.

For further information, see the **AUSVETPLAN management manual *Laboratory preparedness***.

2.5.5 Laboratory diagnosis

The initial approach to AI diagnosis is screening by real-time PCR. Primary screening uses a pan-influenza A assay, as well as specific H5 and H7 assays. Further subtype-specific assays may also be run, if required. Any positive isolates are further characterised by culture in eggs and further molecular analysis. Analysis of viral genetic sequence data allows assessment of pathogenicity (see 'Agent characterisation', below), as well as more detailed phylogenetic analysis.

Isolates obtained from egg culture are identified antigenically by HI, as well as with molecular tools.

CSIRO-ACDP tests

The testing method used by CSIRO-ACDP is shown in Figure 2.1. Further details of tests currently available at CSIRO-ACDP, some of which are supported through the Laboratories for Emergency Animal Disease Diagnosis and Response (LEADDR) network, are shown in Table 2.1.

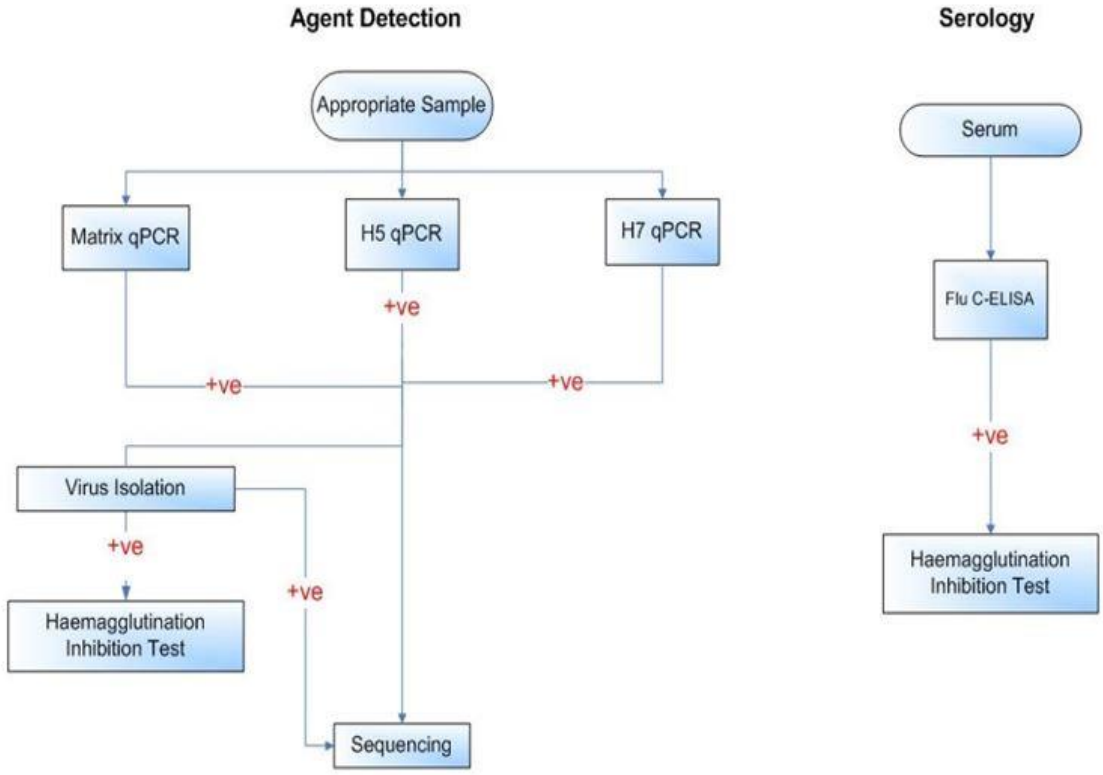

Figure 2.1 The current approach to diagnostic testing at CSIRO-ACDP

Table 2.1 Laboratory tests currently available at CSIRO-ACDP for the diagnosis of avian influenza

Test	Specimen required	Test detects	Time taken to obtain result
Agent detection			
Real-time PCR[a]	Swabs, tissues	Type A influenza, H5 and H7 subtypes, some LPAI subtypes	<1 day
Immunohistochemistry	Formalin-fixed tissues	Viral antigen	2 days
Virus isolation in embryonated eggs	Swabs, tissues	Virus	2–10 days
Agent characterisation			
PCR and sequencing	Swabs, tissues or virus isolate	Viral RNA	2–3 days
Antigenic subtyping (HI)	Virus isolate	Specific HA and NA antigens	1–4 days
Intravenous pathogenicity index	Virus isolate	Virulence of virus	2–10 days
Serology			
ELISA[a]	Serum	Antibody (influenza A)	1 day
HI	Serum	Antibody (specific HA types)	1 day

ELISA = enzyme-linked immunosorbent assay; HA = haemagglutinin; HI = haemagglutination inhibition; LPAI = low pathogenicity avian influenza; NA = neuraminidase; PCR = polymerase chain reaction

a Test also supported as part of the LEADDR network

Source: Information provided by the then CSIRO-AAHL, 2013 (refer to CSIRO-ACDP for most up-to-date information)

Other tests

Agent characterisation: tests for virus subtype

AI viruses are subtyped on the basis of the sequence of the HA and NA genes, as well as their HA and NA antigens. Details of tests for subtyping (PCR and sequencing, and antigenic subtyping) are in Table 2.1.

Agent characterisation

Tests for pathogenicity

The pathogenicity of an influenza virus isolated from a bird can be determined by one or more of the following tests (Swayne & Suarez 2000):

- Molecular pathotyping – sequencing the part of the gene encoding the cleavage site of the HA protein of the virus.

- Chicken pathogenicity test (intravenous pathogenicity index) – a solution of virus in sterile allantoic fluid is inoculated intravenously into eight 4–8-week-old specific pathogen–free chickens. If six or more chickens die within 10 days, the virus is considered to be highly pathogenic for chickens (HPAI virus).

For a timely diagnosis, molecular pathotyping is the preferred method of determining the pathogenicity of an AI virus in Australia. Once an outbreak virus has been characterised, virus detection and virus isolation can be used to confirm virulent infections.

Tests for previous infection

Evidence of previous AI virus infection can be obtained by testing for influenza A group-specific antibody using an ELISA, or by testing for subtype-specific antibody to the HA or NA antigens using an HI test or ELISA, respectively.

Use of serology for AI testing has several limitations:

- HI testing at a single point in time does not provide an indication of recent infection. Repeat blood samples collected 2–3 weeks apart may allow further interpretation of the HI results if a change in titre can be demonstrated.
- Positive serology results could indicate exposure to infection before the current outbreak situation.
- Positive serology results can indicate that the bird had exposure to an antigenically similar virus, and not the specific virus present in the outbreak situation.

Field tests

Currently, no field tests are approved for use during an EAD response involving AI in Australia.

2.6 Resistance and immunity

Host responses to AI virus may vary, mainly with the subtype and strain of the virus, and the species infected. Different subtypes and strains may stimulate different responses, and different species may develop different immune responses.

Innate immunity

Innate immunity is the first nonspecific line of defence against pathogens, comprising physical and chemical barriers to infection, and a range of antimicrobial mechanisms and factors at the cellular and humoral levels. Birds have well-developed innate immune systems. Chickens are generally less able to resist infection with HPAI virus than ducks, which are more efficient in clearing the virus through the capacity of their innate immune system (Barber et al 2010).

Acquired immunity

Maternal (passive) immunity (passed to progeny via the egg) has been demonstrated in both naturally infected chickens and vaccinated chickens. During natural infection, maternal antibody provided limited protection against HPAI (H5N1) virus (De Vriese et al 2010).

Ducklings are partially protected by maternal antibodies (IgY) passed through egg yolk. As in chickens, maternal antibody may interfere with development of immunity in vaccinated ducklings (Magor 2011).

Humoral immunity

Both infection with AI viruses and vaccination elicit a humoral antibody response at both systemic and mucosal levels. The mucosal antibody response (mainly IgA) has not been well characterised (Swayne & Halvorson 2008), but probably plays an important role in recovery from infection and protection from further infections, especially with LPAI virus.

The basis of protective humoral immunity is the development of neutralising antibody against the two major surface proteins, HA and NA (Swayne & Halvorson 2008). These antibodies do not cross-neutralise viruses of different HA or NA subtypes. The neutralising antibodies against the HA and the NA interfere with viral replication in different ways (Qiao et al 2003). In general, inactivated vaccines provide protection through humoral immunity.

Birds other than ducks (see below) that were infected with LPAI viruses were protected against challenge with virulent strains having similar surface antigens (Magor 2011).

In chickens, the IgM response is measurable as early as 5 days post-infection, and IgY can be detected shortly after this. Detectable antibody following infection with a virus of the same HA subtype persists for varying lengths of time, from 6 weeks to 12 months (Fichtner 2003, Pearson et al 2003). Antibody titres to different antigens in various bird species indicate that antibody production may be greatest for chickens, followed by (in decreasing order) pheasants, turkeys, quail and ducks (Suarez & Shultz-Cherry 2000).

In ducks, IgM is produced first (at 3–5 days), but is transient and quickly replaced by IgY (day 12) and IgA. IgA is an important mucosal surface secretory antibody in ducks, and bile IgA has virus-neutralising and HA-inhibition activity. Mucosal surfaces of the respiratory and intestinal tracts of ducklings are not protected by IgA during the first 2 weeks of life. IgY is the primary serum antibody in ducks.

Ducks were usually found to mount a poor HA antibody response to natural and experimental AI infections compared with chickens, based on HI testing. They may be reinfected with the same or similar influenza strains, and do not seem to mount a secondary immune response. Assays for measuring antibody response typically use secondary antibodies specific for chicken IgY; they are therefore highly species specific, and are less reliable for diagnosis in ducks, geese or other wild bird species (Magor 2011, Curran 2012).

Cellular immunity

Cellular immunity contributes minimally to protection against HPAI virus infection and disease in poultry, because mortality following HPAI is rapid, precluding the 1-week timeframe needed to induce a cytotoxic T-lymphocyte (CTL)–specific immune response (Swayne & Kapczynski 2008). However, some studies indicate that cellular immunity can limit the severity and duration of disease following HPAI virus infection in chickens and turkeys. Whereas inactivated vaccines do not stimulate memory T lymphocytes, live vaccines stimulate nucleoprotein-specific CTLs. This results in protection from lethal challenge, and allows more rapid clearance of virus and recovery following influenza virus challenge. There is also some evidence of cross-protection from cellular immunity following H9N2 infection against lethal H5N1 virus challenge (Seo & Webster 2001).

2.7 Vaccination

Effective vaccination reduces susceptibility to infection. When infection does occur, it reduces clinical signs of disease and the amount of virus shed into the environment. Under some circumstances, vaccination may therefore provide valuable assistance in eradication programs. Vaccination may be considered to assist in managing particular compartments of birds that are at risk of infection, such as captive endangered species.

Human and animal influenza virus vaccines continue to be developed and assessed, in response to overseas incidents of human infections with H5 and H7 AI virus, and human deaths associated with H5N1 and H7 viruses. Although new technologies will influence poultry vaccines in the future, the types of vaccine currently licensed by overseas authorities for use in poultry include:

- inactivated whole AI virus vaccines
- live genetically modified vaccines, including fowl pox–vectored and Newcastle disease virus–vectored vaccines
- inactivated genetically modified vaccines.

Many vaccines used recently around the world are inactivated whole AI virus antigen in an oil-based emulsion adjuvant, produced according to OIE standards. Inactivated vaccines are made by formalin treatment of infected allantoic fluids from chicken embryos to inactivate the agent(s), and adjuvanted by making a water-in-oil emulsion using mineral oil.

As of August 2020, one avian influenza (H5N2) vaccine is registered in Australia, and three other active constituents (H7N1, H5N9, H5N2) have been approved by the Australian Pesticides and Veterinary Medicines Authority (APVMA). If any of these vaccines or active constituents were to be considered for use, the APVMA would need to be consulted to determine the conditions and implications of use, as well as any other regulatory requirements.

Following vaccination of hens with inactivated vaccine (H5N1/H5N2), progeny demonstrated high to moderate levels of maternal antibody at 1–5 days of age and low levels by 7 days. Progeny of vaccinated hens that were vaccinated at 5 days of age had a poorer response to vaccination (and therefore increased susceptibility to AI virus infection) than those vaccinated at 10 days, because of interference from maternal antibody (Maas et al 2011).

Immunity to inactivated whole virus vaccines results primarily from response to the HA protein and, to a lesser degree, the NA protein. These vaccine technologies produce safe, pure and potent vaccines; commercial inactivated vaccines have been shown to protect against disease and prevent mortality, particularly if the antigen in the vaccine is closely matched with the field virus. However, they require handling and injection of individual birds. Another significant disadvantage is that they are not able to prevent virus shedding in chickens challenged with antigenically different viruses. This will support virus persistence in the field, demonstrating the need for regular review of vaccine viruses for their antigenic relatedness to field strains.

The inactivated vaccines use homologous HA determinants (eg H5) and either a homologous or heterologous NA determinant to provide protection against known current field strains of AI virus. The use of heterologous NA subtype vaccines provides an opportunity to use serological surveillance to detect circulating field virus by detecting antibodies to the NA subtype of the field virus – that is, a DIVA (differentiating infected from vaccinated animals) strategy. Vaccine technologies are being developed that will enable DIVA testing based on viral proteins other than the NA, removing the need to use heterologous NA subtypes for DIVA purposes.

Live fowl pox–vectored AI virus vaccines have the advantage that the subsequent immunity is not suppressed by maternal antibodies (Bublot et al 2006), and stimulate both cellular and humoral

antibody responses. This vaccine also has the advantage that it can be administered by injection at 1 day of age in the hatchery. A Newcastle disease virus–vectored AI vaccine has recently been developed that can be administered by spray in the hatchery.

Possible problems with the use of vaccine include the following:

- Inactivated vaccines have been associated with antigenic drift in field viruses.
- Vaccination may favour the emergence of more virulent variants through indirect selection of viruses.
- Vaccination could possibly mask low levels of circulating field virus by not fully preventing virus excretion and by masking clinical signs in susceptible animals.
- Vaccines may decrease in efficacy when used for a long period, as a result of antigenic drift in field viruses, and should be changed to match the predominant antigenic types.

The use of a vaccine will be under the control of CVOs in each state or territory.

2.8 Treatment of infected animals

Treatment of infected birds is not permitted because the policy is eradication of HPAI and LPAI (H5/H7). There is no effective treatment for AI virus infection, and prognosis for birds affected with virulent disease is poor. Those that survive are usually in poor condition and may continue to shed virus, thus posing a risk to other farms. Additionally, they may resume laying only after a period of several weeks, if at all.

Treatment for special or rare birds could be considered on a case-by-case basis if the biosecurity risks can be adequately managed.

3 Implications for Australia

3.1 Potential pathways of introduction

Avian influenza (AI) virus could be introduced into Australia through:

- transfer from asymptomatic waterfowl to susceptible flocks via close contact; this is the most likely pathway of introduction into Australian poultry
- migration of birds on established flyways
- contaminated poultry products, fomites or people.

3.2 Social, economic and environmental effects

One of the biggest impacts of an outbreak of highly pathogenic avian influenza (HPAI) or low pathogenicity avian influenza (LPAI) (H5/H7) involving the poultry industry would be on the domestic economy. Chicken meat and eggs are produced very efficiently in Australia, and provide the cheapest source of animal-based protein available to the population; this is reflected in the consumption rate. Loss of availability of these products would cause economic stress to the majority of the population and the domestic economy.

Domestic supply may be affected when the disease is detected; the effects may be more severe if the disease is widespread and movement restrictions impede the movement of product to markets. For affected producers, the main financial losses during an outbreak would be from flock mortalities, which can be high, and reduced productivity of affected flocks. A stamping-out policy would cause further loss of income to both affected properties and poultry companies for an extended period. Disruption to the flow of product, potential declines in human consumption of poultry and poultry products, and subsequent decreases in production may cause job losses on farms, and in service and associated industries (eg feedmills, transporters), depending on the time it takes to bring the outbreak under control. In large outbreaks, flow-on effects to whole communities can be expected to affect other industries, including tourism. Even a small outbreak would result in dislocation of the industry and its normal marketing patterns. Infection in grandparent and foundation flocks would cause loss of some valuable genetic material (see the **AUSVETPLAN enterprise manual *Poultry industry*** for information on the structure of the poultry industry).

Export markets for poultry products are likely to close immediately upon declaration of a HPAI or LPAI (H5/H7) outbreak. The extent of closure will depend on the market in question and the implementation of trade restrictions.

Zoos and premises holding cage birds, including pet shops and aviaries, may be affected directly by the outbreak, or indirectly through movement controls and restricted public access.

Strains of HPAI viruses circulating overseas have resulted in die-offs of waterbirds and deaths of multiple avian species, including birds of prey. During the 2016–17 epizootic of an HPAI (H5) virus (Guangdong lineage), 112 wild bird mass mortality events were reported from across Europe (Alarcon et al 2018). During the same epizootic, approximately 13 600 birds of 71 species were reported dead in the Netherlands ((Kleyheeg et al 2017). Between 2016 and 2018, marine birds made up the largest group of reported species mortalities during the HPAI (H5N8) epidemic in Africa (including South Africa); terns, particularly swift terns, were the worst affected systematic group, followed by the African penguin (*Spheniscus demersus*) (Khomenko et al 2018).

Sites of wild bird die-offs during HPAI epidemics have included wetlands of international importance (Ramsar sites) for waterbirds in west Africa, Europe and Asia.[20] Epidemics affecting wild bird species of high conservation status would have a negative impact on decades of conservation efforts; for example, species with small populations could be pushed closer to extinction. Beyond direct impacts on wildlife, shifts in public attitudes could lead to impacts on the Australian tourism industry.

3.3 Critical factors for an Australian response

- AI is a highly contagious infection of poultry and other birds. Clinical manifestations vary with the subtype and strain of the virus, and the avian species infected.
- All avian species appear to be susceptible to infection with AI viruses.
- Some overseas strains of HPAI viruses have been detected in apparently healthy wild birds, especially waterfowl, which suggests that these birds can spread the viruses. This has facilitated rapid intercontinental spread of these viruses through bird migration, though both mechanical and biological transmission.
- AI viruses are stable under a range of environmental conditions. As a result, it can be spread directly from flock to flock by fomites, dust, wind, or drinking water contaminated with infected faeces.
- The predominant means of spread of AI virus is through the movement of live birds, bird products (such as eggs), fomites, people and equipment.
- Environmental factors, such as time of year and weather patterns (eg high rainfall, drought), could be important. AI viruses are readily inactivated by heating, and remain viable for longer in cold and humid environments.
- Racing pigeons pose a unique risk because birds are congregated, transported and flown long distances, and may contact other domestic or wild birds en route.
- Zoo or rare bird collections may be susceptible to infection from both wild birds and commercial poultry operations (if any are located in the vicinity).
- Because of an increased likelihood of interaction between free-range commercial birds and wild birds or their faeces, there is an increased likelihood of AI in free-range enterprises.
- A single common source of infection (eg inappropriately treated water) may allow spread to multiple operations.
- Most commercial poultry farms in Australia have retention dams on the property, which encourage wild waterfowl.

[20] www.cms.int/sites/default/files/document/Scientific_Task_Force_AI_Wild_Birds_Statement_Feb2021_0.pdf

4 Policy and rationale

For international trade purposes, infection of poultry by any highly pathogenic avian influenza (HPAI) or low pathogenicity avian influenza (LPAI) viruses of the H5 and H7 subtypes is notifiable to the World Organisation for Animal Health (OIE). Avian influenza (AI) viruses in caged, zoo or wild birds do not fall into the OIE 'notifiable' AI categories.

The finding of any strain of HPAI or LPAI virus is notifiable to the chief veterinary officer (CVO) of the state or territory in which the finding is made.

The policy to be implemented for disease control will be informed by initial work on virus subtyping and pathogenicity completed under the Laboratories for Emergency Animal Disease Diagnosis and Response (LEADDR) program or through the CSIRO Australian Centre for Disease Preparedness (CSIRO-ACDP).

4.1 Introduction

4.1.1 Summary of policy

HPAI (any subtype) or LPAI (H5/H7) in poultry, or in cage or zoo birds

An outbreak of HPAI (any subtype) in poultry, or cage (aviary) or zoo birds in Australia is defined as infection with influenza A virus (with or without clinical signs) accompanied by confirmed laboratory diagnosis that meets the pathotyping criteria for HPAI virus.

An outbreak of LPAI (H5/H7) in poultry, or cage or zoo birds in Australia is defined as infection with influenza A virus (with or without clinical signs) accompanied by confirmed laboratory diagnosis that identifies an H5 or H7 subtype that meets the pathotyping criteria for LPAI virus.

The policy is to use stamping out to control spread of HPAI (any subtype) and LPAI (H5/H7), and to reduce the potential for mutation of LPAI (H5/H7) virus to HPAI virus. The response will depend on the assessed risk. A combination of strategies may be employed, including:

- *stamping out* by destruction, disposal and decontamination of all birds and contaminated avian products on infected premises (IPs) to remove the source of infection. Risk assessment of dangerous contact premises (DCPs) will identify premises that have a higher risk of being infected, and management will reflect the identified risk
- *modified stamping out* (see glossary) for LPAI (H5/H7) in cage or zoo birds, based on a risk assessment
- *biosecurity controls such as quarantine and the use of declared areas and movement controls over properties* – this includes quarantine of IPs, DCPs, suspect premises (SPs) and trace premises (TPs); declaration of restricted and control areas; and restriction on movements of birds, avian products and associated items in declared areas to prevent the spread of infection. A national livestock standstill is generally not considered necessary for containment of AI
- *tracing and surveillance* to determine the source and extent of infection, and to establish proof of freedom from the disease

- *flock or area depopulation* by pre-emptive slaughter, process slaughter or controlled marketing, depending on information derived from tracing, surveillance and the epidemiology of the outbreak (see also Section 4.3.1)
- *increased biosecurity* at poultry establishments (such as mandatory housing of free-range poultry), and premises holding cage or zoo birds
- *a public awareness campaign* to communicate risk and promote cooperation from industry, zoos, cage-bird owners and the community
- *protection of work health and safety, and public health*, in consultation with human health authorities – this will include a requirement that any personnel engaged in eradication activities take appropriate personal protective measures, including vaccination.

Vaccination may be considered if an outbreak of HPAI or LPAI (H5/H7) is likely to spread or has become widespread.

LPAI (not H5/H7) in poultry, or in cage or zoo birds

AI that is caused by a strain of virus that is not HPAI or LPAI (H5/H7) virus, and that is producing no or mild clinical disease, is not considered an immediate threat to Australia's domestic or zoo birds, or public health. Such AI virus strains are classified as LPAI (not H5/H7), and their detection in Australia would not be treated as an emergency animal disease (EAD) outbreak.

When the CVO determines that an infection is caused by such a virus, an epidemiological risk assessment will be carried out, taking into account the virus subtype, the species of bird involved, the clinical status of the birds, and their proximity to commercial or other significant bird establishments and populations, and to public amenity areas. No action will be required unless the risk assessment indicates an unacceptable threat to animal or public health. When a response is necessary, it may include:

- *tracing and surveillance* to determine the spread of infection
- *increased biosecurity*
- *protection of public health*, in consultation with human health authorities
- *an industry-arranged and managed control program*.

HPAI or LPAI in wild birds

AI infections classified as HPAI or LPAI virus in wild birds (free-flying birds not under any control or ownership) are not considered to pose an immediate threat to Australia's domestic or zoo birds, or to public health. Their detection would therefore not be treated as an EAD for the purposes of AUSVETPLAN.

In response to a finding of HPAI virus infection in wild birds, the Consultative Committee on Emergency Animal Diseases (CCEAD) will be convened, and an epidemiological risk assessment of the situation will be conducted. The assessment will take into account the virus subtype, the possible source of infection, the species of birds affected, the clinical status of the birds, and their proximity to commercial birds and public amenity areas.

No action will be required unless the risk assessment indicates an unacceptable threat to animal or public health. When a response is necessary, it will be in line with the level of assessed risk and may include:

- *declaration of restricted areas*
- *surveillance* to determine the extent of infection
- *enhanced biosecurity*
- *a public awareness campaign* to communicate risk and promote cooperation from industry, zoos, cage-bird owners and the community

- *protection of public health* in consultation with human health authorities.

The response policy for the detection of HPAI virus in wild birds is detailed in Appendix 3.

If only LPAI virus infection is detected in wild birds, no further action is required. However, consideration may be given to increasing surveillance and biosecurity in commercial poultry, zoo birds, and wild birds in the immediate vicinity of the wild bird LPAI virus detection to monitor disease spread.

4.1.2 Case definition

For the purpose of this manual, a case of AI is defined as laboratory-confirmed infection with AI virus in a susceptible animal with or without clinical signs.

Notes:

- Positive serology in the absence of detection of AI virus, with no clinical or epidemiological evidence supporting infection, does not constitute a definition of a case.
- AUSVETPLAN case definitions guide when a response to an EAD incident should be undertaken. AUSVETPLAN case definitions do not determine when international reporting of an EAD incident is required.
- At the time of an outbreak, revised or subsequent case definitions may be developed with the agreement of the CCEAD.

4.1.3 Cost-sharing arrangement

In Australia, HPAI caused by virus of subtypes H5 or H7 is included as a Category 2 EAD in the Government and Livestock Industry Cost Sharing Deed in Respect of Emergency Animal Disease Responses (EAD Response Agreement – EADRA). HPAI caused by a virus of subtype not H5 or H7 and LPAI caused by a virus of subtype H5 or H7 (LPAI (H5/H7)) are included in the EADRA as Category 3 diseases.

This manual addresses the detection of active or recent infection with HPAI virus in wild, cage or zoo birds, and the detection of active infection with LPAI (H5/H7) virus in cage or zoo birds. The manual also contains a response policy for detections of LPAI (not H5/H7) – that is, AI caused by subtypes of AI virus other than H5 and H7, and not classified as HPAI. However, responses to these types of detection are not covered by the EADRA.

4.1.4 Criteria for proof of freedom

The OIE *Terrestrial Animal Health Code*[21]

recommends that the status of a country, zone or compartment for AI be determined on the basis of criteria that include the following:

- AI is notifiable in the whole country, and all notified suspect occurrences of AI are subjected to field and, where applicable, laboratory investigations.
- Appropriate surveillance is in place.
- All epidemiological factors are considered.

Freedom from infection with HPAI virus and LPAI (H5/H7) virus can be regained according to the relevant article of the Terrestrial Code. Proof of freedom from infection in a declared area can be established by passive surveillance and active surveillance (using both targeted and random sampling) to determine the time that has elapsed since the area's last reported case, and the resolution of all declared premises. Further evidence of freedom is provided by continued passive surveillance (investigation of all suspect clinical cases, with negative results) in both previously infected and uninfected areas.

Importing countries may be prepared to accept variations from the OIE criteria and allow imports of Australian live birds, hatching eggs and avian products, as part of normal bilateral agreements, on a case-by-case basis.

For commercial poultry, typically Australia will not undertake proof-of-freedom activities for LPAI (not H5/H7) because the disease is endemic in wild bird reservoirs. Proof-of-freedom activities will be conducted for HPAI and LPAI (H5/H7).

Section 7 provides details on the procedures for surveillance and proof of freedom.

4.1.5 Governance

Governance arrangements for the response to EADs are outlined in the **AUSVETPLAN *Overview***.

Information on the responsibilities of a state coordination centre and local control centre is available in the **AUSVETPLAN management manual *Control centres management* (Parts 1 and 2)**.

Disease-specific governance issues

In some cases (eg LPAI (not H5/H7) in poultry, and zoo and cage birds; HPAI or LPAI in wild birds), government regulatory action may not be required, and a decision may be made to turn the response to industry or to take no further action.

[21] Because of ongoing changes to the Terrestrial Code (www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.10.4.htm), readers are referred to relevant sections of the code for current requirements for proof of freedom

4.2 Public health implications

All AI viruses have the potential to infect people. All outbreaks of AI in birds in Australia, regardless of pathogenicity, should be reported to the relevant state or territory health agency to enable assessment and advice regarding the mitigation of human health risks.

Appropriate work health and safety (WHS) measures, in line with the known or theoretical risk, should be implemented, with advice from human health authorities. Recent human infections with LPAI (H7N9) virus in China, some resulting in death, emphasise the need to remain vigilant in applying WHS measures.

Workers must be protected from infection with AI viruses wherever they have contact with infected poultry, products and premises.[22] Personnel involved in eradication activities will require appropriate training and supervision to ensure that all activities are managed appropriately with regard to WHS. They should be vaccinated with the currently available seasonal influenza virus vaccine, and protected from infection by wearing appropriate personal protective equipment (PPE), in accordance with national guidelines.

Personnel showing symptoms consistent with influenza virus infection must not come into contact with birds. Personnel may also be asked to take part in monitoring by state or territory public health departments. Those who do not agree to such measures should not be engaged in activities in which they could come into contact with infected birds.

4.3 Control and eradication policy

Eradication of outbreaks of HPAI or LPAI (H5/H7) in poultry (or in cage or zoo birds)

The default policy for an outbreak of HPAI or LPAI (H5/H7) in poultry, or in cage or zoo birds is to contain and eradicate the disease in the shortest possible time, without the use of vaccination.

Eradication will be achieved by stamping out, quarantine and movement controls, decontamination of infectious material on IPs and DCPs, targeted tracing and surveillance, and increased biosecurity by all levels of the poultry production and processing industries, and by zoos and cage-bird owners. Ongoing surveillance for AI virus in at-risk premises will give confidence that infection has been contained and is not establishing widely.

Detection in Australian poultry flocks, or in cage or zoo birds, of viruses that are classified under the EADRA as LPAI (H5/H7) virus will pose special challenges because the viruses may produce no, or mild, clinical signs of infection. The policy is to limit the spread of the infection and the potential for mutation of the virus to HPAI virus; the response will depend on the assessed risk. A plan based on stamping out or a modified stamping-out approach may be used. Since disease might be difficult to detect, extended surveillance strategies may need to be developed.

If vaccination is considered for control purposes, it will be supported by intensive industry liaison across all poultry, cage bird and zoo bird sectors, and public awareness programs. Public awareness programs will aim to:

- maximise reporting of suspect cases by veterinarians, owners of poultry and other birds, and managers of premises containing birds
- gain community cooperation

[22] www.who.int/influenza/human_animal_interface/en

- build confidence in disease control measures.

The control options for various AI scenarios are summarised in Table 4.1. The rationale for the policy is described in more detail in the remainder of this section.

Table 4.1 Control measures to be used for outbreaks of HPAI and LPAI

	Disease subtype				
	HPAI	**LPAI (H5/H7) in poultry**	**LPAI (H5/H7) in zoo or cage birds**	**LPAI (not H5/H7) in poultry, or zoo or cage birds**	**HPAI or LPAI in wild birds**
Control measure					
Stamping out of infected birds	Yes	Yes[a]	Yes[a,b]	+/–	No
Biosecurity controls	Yes	Yes	Yes	+/–[c]	No
Declared areas	Yes	Yes	+/–	No	No
Movement controls	Yes	Yes	Yes	+/–[b]	No
Tracing and surveillance	Yes	Yes	Yes	+/–	Surveillance only. Tracing not possible
Vaccination[d]	+/–	+/–	+/–	+/-	No
Area depopulation	+/-	+/–	No	No	No
Pre-emptive slaughter	+/–	+/–	No	+/–	No
Decontamination	Yes	Yes	Yes	+/–	No
Increased biosecurity[f]	Yes	Yes	Yes	Yes	Yes[f]
Industry or company-arranged control program[e]	No	No	+/–	+/–	No
Public awareness campaign	Yes	Yes	+/–	+/–	+/–

+/– = control measure may or may not be used; HPAI = highly pathogenic avian influenza; LPAI = low pathogenicity avian influenza

a Exceptions exist for special and rare birds (see Section 4.4).

b May be imposed by industry, jurisdiction or property owner; may be recommended by human health authorities.

c Depends on risk assessment.

d Depends on limited resources, including vaccine.

e Government may support this measure.

f Biosecurity controls should be in place on an ongoing basis because wild birds are reservoir hosts for AI viruses in Australia.

Stamping out

Stamping out involves the destruction of all susceptible birds on an IP when a risk assessment indicates that it is appropriate to control the spread of disease. It is the preferred control measure for HPAI and LPAI (H5/H7) unless the spread or likely spread of infection indicates that stamping out alone will not achieve eradication. Destruction of birds infected with HPAI or LPAI (H5/H7) virus and in-contact birds should be completed as rapidly as possible to reduce:

- shedding of the virus and spread of disease
- the potential for mutation of LPAI (H5/H7) viruses to HPAI viruses.

Destruction of infected and suspect infected birds also reduces the likelihood of emergence of new viral subtypes that may sustain person-to-person transmission.

All susceptible birds on HPAI IPs will be subject to stamping out. Poultry infected with HPAI or LPAI (H5/H7) virus will not be allowed to move for process slaughter.

Modified stamping out (see glossary) may be used during an outbreak when a risk assessment indicates that it is appropriate. This may include a combination of stamping out, controlled marketing and process slaughter of birds that are not infected but are at risk (eg additional sheds around an infected shed), provided that epidemiological assessment indicates that the birds pose limited risk of disease spread and that biosecurity controls can be imposed. Exceptions to stamping out will be assessed on a case-by-case basis, including consideration of rare or valuable birds.

Birds on other high-risk premises (SPs, TPs and DCPs) may be subject to stamping out. Such premises include premises that are adjacent to an IP, have common ownership or management with an IP, share equipment or staff with an IP, or are serviced by the same company as an IP. Decisions on the destruction of birds will be based on the measures used to control the outbreak and the epidemiology of the outbreak.

Pre-emptive destruction (see glossary) of birds on premises that may or may not be infected will be considered. These properties could be contiguous with the IP and in close proximity, or all designated properties within a defined area or buffer. Pre-emptive destruction may also be necessary for welfare reasons. Decisions on pre-emptive destruction will depend on the measures used to control the incident, the success of other activities and the epidemiology of the incident.

Process slaughter (see glossary) may be applied to premises of relevance (PORs) in the control area (CA) or at-risk premises (ARPs) in the restricted area (RA), subject to risk assessment and/or under circumstances recommended by the CCEAD. This might be appropriate where large numbers of poultry need to be disposed of quickly and the product is deemed fit for human consumption or is treated. Process slaughter enables depopulation through marketing of cooked (or otherwise heat-treated) meat and may limit the economic impact of the outbreak.

All process slaughter activities require negative laboratory test results (serology and polymerase chain reaction – PCR) from a representative sample of the population(s) to be depopulated, and application of appropriate biosecurity controls.

Where stamping out cannot be achieved rapidly, supporting disease control measures such as vaccination should be considered as part of a comprehensive program to reduce risk. Consideration may be given to orderly controlled marketing of affected flocks by allowing flocks to remain alive on-property until the infection has run its course or burnt out, birds have recovered, and virus shedding has stopped. This option may be effective if the affected property is large and isolated. Movements will need to be considered, especially movements of people, vehicles and equipment that are necessary to retain the flock.

In an explosive outbreak situation, area depopulation will be considered, based on identification of premises at risk. In this situation, depopulation of flocks at risk provides a buffer to prevent disease spreading to more distant susceptible populations.

4.3.1 Epidemiological assessment

Epidemiological investigation or assessment draws on multiple sources of information to build understanding of the disease and how it is behaving in an outbreak. This helps inform response decision making.

The key objectives for an epidemiological assessment will be to identify:

- the spatial distribution of infected and free animal populations
- potential vectors involved, including as potential amplifying hosts
- the source of infection
- the prevalence of infection
- pathways of spread and the likely size of the outbreak
- risk factors for the presence of infection and susceptibility to disease (including weather and insect populations).

Epidemiological assessment, and tracing and surveillance activities (see Section 4.3.3) in an EAD response are interrelated activities. Early findings from tracing and surveillance will be inputs into the initial epidemiological assessment (eg considering spatial distribution of infection). The outcomes of the initial epidemiological assessment will then guide decisions on subsequent tracing and surveillance priorities.

The outcomes of the epidemiological assessment will also be used initially to determine the feasibility of eradication versus long-term control and to guide the selection of other appropriate response measures (including the application of movement controls) and assess the progress of disease control measures.

Ongoing epidemiological assessment is important for any EAD response to aid evaluation of the continued effectiveness and value of response measures, and assessment of the progress of disease control measures. Ongoing epidemiological assessment will consider the outcomes of tracing and surveillance activities, and will contribute evidence to support any later claims of disease freedom.

4.3.2 Quarantine and movement controls

See Section 5 for details on declared premises and areas, and Section 6 for recommended quarantine and movement controls.

Quarantine

Quarantine will be immediately imposed on all premises and areas on which infection is either known or suspected.

Premises will be declared (see Section 5.1). An RA and CA will be declared around the IPs.

Movement controls

Movement controls are best implemented through the declaration of declared areas and linking permitted movements to each area. As a general principle, the aim of movement controls is to reduce the spread of disease by preventing the movement of infected animals, infected animal products and infected vectors (where relevant for the disease), and by allowing movements that pose a minimal risk.

Section 6.4 provides details on movement controls for live animals, reproductive material (semen and in vivo–derived embryos), animal products and byproducts, waste products and effluent, and other items that might be contaminated.

4.3.3 Tracing and surveillance

Tracing and surveillance will be conducted to determine the source and extent of infection, establish proof of freedom from the disease, and/or define zones and compartments for trade purposes.

Monitoring of human health, especially in people involved in on-property response operations, may be necessary and should be undertaken with jurisdictional human health authorities.

Tracing

The first reported case (index case) may not be the primary case for the outbreak. Tracing may assist in identifying earlier cases (and the estimated date of introduction), determining the source of infection (and the estimated onset of infectiousness) and determining the extent of spread. During a normal poultry production cycle, many routine movements occur to and from poultry premises. Tracing of these movements should be prioritised according to the highest-risk activities and the known epidemiological risk factors.

Tracing periods should take into account the OIE nominated incubation period of 21 days, the age of the flock at risk and the duration of the outbreak to date. They may need to be varied during the response according to the measures being employed.

States and territories will be responsible for tracing high-risk movements within their jurisdictions.

Tracing will be used to determine the movements of commodities and people associated with IPs, DCPs, SPs and TPs (see Section 5.3.1 for premises status classifications), including:

- live poultry
- poultry products – such as meat, eggs (fertile, table), semen, feathers and offal
- waste – such as dead birds, manure and used litter
- equipment – such as live bird transport vehicles, feed delivery vehicles, farm vehicles and equipment, contractor vehicles, catching equipment, dead bird pick-up equipment, veterinary equipment, workers' clothing, scales, loaders, crates, egg trays and fillers, pallets, trolleys, cages, and vaccination and debeaking equipment
- feed and fresh litter
- people – farm workers, employees, share farmers, poultry service workers, veterinary practitioners, tradespeople (eg electricians, plumbers), company personnel, sales representatives, vaccinators, pick-up crews, technicians, drivers (livestock, feed, egg, litter), contractors, livestock agents, relatives and other visitors.

As a first priority, movements to and from IPs and, where possible, DCPs should be traced for at least 21 days before the first observation of unusual morbidity or mortality. High-risk movements that need to be traced are those of birds, eggs, poultry products, manure, litter and waste. Movements of feed, equipment (including egg fillers, cages, trolleys and pallets) and people also need to be traced, and contact flocks assessed for infection for the 21 days before unusual morbidity or mortality was observed on the IP.

The original source of likely introduction of the virus to the birds should be traced, as it could remain a source of further virus dissemination.

Tracing of zoo birds and significant holdings of cage birds will also be required. If HPAI virus has been detected in wild birds, epidemiologically based investigations in the area in which the infected birds were found may be necessary.

Surveillance

Surveillance will be necessary to identify the source and foci of infection, determine the extent of spread and assess the impact of control activities. Surveillance will also be used to define declared areas and provide evidence for proof of freedom when the outbreak is contained. It will also be required if zones are to be established for trade.

Active surveillance should be initiated as soon as HPAI or LPAI (H5/H7) is suspected. Initially, the location of all commercial and backyard poultry, zoo birds and significant holdings of cage birds in the RA should be identified and mapped. A sample of birds of any domestic species that die in the RA should be investigated for AI, with specimens submitted to approved laboratories for diagnostic testing.

Intensive active surveillance aims to identify potential new cases of AI. Because of the risk of spread of virus by personnel, equipment and vehicles, the following measures could be adopted to enable continuing surveillance, while minimising multiple farm visits by inspectors and other authorised personnel to premises in the RA and CA:

- monitoring of dead bird pick-up and transport to a laboratory for suspicious cases
- reporting on flocks by, for example, telephone, fax, email or text messages
- telephone surveys.

Field surveillance visits can then be arranged to any potential new cases requiring investigation.

Sentinel animals may be used on decontaminated IPs as part of demonstrating proof of freedom (see also Section 4.3.14). More detailed protocols, including the frequency of sampling and numbers of birds to test under cage, barn and free-range situations, are detailed in Section 7.1.

Restricted area

As a priority, clinical investigation, and appropriate sampling and testing will be undertaken on all DCPs, SPs and TPs. Where findings from clinical examination and diagnostic testing are negative, telephone or field surveillance visits should be conducted on a regular basis (eg every other day) to confirm absence of disease. Dead bird pick-up and testing should be a component of monitoring.

Active surveillance will be conducted on the at-risk poultry population – commercial flocks, fancy or backyard flocks, zoo or cage birds, or all flocks – based on an epidemiologically sound risk assessment. This may include health monitoring by:

- twice-weekly (or more frequent, if needed) reporting by telephone, fax, email or text messaging by poultry owners and handlers, veterinarians, zoo personnel, waste removalists and dead bird pick-up personnel
- weekly tracheal and cloacal swabbing of dead birds for testing by PCR of the commercial flocks in the RA, based on a risk assessment of the epidemiological picture.

Passive surveillance will also be conducted to complement the active surveillance. This involves investigation of disease reports (ie SPs) from the public, poultry farmers, veterinarians, zoos or cage-bird owners. It includes the following:

- All reports of a decline in the health of birds (eg increase in mortality or morbidity consistent with clinical signs of AI; significant decline in feed or water consumption, or egg production) should be investigated.
- A sample of birds of any domestic avian species that die in the RA should be checked for gross AI lesions, and specimens should be submitted to approved laboratories for diagnostic testing. People coming into contact with potentially infected animals or contaminated materials should take appropriate precautions, including use of PPE.

Active and/or passive monitoring and surveillance of disease consistent with influenza in other susceptible species (eg feral and domestic pigs) should be considered, using a risk-based approach to determine possible transmission mechanisms and virus reservoirs that warrant control.

Control area

Flock health could be monitored by:

- follow-up of any unusual disease conditions, including in wild birds
- weekly laboratory testing (PCR and other tests, as appropriate) of meat chickens and commercial spent hens at abattoirs
- weekly telephone surveillance of owners, managers or veterinarians of susceptible flocks, including cage and zoo birds
- weekly reporting on flock health
- quarantine of suspicious flocks, with appropriate flock health testing and reporting of sick or dead birds.

Surveillance of wild birds to determine their potential involvement in dissemination of the disease may also be attempted. However, virus in wild birds is likely to have limited impact on the spread of the disease if biosecurity practices to limit access of wild birds to domestic and zoo birds, especially commercial poultry, are effective.

Monitoring will be needed to ensure the early detection of AI in mammalian species, especially pigs, dogs and cats.

Monitoring of human health, especially in people involved in on-property response operations, may be necessary. It should be undertaken with departmental health authorities.

4.3.4 Zoning and compartmentalisation for international trade

Where it is not possible to establish and maintain disease freedom for the entire country, establishing and maintaining disease-free subpopulations, through zoning and/or compartmentalisation,[23] may be considered.

In the case of a limited disease outbreak, a containment zone[24] may be established around the areas where the outbreak is occurring, with the purpose of maintaining the disease-free status of the rest of the country outside the containment zone.

All zoning applications would need to be prepared by the Australian Government in conjunction with the relevant jurisdiction(s) and agreed to by the CCEAD. Zoning is usually negotiated after a disease outbreak has begun.

Compartmentalisation applications typically need to be negotiated before an outbreak occurs, and will require input from the relevant industries.

Recognition of both zones and compartments must be negotiated between the Australian Government and individual overseas trading partners. Zoning and compartmentalisation would require considerable resources that could otherwise be used to control an outbreak. Careful consideration will need to be given to prioritising these activities, because the resulting competition for resources could delay the quick eradication of the disease and recognition of disease freedom.

Agreements between trading partners take time to develop, consider and finalise, because of the need to provide detailed information on activities such as biosecurity, surveillance, traceability and diagnostics to support the approach that is developed. An importing country will need assurance that its animal health status is not compromised if it imports from an established disease-free zone in Australia. Trading partners may not accept a zoning or compartmentalisation proposal, regardless of the information provided. Eradication of disease may be achieved before zoning or compartmentalisation applications are finalised.

The OIE guidelines for zoning and compartmentalisation are in Chapter 4.4 and Chapter 10.4 of the OIE *Terrestrial animal health code*.

4.3.5 Vaccination

Control of AI without the use of vaccination is the preferred policy. Vaccination has not been necessary in Australian outbreaks to date, but its usefulness has been demonstrated in overseas outbreaks (Capua et al 2003).

[23] With zoning, disease-free subpopulations are defined primarily on a geographical basis. With compartmentalisation, disease-free subpopulations are defined primarily by management practices (such as the biosecurity plan and surveillance practices of enterprises or groups of enterprises).

[24] The OIE defines a 'containment zone' as an infected zone within a previously free country or zone, which includes all suspected or confirmed cases that are epidemiologically linked and where movement control, biosecurity and sanitary measures are applied to prevent the spread of, and to eradicate, the infection or infestation. The Australian Government Department of Agriculture and Water Resources commissioned a report on what would be required for the establishment of containment zones in Australia. This report is available at www.ausvet.com.au/tools-resources.

4.3.6 Treatment of infected animals

Treatment of infected birds is not permitted because the policy is eradication. There is no effective treatment for AI, and prognosis for birds affected with virulent disease is poor. Those that survive are usually in poor condition and may continue to shed virus, thus posing a risk to other farms. Additionally, they may resume laying only after a period of several weeks, if at all.

Treatment for special or rare birds could be considered on a case-by-case basis if the biosecurity risks can be adequately managed.

4.3.7 Treatment of animal products and byproducts

All products from infected birds, and other items that contained, transported or came into contact with such products or byproducts should be considered to be contaminated with AI virus. People who come into contact with infected birds need to follow strict decontamination procedures if AI virus is to be contained to IPs and DCPs (see the **AUSVETPLAN operational manual *Decontamination*** and the nationally agreed standard operating procedure *Personal decontamination – entry and exit procedures*[25]). In addition, it needs to be ensured that products from other premises in the area of infection do not transfer AI virus before infection is diagnosed on those premises.

Treatment and disposal of manure and litter will depend on the premises and circumstances, and will require assessment of the most appropriate location and means of disposal. The **AUSVETPLAN operational manual *Decontamination*** must be consulted when deciding on the most appropriate means of decontamination.

4.3.8 Destruction of animals

Efficient, humane procedures must be used to kill infected birds, preferably without moving them from the site. Methods include neck dislocation, decapitation or lethal injection for individual birds; and use of carbon dioxide or foam for destruction of flocks in situ. Some methods, such as ventilation shutdown, have significant negative animal welfare impacts and are not generally supported. Destruction methods are described in the **AUSVETPLAN operational manual *Destruction of animals***, and a decision-making guide is available in the **AUSVETPLAN resource document *Methods for the destruction of poultry, pet/zoo birds and aviary species***. Political, social, operational, technical, animal welfare and financial factors must be considered in determining the most appropriate destruction method.

The most appropriate method will depend on the zoonotic potential of the virus, the species of bird, the premises type, the weather, the availability of trained personnel, the speed with which destruction is required and the available physical resources. Handling dead birds produces less airborne contamination than catching and handling live birds, reduces the exposure of workers to contamination and makes working in the recommended protective equipment more bearable. However, handling dead birds in cages while rigor mortis is present may create significant challenges in animal removal.

Airborne dispersal of virus should be minimised at all times by closing up bird houses, and shutting down fans or reducing their speed during depopulation. Depopulation activities should occur inside

[25] www.animalhealthaustralia.com.au/what-we-do/emergency-animal-disease/nationally-agreed-standard-operating-procedures

sheds as far as practicable. Infected or potentially infected birds should not be moved between sheds, or carried outside to skips or containers, unless these are the only methods available for depopulation.

Disinfection of the litter surface and containment of feathers, dander and so on will reduce the load of virus that can potentially be spread. Access of wild birds to premises containing domestic or zoo birds, especially commercial poultry, should be taken into account when deciding on the order in which to start depopulation operations.

4.3.9 Disposal of animals, and animal products and byproducts

One of the major objectives of the eradication program is prompt and effective disposal of contaminated material that cannot be effectively treated (eg dead birds, eggs, litter, manure, fresh or frozen carcases, plant and equipment, and building materials).

Disposal may be either on or off the IP or DCP. The best method should be determined by risk assessment, taking into account factors such as the proximity to appropriate disposal sites, the risk of virus spread via transport, and the impact on businesses and the community. Available methods of disposal are described in the **AUSVETPLAN operational manual *Disposal***. The most likely on-site disposal methods include composting and deep burial. Off-site disposal by burial at a common site, incineration, composting or rendering could be used if on-site disposal is not suitable or practical.

If infected material must be transported for disposal, particular attention should be paid to preventing the spread of the virus. For example, truck body trays must be leakproof, and all loads must be carefully covered to ensure that material cannot blow out.

Disposal of large numbers of birds in a short time presents environmental and logistical problems. A poultry shed full of meat birds close to market weight contains about 75–90 tonnes of organic material, of which 75% is water.

4.3.10 Decontamination

Decontamination entails cleaning and disinfection of the infected site to remove all infective material. The **AUSVETPLAN operational manual *Decontamination*** must be consulted when deciding on the most appropriate means of decontamination.

Susceptibility of virus to chemical and physical agents

The lipid in the AI virus envelope makes the virus highly susceptible to detergents. On clean surfaces, AI viruses are destroyed by sodium hypochlorite, quaternary ammonium compounds, sodium hydroxide, phenolic compounds, acidified ionophores, chlorine dioxide, strong oxidising agents and sodium carbonate/sodium silicate combinations. Organic material must be removed by dry or wet cleaning before disinfectants will work properly. Detergent, steam, alkalis and phenolic compounds can be used to remove organic material before the use of other treatments, such as hypochlorite, for virus inactivation. The virus is also readily inactivated by enzymes such as peptidases, neuraminidases and haemagglutinases (WHO 2007b).

Where biofilms are present, surfactants and sequestrants are used to ensure penetration and destruction of the virus by sanitisers. Any detergent used must not neutralise the sanitiser.

AI virus is relatively stable in faeces and litter; thus, all buildings, equipment, vehicles, manure and litter on IPs and DCPs must be cleaned and disinfected, or destroyed. People exiting IPs, DCPs, dangerous contact processing facilities (DCPFs), SPs and TPs should undergo personal

decontamination procedures. Ideally, organic matter removed from surfaces during cleaning should be contained and treated in a manner that will destroy the virus

Contaminated fomites, such as clothing, footwear, crates, feed sacks, egg fillers and other equipment, should be decontaminated, if possible, or destroyed. All items to be disinfected must be thoroughly cleaned before disinfection.

Decontamination should include standard insect vector and rodent control measures to minimise mechanical spread of the virus to nearby premises.

4.3.11 Wild animal management

Wild birds, particularly waterfowl, that visit premises holding birds may harbour and shed AI virus. They may introduce AI to an area and have been implicated as the source of AI outbreaks (see Section 2.3.2). There is a growing body of evidence that wild birds have an ongoing role in the spread of infection during an outbreak. They pose a serious risk of spread of AI virus between farms, particularly early in an outbreak before quarantine measures have been imposed, if they are not discouraged from production areas.

To minimise the risk of infection of commercial, cage or zoo birds from wild birds, high-level biosecurity must be practised. During eradication procedures, it is essential that quarantined and other bird houses, litter, waste, compost piles and contaminated sites are bird-proofed to prevent access by wild birds.

Steps should be taken to make IPs and DCPs less attractive to wild birds, including cleaning up feed spills. Netting or draining of dams are unlikely to be practicable, but are feasible under certain circumstances and should be evaluated as options. The use of drones and laser lighting has been demonstrated to discourage wild birds from landing and settling on range areas and water bodies.

Wild bird management must continue long after destruction, disposal and decontamination have been completed, and throughout targeted or passive surveillance and epidemiological investigations, because wild birds present a source of reinfection of a site, particularly if they have been implicated in the initial index case.

Scientifically, destruction of wild waterfowl is not supported. Experts do not recommend the lethal removal of wild birds to prevent the spread of LPAI or HPAI. Because of the high number and constant movement of wild birds, the use of lethal methods is neither practical nor environmentally sound (FAO & SPC nd).

4.3.12 Vector management

Rodents, wild and domestic animals (eg dogs and cats), and flying insects may act as mechanical vectors for AI virus. Appropriate biosecurity measures must be implemented to ensure that these potential vectors cannot access or remove material from bird areas or sheds, or composting or disposal sites.

Rodent and fly control measures, such as baiting, chemical control or vermin-proofing, should be implemented as practicable for each site.

For further information, see the **AUSVETPLAN operational manual *Wild animal response strategy***.

4.3.13 Public awareness and media

The *Biosecurity incident public information manual*[26] provides a guide for undertaking activities associated with public information management.

Details on enhancing farm biosecurity practices and practising good biosecurity are available at the Farm Biosecurity website,[27] and in the *National farm biosecurity mannual: poultry production*,[28] *National water biosecurity mannual: poultry production*,[29] *National farm biosecurity manual for chicken growers*[30] and *National farm biosecurity technical manual for egg production*.[31]

In addition to biosecurity messaging, a media campaign must emphasise the importance of poultry producers, bird owners and zoo personnel inspecting susceptible animals regularly, and reporting suspicious clinical signs and unusual deaths promptly.

Details of any imposed movement controls need to be made available and clearly explained.

Human health considerations need to be highlighted; people should be directed to human health authorities for targeted information on hygiene practices when dealing with poultry and poultry products.

Information must be provided to the public to address concerns about the safety of poultry products.

4.3.14 Other strategies

Restocking of flocks or areas should only be undertaken after a risk assessment and consideration of the epidemiological situation. No restocking should take place before the outbreak has been brought under control in the area where infection was widespread.

Before full repopulation, sentinel birds may be used to determine the effectiveness of decontamination measures. This approach will delay full repopulation, but has the advantage of lowering costs should a fully restocked premises become infected as a result of inadequate decontamination.

It is vital that sentinel birds have ample opportunity to be exposed to AI virus should it remain in the decontaminated area. In cage layer operations, this may require allowing access of sentinel birds to cages, the floor and manure collection areas. In free-range operations, access to all production and housing areas (eg laying areas, feeders, night housing) must be allowed.

When determining the time between decontamination and restocking of premises with sentinels or full repopulation, virus viability outside the host (see Section 2.4.2) should be considered. This will take into account factors influencing virus viability, including temperature, humidity, salinity, pH, surface type, ultraviolet light and chemical application. Economic and social factors associated with delayed recommencement of business operations should also be considered.

Historically, restocking of premises with sentinel birds or full repopulation has not been allowed until at least 21 days following cleaning and disinfection. The basis for this is unclear because limited information is available on virus viability on surfaces that are likely to be found in poultry sheds. However, viability of virus in dust in poultry houses has been reported for 2–5 weeks after

[26] www.animalhealthaustralia.com.au/our-publications/ausvetplan-manuals-and-documents
[27] www.farmbiosecurity.com.au
[28] www.agriculture.gov.au/pests-diseases-weeds/protect-animal-plant/bird-owners/poultry_biosecurity_manual
[29] www.agriculture.gov.au/pests-diseases-weeds/protect-animal-plant/bird-owners/water_biosecurity
[30] www.farmbiosecurity.com.au/industry/chickens
[31] www.farmbiosecurity.com.au/industry/eggs

depopulation (Webster et al 1978). Longer periods for restocking of IPs and DCPs may be appropriate, and may be informed through risk assessment.

Methods of lowering the risk of infection in restocked populations that have been used successfully in overseas outbreaks include an extended period (60 days) after cleaning and disinfection, with no sentinel birds; dead bird sampling of repopulated flocks; and the use of unvaccinated birds as sentinels in vaccinated flocks.

During high-risk periods, free-range and outdoor poultry may need to be housed indoors as an additional control tactic to limit virus spread, and to mitigate the risk presented by local wild birds.

Recommended species

Ideally, the sentinel bird species should be one that is highly susceptible to AI virus (especially to the outbreak strain). Chickens, turkeys and other gallinaceous poultry are highly susceptible to clinical infection and would be the first choice. However, where consideration is being given to partial restocking as part of the sentinel process, other species could be used.

Sampling and testing

Sentinel birds must be identifiable, free from infection and serologically negative before they are used. Appropriate testing methodology must be applied to the sentinel birds to ensure the validity of the results.

It is recommended that cloacal and tracheal swabs are collected 14 days after introduction. Serological sampling should be undertaken at 21 days after introduction. Daily health monitoring must be completed for as long as sentinel birds are in place.

Fate of sentinel birds

Sentinel birds may not need to be destroyed at the end of the sentinel period. Depending on the numbers involved, and the species and age of the birds, provided that they exhibit no clinical, serological or molecular evidence of AI infection, they may be reclassified from sentinel to commercial and become part of a staged repopulation.

4.3.15 Stand-down

The stand-down phase begins when:

- the investigation and alert phase fails to confirm the presence of AI
- HPAI or LPAI (H5/H7) has been eradicated, and proof-of-freedom activities have been completed
- eradication of AI is not considered feasible, cost-effective or beneficial, or
- the National Management Group (NMG) formally declares that the AI incident is over.

During the stand-down phase, the following activities take place:

- If the presence of AI is not confirmed in the initial investigation and alert phase, people and agencies contacted during this phase are notified that the disease has not been confirmed.
- If control and eradication measures have been undertaken, a senior operational manager ensures that a written plan is developed and implemented so that operations are wound up systematically.
- The state coordination centre and local control centre(s)
    - develop and implement an ongoing management program, if required

- recover, decommission and dispose of stores and equipment
- arrange appropriate archiving of all records
- finalise accounts
- conduct debriefings and record all learnings.

- The CCEAD (if established for the response) concludes its activities, arranges for a final report, provides its recommendations to the NMG (if established for the response) and stands down.
- The NMG (if established for the response) concludes its activities and stands down.
- Specific actions taken by the CCEAD and the NMG during the stand-down phase are described in the EADRA and supporting documentation.

If necessary, relief and recovery activity will continue after disease control and eradication operations have wound down.

4.4 Other control and eradication options

Exemptions for rare or valuable birds

When an IP (or a premises that is in direct contact or contiguous with an IP) or DCP (based on risk assessment) on which stamping out is to be applied contains rare or valuable poultry, or aviary or zoo birds, the primary objective remains eradication of AI virus. A modified approach may be appropriate in some situations, in which uninfected birds or flocks on these properties are exempt from destruction. This would only apply if the risks were assessed as acceptable, taking into account factors such as biosecurity, public health, potential exposure, movement controls, ongoing tracing and surveillance, and timeliness in achieving disease eradication. In all cases, owners of birds will be afforded natural justice in this decision making.

An exemption from stamping out may be considered for:

- rare breeds that are listed on the Rare Breeds Trust of Australia priority list, or other lists of similar or academic recognition
- birds with unique genetics – that is, genetically distinct birds, with a documented breeding history, that are in a viable reproducing population or part of a collective breeding situation (sufficient to maintain genetic variation). In some cases, individual birds may participate in geographically dispersed breeding programs, which may include some grandparent flocks. Sufficient proof would be required demonstrating genetic uniqueness
- endangered or rare species – for example, species listed by the Convention on International Trade in Endangered Species, or species on the International Union for Conservation of Nature Red List of Threatened Species
- irreplaceable birds (which cannot be replaced because of limited global availability or import restrictions)
- birds with specialised skills or attributes (eg animal actors, airport bird control raptors, public performance birds)
- pet birds.

Subject to a risk assessment and in consultation with human health authorities, rare or valuable birds on LPAI (H5/H7) IPs (or premises that are in direct contact or contiguous with an LPAI (H5/H7) IP) or LPAI (H5/H7) DCPs may be exempt from stamping out if they:

- are showing no clinical signs of AI virus infection (LPAI H5/H7) and are tested as free from active infection, where practical (testing may not be practical for some zoo birds)

- are placed under quarantine
- are subject to surveillance; such surveillance, if part of the eradication campaign, would be subject to cost-sharing arrangements
- are covered by an approved biosecurity plan developed by the owner or veterinarian, in consultation with jurisdictional (including health) authorities
- are subject to an acknowledgment from those looking after the birds that they are aware of the risks to human health, and agree to use PPE when handling the birds or working in their enclosures, and avoid close contact with the birds
- do not pose a risk to commercial enterprises or other susceptible animal populations.

Vaccination for such birds may be considered where a case can be made for protection of high-value birds at risk of infection. The use of AI vaccines in Australia is only permitted in accordance with a decision by the CCEAD, with the priorities being to stop spread of the outbreak and to protect rare, endangered and valuable birds (targeted vaccination). Vaccination will only be approved if the strain of AI virus subject to the emergency response is present in Australian wild bird populations. Identification of individual birds may be necessary for smallholdings and zoos.

Given the value of some zoo birds, AI vaccination may be an appropriate preventive measure, although it may not increase their protection if they are already housed in biosecure units. An application will need to be made for non-emergency vaccination, declaring that the flock or bird is of exceptional value, and that certain conditions and expenses can be met.

In the event of a diagnosed or reported outbreak of LPAI (not H5/H7) in a rare or valuable bird flock, management of the flock should be subject to risk assessment, including the risk to public health, involving human health authorities.

No exemption will be considered for specific pathogen–free (SPF) flocks or grandparent flocks in IPs where depopulation is the nominated disease strategy. However, where an SPF flock or grandparent flock is present on a DCP, a risk assessment should be undertaken to determine the likelihood of infection. DCPs containing birds that are highly likely to be excreting virus currently or in the immediate future should be slaughtered-out before the flocks excrete virus; however, further monitoring and investigation may be considered for DCPs containing birds that are less likely to be excreting virus currently or in the immediate future (eg earlier in the incubation period). This approach will limit the impact of disease control measures on the industry, while reducing the costs associated with management of the disease.

Strategy if the disease becomes established

If HPAI or LPAI (H5/H7) were to become established in commercial poultry populations, properly applied biosecurity measures and widespread vaccination, with an appropriate vaccine, could limit transmission of infection. A risk-based control program, involving stamping out of infected flocks and some or all of the following activities (depending on the epidemiology of the outbreak), may be applied:

- consultation with industry
- public education about the disease, the control program and the need to maintain good records
- surveillance for the presence of infection in populations at risk, including meat chickens, layers, cage birds and zoo birds
- tracing to identify the source and/or spread of the virus
- reporting of suspect flocks, and their quarantine until they can be confirmed negative or be destroyed
- upgrading of hygiene and other biosecurity procedures

- preventing infection through programs to encourage isolation and bird-proofing of premises, exclusion of wildlife and rodents, and treatment of drinking water
- effective vaccination of all breeder and layer poultry flocks in the infected area, and of other birds considered to be at significant risk, including rare and valuable birds
- public–private sector cooperation to improve future control strategies.

Although a vaccination policy would assist the poultry industry economically, it may have implications for Australia's trading status, and the CCEAD would need to develop strategies to re-establish Australia's status of freedom from HPAI virus infection.

4.5 Funding and compensation

Details of the cost-sharing arrangements can be found in the Government and Livestock Industry Cost Sharing Deed in Respect of Emergency Animal Disease Responses.[32] Details of the approach to the valuation of, and compensation for, livestock and property in disease responses can be found in the **AUSVETPLAN operational manual *Valuation and compensation***.

[32] www.animalhealthaustralia.com.au/what-we-do/emergency-animal-disease/ead-response-agreement

5 Declared areas and premises

When an emergency animal disease (EAD) is first suspected, the premises involved would undergo a clinical and/or epidemiological investigation. If the case definition, as defined in the relevant AUSVETPLAN response strategy, is met (ie the index case[33]), the relevant chief veterinary officer (CVO) or their delegate will determine the premises classification and may declare the premises an infected premises (IP).

After the identification of the first IP, a restricted area (RA) and a control area (CA) may be declared.[34] A transmission area (TA) may also be defined, if appropriate. All premises within these areas will be classified.

At the beginning of an EAD incident, the initial premises classifications would be IP, at-risk premises (ARP), premises of relevance (POR), unknown status premises (UP) and zero susceptible species premises (ZP).

Any premises within the RA or CA will have only one classification at any one time. After an epidemiological investigation, clinical assessment, risk assessment or completion of control measures, a premises may be reclassified.

Once the first IP has been identified, intelligence gathering through veterinary epidemiological investigations would quickly lead to the identification of suspect premises (SPs) and trace premises (TPs). These would be high priorities for follow-up investigation by the relevant state or territory authorities. In a worst-case scenario, an SP could become an IP; therefore, SPs need to be investigated as a matter of very high priority. Similarly, investigation and risk assessment of a TP might identify it as an IP, dangerous contact premises (DCP) or dangerous contact processing facility (DCPF). An SP or TP might also be assessed as negative and qualified as SP-AN or TP-AN, and eventually reclassified as an ARP, POR or ZP.

All premises classifications are subject to change as a result of a modification in the case definition(s) or investigation(s) as the incident response proceeds.

Classifications should be applied with information needs of managers in mind. They should assist managers to monitor and report progress. Premises classifications to be used should be agreed early in a response, so that control centre personnel can apply the correct and consistent classifications and definitions from the outset of the investigation and response.

[33] The first case to come to the attention of investigators

[34] This is invariably the case with highly contagious diseases (eg foot-and-mouth disease, equine/avian/swine influenza, classical swine fever) but may not apply to less contagious diseases (eg Hendra virus, anthrax, Australian bat lyssavirus).

5.1 Declared areas

Maintaining movement restrictions on areas for long periods has important implications for resource management, animal welfare, business continuity, and socioeconomic impacts on producers and regional communities.

During the course of an EAD response, it may become necessary for a CA or RA to be expanded, as additional geographical areas or new foci of infection are identified. Later in the response, as control is achieved, mechanisms for gradually reducing the size of the CA and RA can be introduced.

An EAD may involve multiple foci of infection, with several jurisdictions potentially involved. Since disease might be controlled at different rates in different areas, there may be the opportunity to progressively lift restrictions on an area basis. This would involve reclassifying previously declared areas (RAs and CAs), with a staged approach to lifting of movement restrictions. This is a key step in the recovery process and will have positive benefits on the community.

5.1.1 Restricted area (RA)

An RA is a relatively small legally declared area around IPs and DCPs that is subject to disease controls, including intense surveillance and movement controls.

An RA will be a relatively small declared area[35] (compared with a CA – see below) drawn with at least 1–5 km radius around all IPs and DCPs, and including as many SPs, TPs and DCPFs as practicable. Based on risk assessment, the RA is subject to intense surveillance and movement controls, and other relevant disease controls. The purpose of the RA is to minimise the spread of the EAD. The RA does not need to be circular but can have an irregular perimeter, provided that the boundary is initially an appropriate distance from the nearest IP, DCP, DCPF, SP or TP. Multiple RAs may exist within one CA.

The boundaries will be modified as new information becomes available, including from an official surveillance program. The actual distance in any one direction will be determined by factors such as terrain, the pattern of livestock movements, livestock concentrations, the weather (including prevailing winds), the distribution and movements of relevant wild (including feral) animals, and known characteristics of the disease agent. In practice, major geographic features and landmarks, such as rivers, mountains, highways and roads, are frequently used to demarcate the boundaries of the RA. Although it would be convenient to declare the RA on the basis of local government areas, this may not be practical, as such areas can be larger than the particular circumstances require.

5.1.2 Control area (CA)

A CA is a disease-free buffer between the RA and the OA (see below). Specific movement controls, surveillance strategies, and other relevant disease controls will be applied within the CA to maintain its disease-free status and prevent spread of the disease into the OA.

An additional purpose of the CA is to control movement of susceptible livestock for as long as is necessary to complete tracing and epidemiological studies, to identify risk factors and forward and backward risk(s).

The CA will be a larger declared area around the RA(s) – initially, possibly as large as the state or territory in which the incident occurs – where restrictions will reduce the risk of disease spreading

[35] As defined under relevant jurisdictional legislation

from the RA(s). The CA will have a minimum radius of 2–10 km, encompassing the RA(s). The actual distance in any one direction will be determined by factors such as terrain, the pattern of livestock movements, livestock concentrations, the weather (including prevailing winds), the distribution and movements of relevant wild (including feral) animals, and known characteristics of the disease agent. In practice, major geographic features and landmarks, such as rivers, mountains, highways and roads, are frequently used to demarcate the boundaries of the CA. The boundary will be adjusted as confidence about the extent and distribution of the incident increases.

In general, surveillance and movement controls will be less intense in the CA than in the RA, and disease-susceptible animals and their products may be more likely to be permitted to move under permit within and from the area than those originating from the RA.

5.2 Other areas

It is possible that other types of areas (eg vaccination area or surveillance area), which are not legally declared, may be used for disease control purposes in some jurisdictions.

5.3 Premises classifications

Detailed guidelines for classifying premises statuses are provided in the **AUSVETPLAN guidance document *Declared areas and application of premises classifications in an EAD response***, and the definitions are in the Glossary.

5.3.1 Premises status classifications

For avian influenza (AI), the premises classifications to be used are:

- infected premises (IP)
- suspect premises (SP)
- trace premises (TP)
- dangerous contact premises (DCP)
- dangerous contact processing facility (DCPF)
- approved processing facility (APF)
- approved disposal site (ADS)
- at-risk premises (ARP)
- premises of relevance (POR)
- resolved premises (RP)
- unknown status premises (UP)
- zero susceptible species premises (ZP).

5.3.2 Qualifiers

Please also refer to the **AUSVETPLAN guidance document *Declared areas and premises classifications*** for more detail on qualifiers.

For avian influenza (AI), the qualifiers to be used are:

- assessed negative (AN)
- sentinels on site (SN)
- vaccinated (VN).

5.4 Reclassifying premises and previously declared areas

5.4.1 Reclassifying previously declared areas

The lifting of restrictions in declared areas is managed by jurisdictions according to their local legislation, regulations and processes.

The key principles for reclassifying a previously declared area during a response should include the following, noting that not all will be relevant for some diseases:

- The area should be epidemiologically distinct from other declared areas.
- All TPs and SPs have been investigated and reclassified, and all IPs, DCPs and DCPFs in the area have been reclassified as RPs (or APFs).
- All tracing and surveillance associated with EAD control has been completed satisfactorily, with no evidence or suspicion of infection in the area.
- A minimum period of 42 days[36] has elapsed since predetermined disease control activities and risk assessment were completed on the last IP or DCP in the area or a risk assessment supports reclassification.
- An approved surveillance program (including the use of sentinel animals, if appropriate) has confirmed no evidence of infection in the RA (see below).
- For vector-borne diseases, vector monitoring and absence of transmission studies indicate that vectors are not active.

Lifting of restrictions is a process managed by the relevant CVO under jurisdictional legislation and consistent with the most current agreed Emergency Animal Disease Response Plan (EADRP). When the appropriate conditions are satisfied, an affected jurisdiction can, in consultation with the Consultative Committee on Emergency Animal Diseases (CCEAD), reduce the size of either or both the CA and RA or lift all restrictions as surveillance/monitoring indicates change in risk. The previous part of the RA would then become part of the CA. Jurisdictions should be able to present documented evidence that the appropriate conditions have been met.

When an RA is lifted and becomes part of the CA, it will have a lower risk status, and the movement restrictions that apply will be consistent with those applying within the CA. Over time, all of the RAs will be reduced and lifted.

[36] The minimum period uses, or is based on, the disease-specific incubation periods defined by the OIE – two incubation periods is a common guideline.

If more than one jurisdiction is affected, each will use its own appropriate legal jurisdictional mechanisms to lift the declaration of the RA or CA, coordinating with each other and consulting with the CCEAD to ensure wide communication and coordination.

After a further period of surveillance and monitoring, and provided that the additional surveillance and monitoring find no evidence of infection, a jurisdiction, in consultation with the CCEAD, could lift the CA. This would result in the lifting of all the remaining regulatory controls associated with the response, and a return to business as usual.

6 Movement controls

6.1 Principles

The principles for the recommended quarantine practices and movement controls are as follows:

- Containment and eradication of Avian influenza (AI) is the highest priority. Therefore, 'normal business movements' are not allowed.
- Live animals pose the greatest risk of disease spread; therefore, their movements from all premises within the restricted area (RA) and control area (CA) must be strictly controlled.
- The outside area (OA) should remain as 'clean' as possible. Therefore, movement of animals from the RA to the OA is prohibited, and movement of products is generally prohibited. Movement of animals and products from the CA to the OA will also be restricted.
- Trace premises (TP) and suspect premises (SP) are temporary classifications, and every effort should be made to resolve the status of these premises as soon as possible.
- The numbers of susceptible animals within the RA should be minimised. Therefore, movements of animals into the RA will be limited and usually for slaughter only.
- Movement restrictions are more stringent within the RA than within the CA, and will be more stringent in the early stages of the response.
- Movement controls may be varied during a response from those listed here. However, this will involve a variation to the agreed Emergency Animal Disease Response Plan, with endorsement by the Consultative Committee on Emergency Animal Diseases (CCEAD) and the National Management Group (NMG).
- Recommended movement controls apply to any movement off a premises, whether on foot or by vehicle, that involves either public or private land.
- All movement control matrixes and narratives are for guidance.
- Application for a movement permit does not automatically mean that one will be granted.
- In emergency or exceptional circumstances, any proposed movement may be considered by the jurisdictional chief veterinary officer (CVO) on a risk-assessed case-by-case basis.
- Interstate movements will need to meet the import requirements of the receiving jurisdiction.

6.2 Guidelines for issuing permits

In an emergency animal disease (EAD) event, quarantine and movement controls must strike a balance between quick and effective disease control and business continuity. Therefore, it is not appropriate to simply prohibit all movement of animals and products. On the other hand, diligence needs to be applied to minimise the risk of further spread of the disease.

Recommended biosecurity and movement controls in each AUSVETPLAN response strategy provide guidance on which movements can be allowed and under what conditions. This is based on an analysis of the disease risks that are presented by a specific movement, of a specific commodity, at a specific time during the EAD response phase. Each disease strategy will indicate whether a proposed movement is:

- allowed (under normal jurisdictional, including interstate, requirements)
- prohibited – except under the conditions of a general, special or emergency permit
- prohibited.

Permits may not be available until the relevant CVO provides approval for movements, and this may not be available in the early stages of a response. When assessing risk for the purposes of issuing a permit, the elements to consider may include:

- sources of risk
    - risk material such as live or dead susceptible animals, semen, embryos, meat, meat products, waste products, offal, paunch screenings, manure, render material, fertiliser, biological specimens, casings, used wrappers and cartons, effluent, fomites (vehicles, people, nonsusceptible animals, crops, grains, hay silage and mixed feeds)
    - presence of the disease agent on both the originating and destination premises, and uncertainty
    - location of source and destination premises
    - fate at destination premises (eg for slaughter vs for growing out)
    - current vector activity, if relevant
    - organisation and management issues (ie confidence in animal tracing and surveillance, biosecurity)
    - proposed use of the animals or products
    - proposed transport route
    - vaccination status of the animals, if relevant
    - security and monitoring at the destination
    - environment and natural events
    - community and human behaviour
    - risk of sabotage
    - technology
    - regulations and standards
    - available resources for compliance and enforcement
- areas of impact
    - livestock health (health of affected species, including animal welfare)
    - human health (including work health and safety)
    - trade and economic impacts (including commercial and legal impacts)
    - environmental impacts
    - organisational capacity
    - political impacts
    - reputation and image
    - proposed risk treatment measures
    - vaccination
    - destruction of animals
    - processing of product
    - disinfection or other treatment of animals, vehicles and fomites
    - vector control, if relevant
    - security
    - communication.

6.3 Types of permits

Permits are either general or special. Emergency permits are a form of special permit. Permits are legal documents that describe the animal(s), commodities or things to be moved, the origin and destination, and the conditions to be met for the movement. Either type of permit may include conditions. Once permit conditions have been agreed from an operational perspective, all permit conditions must be met for every permit. Both general and special permits may be in addition to documents required for routine movements between or within jurisdictions (eg health certificates, waybills, consignment notes, National Vendor Declarations – NVDs).

General permit

General permits (GPs) are used for lower-risk movements, and create a record of each movement to which they apply. They are granted without the need for direct interaction between the person moving the animal(s), commodity or thing and a government veterinarian or gazetted inspector of stock. The permit may be completed via a webpage or in an approved place (such as a government office or commercial premises). A printed version of the permit must accompany the movement. The permit may impose preconditions and/or restrictions on movements. GPs may not be available until the relevant CVO gives approval for general movements, and this may not be available in the early stages of a response.

Special permit

Special permits (SpPs) are issued by the relevant government veterinarian or gazetted inspector of stock. They are used for higher-risk movements, and therefore require formal application and individual risk assessment. SpPs describe the requirements for movement of an animal (or group of animals), commodity or thing, for which a specific assessment has been conducted by the relevant government veterinarian or gazetted inspector of stock. A printed version of the permit must accompany the movement. The permit may impose preconditions and/or restrictions on movements.

Emergency permit

An emergency permit is an SpP that specifies strict legal requirements for an otherwise high-risk movement of an animal, to enable emergency veterinary treatment to be delivered, to enable animals to be moved for animal welfare reasons, or to enable any other emergency movement under exceptional circumstances. These permits are issued on a case-by-case basis under the authorisation of the relevant CVO.

Other movement requests

Movements not reflected within any of the movement control matrixes or narratives may be considered by the relevant jurisdictional CVO on a risk-assessed case-by-case basis.

6.4 Recommended movement controls

Refer to the **AUSVETPLAN enterprise manual *Poultry industry (chickens, ducks and turkeys)*** for detailed industry information.

Movement controls are commensurate with the determined risk, taking into account the need to minimise impacts on business continuity, and return to normal business and trade as soon as possible.

Refer to Appendix 4 for movement permit conditions.

6.4.1 Live susceptible animals

All movements of live birds and bird products off infected premises (IPs), dangerous contact premises (DCPs), SPs and TPs are prohibited.

The status of TPs and SPs must be resolved before movement permits for birds or bird products can be applied for.

Where possible, RAs should not include hatcheries.

Repopulation assessment will occur once the RA has been resolved into a CA.

For movements from OA to OA, company records must be kept and made available for inspection, if required.

Premises other than IPs, DCPs, SPs and TPs

Table 6.1 shows the recommended movement controls for live day-old chicks from premises other than IPs, DCPs, SPs and TPs for farm-to-farm movement other than slaughter.

Table 6.1 Recommended movement controls for live day-old chicks from premises other than IPs, DCPs, SPs and TPs for farm-to-farm movement other than slaughter

To→ From ↓	**RA (ARP)**	**CA (POR)**	**OA**
RA (ARP)	Prohibited, except under SpP – conditions 2, 3, 4 (replacing CA or OA with RA), 5, 6, 9, 11, 14, 30, 65, when risk assessment has been completed and movement will not adversely affect the response	Prohibited	Prohibited
CA (POR)	Prohibited, except under SpP – conditions 1, 2, 3, 4, 5, 6, 9, 11, 14, 30, 65	Prohibited, except under SpP – conditions 1, 2, 3, 4, 5, 6, 9, 32	Prohibited, except under SpP – conditions 1, 2, 3, 4, 5, 6, 9, 32
OA	Prohibited	Prohibited, except under GP – conditions 3, 5, 7, 8, 9, 10, 11, 14	Allowed under normal jurisdictional, including interstate, requirements[a]

ARP = at-risk premises; CA = control area; GP = general permit; OA = outside area; POR = premises of relevance; RA = restricted area; SpP = special permit
a Company records must be kept and made available for inspection, if required.

Table 6.2 shows the recommended movement controls for other poultry from premises other than IPs, DCPs, SPs and TPs for farm-to-farm movement other than slaughter. This includes movement of pullets to layer farms, and pullets from breeder rearers to breeder producers; live bird sales to backyard or hobby farms, auctions, markets and so on; and 'brood and move' in breeder and broiler farm operations.

Table 6.2 Recommended movement controls for live birds other than day-old chicks from premises other than IPs, DCPs, SPs and TPs

To→ From ↓	**RA**	**CA**	**OA**
RA	Prohibited,[a] except under SpP – conditions 2, 3, 4 (replacing CA or OA with RA), 5, 6, 9, 11, 14, 30, 65, when risk assessment has been completed and movement will not adversely affect the response	Prohibited[a]	Prohibited[a]
CA	Prohibited,[a] except under SpP – conditions 1, 2, 3, 4, 5, 6, 9, 11, 14, 30, 65	Prohibited,[a] except under SpP – conditions 2, 4, 5, 6, 8, 9, 11, 14, 24, 30, 32, 57, 58, 59, 65	Prohibited,[a] except under SpP – conditions 2, 5, 6, 8
OA	Prohibited[a]	Prohibited,[a] except under GP – conditions 9, 10, 11, 14, 24, 30, 41	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area; SpP = special permit
a Although movement of live birds between farms is prohibited, these birds may:
– be allowed to move to slaughter (see Table 6.3)
– remain on farm if welfare conditions can be met, provided biosecurity risks do not increase
– be destroyed.

Table 6.3 shows the recommended movement controls for live birds to slaughter from premises other than IPs, DCPs, SPs and TPs. This includes meat birds, spent hens and breeders for human consumption, including emergency process slaughtering.

Table 6.3 Recommended movement controls for live birds to slaughter from premises other than IPs, DCPs, SPs and TPs

To→ From ↓	**RA**	**CA**	**OA**
RA	Prohibited, except under SpP[a] – conditions 2, 5, 6, 11, 12, 14, 15, 16, 18	Prohibited, except under SpP[a] – conditions 2, 5, 6, 11, 12, 14, 15, 16, 18	Prohibited[b]
CA	Prohibited, except under SpP[a] – conditions 2, 5, 6, 11, 14, 15, 16, 19	Prohibited, except under SpP[a] – conditions 2, 5, 6, 11, 14, 15, 16	Prohibited, except under SpP[a] – conditions 2, 5, 6, 11, 14, 15, 16
OA	Prohibited, except under SpP – conditions 2, 5, 6, 11, 14, 15, 16, 19	Prohibited, except under GP – conditions 2, 5, 6, 10, 11, 14, 20	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area; SpP = special permit
a See also permit conditions for movement of meat and meat products (Section 6.4.3) and other animal byproducts (Section 6.4.5) for restrictions that may apply post-slaughter.

b Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis)

Other susceptible species

Other susceptible species (eg pigs and birds other than the principal farm species) should not be moved from IPs, DCPs, SPs and TPs without risk assessment, and the issue of an appropriate permit.

6.4.2 Carcasses

IPs, DCPs, SPs and TPs

Table 6.4 shows the recommended movement controls for dead (whole) birds from IPs and DCPs for disposal (burial, rendering, composting or incineration).

TPs and SPs will need to be resolved before a movement permit can be considered and issued.

Table 6.4 Recommended movement controls for dead birds to disposal from IPs and DCPs

To → From ↓	RA	CA	OA
IP/DCP	Prohibited, except under SpP – conditions 6, 21, 22, 23, 24, 25, 30, 51, 57, 58, 59, 60	Prohibited	Prohibited

CA = control area; DCP = dangerous contact premises; IP = infected premises; OA = outside area; RA = restricted area; SpP = special permit

Premises other than IPs, DCPs, SPs and TPs

Table 6.5 shows the recommended movement controls for dead (whole) birds from premises other than IPs, DCPs, SPs and TPs for disposal (burial, rendering, composting or incineration). This includes hatchery culls, zoo food and balut eggs.

Table 6.5 Recommended movement controls for dead birds to disposal from premises other than IPs, DCPs, SPs and TPs

To → From ↓	RA	CA	OA
RA	Prohibited, except under SpP – conditions 6, 21, 22, 23, 24, 25, 26, 27	Prohibited, except under SpP[a] – conditions 6, 21, 22, 23, 24, 25, 26, 27	Prohibited
CA	Prohibited, except under SpP – conditions 6, 21, 22, 23, 24, 25, 26, 27	Prohibited, except under SpP – conditions 6, 21, 22, 23, 24, 25, 26, 27	Prohibited
OA	Prohibited, except under GP[b] – condition 28	Prohibited, except under GP[b] – condition 28	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area; SpP = special permit
a Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis)
b Not the preferred option for disposal of dead birds from OA.

6.4.3 Meat and meat products

Premises other than IPs, DCPs, SPs and TPs

Table 6.6 shows the recommended movement controls for meat and meat products from premises other than IPs, DCPs, SPs and TPs (movements from these premises are prohibited). This includes meat, whole birds and all other products recovered from the processing plant (eg offal, feet, tongues, oviducts, ova, frames, bones, pluck) for retail or further processing into products for human consumption or pet food.

Table 6.6 Recommended movement controls for meat and meat products from premises other than IPs, DCPs, SPs and TPs

To→ From ↓	RA	CA	OA
RA	Prohibited, except under SpP – conditions 13, 29, 66	Prohibited, except under SpP – conditions 29, 66	Prohibited, except under SpP – conditions 29, 66
CA	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements	Prohibited, except under GP – condition 29
OA	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area; SpP = special permit

6.4.4 Eggs and egg products

Eggs and egg products on IPs, DCPs, SPs and TPs for disposal

Table 6.7 shows the recommended movement controls for eggs and egg products from IPs and DCPs going for disposal.

TPs and SPs must be resolved before a movement permit can be considered and issued.

Table 6.7 Movement of eggs and egg products for disposal from IPs and DCPs

To→ From ↓	RA	CA	OA
IP/DCP	Prohibited, except under SpP[a] – conditions 6, 21, 22, 23, 30, 36, 42, 57, 58, 59, 60, 61	Prohibited	Prohibited

a Movement from an IP or DCP is not the preferred option. Movement permits will be considered on a case-by-case basis after risk assessment.

Eggs to hatchery or pulping

IPs, DCPs, SPs and TPs

Table 6.8 shows the recommended movement controls for eggs and egg products from IPs and DCPs going for pulping and pasteurisation for human consumption.

Movement of eggs from IPs and DCPs to a hatchery is prohibited.

TPs and SPs must be resolved before a movement permit can be considered and issued.

Table 6.8 Recommended movement controls for eggs and egg products on IPs and DCPs going for pulping and pasteurisation

To→ From ↓	**RA**	**CA**	**OA**
IP/DCP	Prohibited, except under SpP[a] – conditions 6, 21, 22, 23, 30, 36, 39, 42, 50, 57, 58, 59, 60, 61	Prohibited	Prohibited

CA = control area; DCP = dangerous contact premises; IP = infected premises; OA = outside area; RA = restricted area; SpP = special permit

a Movement from an IP or DCP is not the preferred option. Movement permits will be considered on a case-by-case basis after risk assessment.

Premises other than IPs, DCPs, SPs and TPs

Table 6.9 shows the recommended movement controls for fertile eggs to hatchery or pulping (commercial food production) from premises other than IPs, DCPs, SPs and TPs.

Table 6.9 Recommended movement controls for fertile eggs to hatchery or pulping from premises other than IPs, DCPs, SPs and TPs

To→ From ↓	**RA**	**CA**	**OA**
RA	Prohibited	Prohibited, except under SpP – conditions 5, 6, 9, 11, 14, 31, 36, 37, 41, 42, 43, 44, 57, 58, 59, 60, 62, 63, 64, 65	Prohibited[a]
CA	Prohibited	Prohibited, except under GP – conditions 5, 11, 14, 31, 36, 37, 57	Prohibited, except under GP – conditions 5, 11, 14, 31, 36, 37, 57
OA	Prohibited	Prohibited, except under GP – conditions 5, 11, 14, 31, 36, 37, 57	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area; SpP = special permit

a Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis).

Table (shell) eggs to grading or processing facilities from premises other than IPs, DCPs, SPs and TPs

Table 6.10 shows the recommended movement controls for table (shell) eggs to grading facilities or processing facilities from premises other than IPs, DCPs, SPs and TPs. This may include eggs from breeders.

Table 6.10 Recommended movement controls for table (shell) eggs to grading or processing facilities from premises other than IPs, DCPs, SPs and TPs

To→ From ↓	**RA**	**CA**	**OA**
RA	Prohibited, except under SpP – conditions 2, 33, 34, 35, 36, 37, 38, 39, 40, 43	Prohibited, except under SpP – conditions 2, 32, 33, 34, 35, 36, 37, 38, 39, 40	Prohibited[a]
CA	Prohibited, except under GP – conditions 41, 42	Allowed under normal jurisdictional, including interstate, requirements	Prohibited, except under GP – condition 41
OA	Prohibited, except under GP – conditions 41, 42	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area; SpP = special permit

a Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis).

Table eggs from grading facilities (other than IPs, DCPs, SPs and TPs) to retail or processing (pulping)

Table 6.11 shows the recommended movement controls for table eggs from grading facilities (other than IPs, DCPs, SPs and TPs) to retail or processing (pulping).

Table 6.11 Recommended movement controls for table (shell) eggs from grading facility to retail or processing (pulping)

To→ From ↓	RA	CA	OA
RA	Prohibited, except under GP – conditions 39, 41, 43, 44, 58	Prohibited, except under GP – conditions 41, 42	Prohibited[a]
CA	Prohibited, except under GP – conditions 41, 42	Prohibited, except under GP – conditions 41, 42	Prohibited, except under GP – conditions 41, 42
OA	Prohibited, except under GP – conditions 41, 42	Prohibited, except under GP – conditions 41, 42	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area

a Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis).

Fertile eggs to hatchery

Table 6.12 shows the recommended movement controls for fertile eggs to hatchery from IPs, DCPs, SPs and TPs.

Table 6.12 Recommended movement controls for fertile eggs to hatchery from IPs, DCPs, SPs and TPs

To→ From ↓	RA	CA	OA
RA	Prohibited, except under GP – conditions 39, 41, 43, 44, 58	Prohibited, except under GP – conditions 41, 42	Prohibited[a]
CA	Prohibited, except under GP – conditions 41, 42	Prohibited, except under GP – conditions 41, 42	Prohibited, except under GP – conditions 41, 42
OA	Prohibited, except under GP – conditions 41, 42	Prohibited, except under GP – conditions 41, 42	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area

a Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis).

Fertile eggs for research or vaccine production

The following criteria need to be met at the point of origin (nucleus stock supplier, specific pathogen–free egg supplier, research farm, commercial supplier) for fertile eggs being used for research or vaccine production, or going to diagnostic testing facilities:

- no evidence of disease on the property of origin
- biosecure facility
- biosecure transport
- handling and storage of eggs in a biosecure manner.

The decision to permit movements will be made on a case-by-case basis using a thorough risk assessment.

6.4.5 Other animal byproducts

Premises other than IPs, DCPs, SPs and TPs

Table 6.13 shows the recommended movement controls for byproducts from processing facilities other than IPs, DCPs, SPs and TPs. Byproducts include offal, feathers, blood, off-cuts, fat, frames, bones, trim, downgrades and so on that are not fit for human consumption and are being transported to rendering plants.

Table 6.13 Recommended movement controls for byproducts from processing plants on premises other than IPs, DCPs, SPs and TPs

To→ From ↓	**RA**	**CA**	**OA**
RA	Prohibited, except under SpP – conditions 6, 21, 24, 45, 46, 47, 57	Prohibited, except under SpP – conditions 6, 21, 24, 45, 46, 47, 48	Prohibited[a]
CA	Prohibited, except under SpP – conditions 6, 21, 24, 45, 46, 47, 48, 49	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements
OA	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; OA = outside area; RA = restricted area; SpP = special permit

a Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis).

Table 6.14 shows the recommended movement controls for byproducts from rendering facilities other than IPs, DCPs, SPs and TPs. These byproducts include meatmeal, feathermeal, bloodmeal and tallow.

Table 6.14 Recommended movement controls for byproducts from rendering plants on premises other than IPs, DCPs, SPs and TPs

To→ From ↓	**RA**	**CA**	**OA**
RA	Prohibited, except under SpP – condition 49	Prohibited, except under SpP – condition 49	Prohibited, except under SpP – condition 49
CA	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements
OA	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; OA = outside area; RA = restricted area; SpP = special permit

6.4.6 Waste products and effluent

IPs, DCPs, TPs and SPs

Table 6.15 shows the recommended movement controls for manure and used litter (and other waste products, including hatchery waste, processing plant waste, contaminated packaging waste and egg processing waste) from IPs and DCPs.

SPs and TPs must be resolved before a movement permit can be considered and issued.

Table 6.15 Recommended movement controls for manure, used litter and other waste products from IPs and DCPs

To→ From ↓	**RA**	**CA**	**OA**
IP/DCP	Prohibited, except under SpP – conditions 6, 21, 22, 23, 24, 52, 53, 57, 58, 59, 60	Prohibited	Prohibited
SP/TP	Prohibited	Prohibited	Prohibited

CA = control area; DCP = dangerous contact premises; IP = infected premises; OA = outside area; RA = restricted area; SpP = special permit

Premises other than IPs, DCPs, SPs, TPs and at-risk premises (ARPs)

Table 6.16 shows the recommended movement controls for waste products from premises other than IPs, DCPs, SPs, TPs and ARPs. Waste includes hatchery waste, processing plant waste, contaminated packaging waste, egg processing waste, litter and manure.

Table 6.16 Recommended movement controls for waste products on premises other than IPs, DCPs, SPs, TPs and ARPs

To→ From ↓	RA	CA	OA
RA	Prohibited, except under SpP – conditions 6, 21, 23, 24, 45, 50, 51	Prohibited, except under SpP – conditions 6, 21, 23, 24, 45, 50, 51	Prohibited[a]
CA	Prohibited, except under SpP – conditions 6, 21, 23, 24, 45, 50, 51	Prohibited, except under GP – condition 28	Prohibited, except under GP – condition 28
OA	Prohibited, except under SpP – conditions 6, 21, 23, 24, 45, 50, 51	Prohibited, except under GP[b] – condition 28	Allowed under normal jurisdictional, including interstate, requirements

CA = control area; GP = general permit; OA = outside area; RA = restricted area; SpP = special permit
a Not the preferred approach, but can be considered if no other option following risk assessment (decision to be made on a case-by-case basis).
b Not the preferred option for disposal of waste from OA.

ARPs

Table 6.17 shows the recommended movement controls for manure and litter from ARPs.

Table 6.17 Recommended movement controls for manure and litter from ARPs

To→ From ↓	RA	CA	OA
ARP	Prohibited, except under SpP – conditions 2, 5, 6, 11, 12, 21, 22, 23, 24, 28, 52, 53, 54, 58, 59	Prohibited, except under SpP – conditions 2, 5, 6, 11, 12, 21, 22, 23, 24, 28, 52, 53, 54, 58, 59	Prohibited, except under SpP – conditions 2, 5, 6, 11, 12, 21, 22, 23, 24, 28, 52, 53, 54, 58, 59

ARP = at-risk premises; CA = control area; OA = outside area; RA = restricted area; SpP = special permit

6.4.7 Vehicles, including empty livestock transport vehicles and associated equipment

Movements of vehicles and equipment that have had direct contact with susceptible animals, or their products or wastes (including with potentially contaminated mud, etc)

- Movements off quarantined premises (IPs, SPs, TPs, DCPs and DCPFs) should be prohibited and subject to risk assessment on a case-by-case basis. Where movements are allowed under SpP, the vehicles and equipment must be decontaminated before and after use at an appropriate site under the supervision of an authorised government officer. Where decontamination of equipment is not practicable, the equipment should be disposed of in a biosecure manner (see Section 4.3.9).
- Movements off other premises in the RA should be prohibited and subject to risk assessment on a case-by-case basis. Where movements are permitted, the vehicles and equipment must be decontaminated before and after use at an appropriate site under the supervision of an authorised government officer. Where decontamination of equipment is not practicable, the equipment should be disposed of in a biosecure manner (see Section 4.3.9).
- Movements off other premises in the CA should be prohibited except under GP, with the conditions that the vehicles and equipment must be decontaminated before and after use at an appropriate site (eg truck wash-down facility at an abattoir) using a protocol provided by the response authority, and records must be kept of the movement and decontamination protocol used.
- Movements off premises in the OA should not be restricted.

Movements of other vehicles and equipment

- Movements onto or off quarantined premises (IPs, SPs, TPs, DCPs and DCPFs) should be restricted and subject to risk assessment on a case-by-case basis. If the risk assessment concludes that the vehicle or equipment may potentially be contaminated with AI virus, then decontaminate/dispose.
- Movements onto or off other premises with susceptible animals in the RA or CA should be discouraged, where possible. Regular, routine vehicle movements onto farms, such as those for feed deliveries, require particular attention because of the essential nature of these movements, their frequency and the risk that they may present.
- Movements onto or off other premises in the OA should not be restricted.

On leaving the RA, all vehicles will be subject to inspection and may undergo a decontamination process, if warranted.

6.4.8 Nonsusceptible animals

Where nonsusceptible animals could act as mechanical vectors for AI virus – for example, on IPs, DCPs, SPs and TPs – appropriate decontamination measures should be implemented.

Unnecessary movements of nonsusceptible animals onto and off premises with susceptible animals in the RA should be discouraged.

Nonsusceptible species should be prevented from coming into proximity to poultry facilities. They should not be moved from IPs, DCPs, SPs and TPs without risk assessment, and the issue of an appropriate permit.

6.4.9 People

Movement controls should not hinder movements of the general public. However, where humans could act as mechanical vectors for AI virus – for example, on IPs, DCPs, DCPs, SPs and TPs – appropriate decontamination measures should be implemented. Should human infection with the outbreak virus occur, human health authorities will manage public health issues.

Unnecessary movements of people onto and off premises with susceptible animals in the RA should be discouraged.

Within the RA, people who regularly travel from farm to farm and come into contact with susceptible animals will be required to undergo appropriate decontamination of themselves, and their outer wear, equipment and vehicles between properties, and keep detailed records of their movements. They will be required to follow biosecurity controls at each premises they visit.

People involved in delivering feed and other essential materials (eg water, gas, diesel) to declared premises, including IPs, TPs SPs, DCPs and DCPFs, must comply with the following conditions (conditions 58–60 in Appendix 4):

- Driver should not exit the cabin of the truck.
- Driver should not contact poultry.
- Driver and cab are decontaminated if driver exits the truck on declared premises (both cab and driver are decontaminated before the driver re-enters the cab).

Movements on premises other than SPs and TPs within the CA and OA will not be restricted.

6.4.10 Crops, grains, hay, silage and mixed feeds

Crops, grains, hay and silage harvested from paddocks that were sprayed or treated with effluent on an IP or DCP, or mixed feeds made from such constituents, are not permitted to be moved off-site until the premises is declared free from AI and appropriate decontamination has occurred.[37] Other crops and grains may be removed from IPs and DCPs after the material has been decontaminated, and moved to other premises in either the RA or the CA, provided that the vehicle movement requirements are observed.

Movements of feeds onto IPs and DCPs may be necessary for animal welfare reasons; these would be permitted from low-risk premises or premises in the OA, provided that the vehicle movement requirements are observed.

Crops and grains from premises not associated with an IP or DCP have no movement restrictions.

Other feed movements that have, or may have, an association with an IP or DCP will be risk assessed on a case-by-case basis.

Risk management of stored feed on HPAI or LPAI (H5/H7) properties

In assessing the potential disease risk from feed stored in silos or trucks on a feedmill premises associated with IPs or DCPs, the likelihood of contamination of feed must be considered.

Sources of contamination include:

- dust

[37] This will be informed through risk assessment, taking into account environmental conditions, including ambient temperature and humidity.

- aerosols
- movement of infected or potentially infected birds from sheds, resulting in virus aerosolisation, or wind-borne spread of dust or feathers
- eggs (potential for virus transmission via direct or indirect contact)
- handling or removal of manure, resulting in wind-borne spread of dust
- contamination from fomites
- human movements – cross-contamination can be minimised by biosecurity practices, including the use of dedicated staff for various elements of the farm (eg poultry sheds, feedmill operations)
- mechanical transmission by animals (eg dogs, cats, rodents, wild birds) or insects (eg flies), or contamination by infected animals (wild birds).

In assessing the potential disease risk from feed stored in silos or trucks on IPs or DCPs (but not in a feedmill), the likelihood of feed contamination by the following means must be considered:

- feed delivered from the feedmill that is already contaminated with AI virus through mechanisms described above
- introduction of virus into silos or trucks during loading of the feed, where the virus source is from items noted above
- introduction of virus following loading of silos or trucks that are not fully sealed.

Storage conditions (eg time, temperature, location, security) and treatments (eg fumigation, pelleting, acidifying) will affect the viability of the virus in stored contaminated feed.

The impact of spread of AI virus via contaminated feed should also be considered, taking into account the proposed use (eg feeding to poultry on other farms or restocked populations; feeding to other species, such as pigs) or fate (eg disposal) of the feed. Where the feed is to be disposed of, consideration must be given to disposal procedures, and time and exposure pathways that may be created during disposal; for example, feed to be buried may remain uncovered for several hours, with access by wild birds.

6.4.11 Sales, shows and other events

All sales, mobile petting zoos, shows and other events involving live susceptible animals within the RA and CA are prohibited.

Events such as sales and shows in the OA may proceed at the discretion of the relevant jurisdictional CVO, unless the risk associated with such events is deemed unacceptable within the response.

6.4.12 Other movements

Other susceptible species (eg pigs, cats and birds other than the principal farm species) should not be moved from IPs, DCPs, SPs and TPs without risk assessment, and the issue of an appropriate permit.

7 Surveillance and proof of freedom

Following an outbreak of avian influenza (AI), surveillance will be required to demonstrate that infection has been eradicated from the population and enable any remaining movement restrictions to be lifted. Proof of freedom will also be needed to satisfy trading partners and regain access to international markets.

A key requirement for trading partners will be evidence of an effective surveillance program capable of detecting infection if present in the population, and analysis of data to support the case for disease freedom. Descriptions of the veterinary services, demographics of susceptible populations and relevant industry structures should be included to justify the design of the surveillance program.

7.1 Surveillance

Principles for designing a post-outbreak surveillance program

To provide confidence that AI is no longer present, a comprehensive surveillance program will be required, and evidence of this program may need to be provided to international trading partners to support export market access. This program will need to be carefully designed, detailed and followed to ensure that it produces sufficient data that are reliable and acceptable to international trading partners, while avoiding a program that is excessively costly and logistically complicated. Following an outbreak, the program must demonstrate absence of infection with AI viruses in susceptible poultry populations during the 3 months preceding declaration of freedom. The post-outbreak surveillance program will build on the previous surveillance, tracing and diagnostic testing done during the control phase. It should include clinical and serological surveillance, and targeted and random components.

Clinical surveillance

The aim of clinical surveillance is to look for evidence of infection by detecting clinical signs of AI at the flock level, by physical examination of susceptible animals. It involves monitoring of production parameters – such as increased mortality, reduced feed and water consumption, reduced production rates, or a decline in egg production – and the presence of clinical signs consistent with a respiratory infection with AI virus. Where clinical disease is suspected, laboratory testing should be undertaken by sampling and testing animals suspected of infection. This may include serology and virus isolation.

The approaches used for clinical surveillance will be a continuation of measures in place during the response and should include:

- a public relations and awareness campaign for producers (including backyard producers), zoo/cage-bird owners, company technical personnel and animal health professionals (eg veterinarians, stock inspectors, meat inspectors) to immediately report suspicions of AI to government veterinary services
- enhanced clinical inspection of poultry at abattoirs
- ensuring notification of suspected disease to authorities on suspect premises pending diagnosis
- effective veterinary investigations and diagnostic services that demonstrate that suspect cases are promptly investigated
- use of standardised investigation protocol and reporting forms.

In addition to clinical and/or laboratory investigation of suspect cases reported to authorities (passive surveillance), some active surveillance would also be expected, to look for the disease in groups of animals that are at particularly high risk.

Sampling regime

Surveys based on random sampling are important in providing reliable evidence that AI virus infection is not present in a country. The sampling strategy will be designed to demonstrate the absence of AI virus circulation at an acceptable level of statistical confidence. Important factors that need to be taken into account when designing the sampling regime include:

- design prevalence – the minimum level of infection that would be detected if the disease is present
- target population – the population under surveillance, which should cover all susceptible species, including, where appropriate, wild birds
- level of statistical confidence required in the results
- sensitivity and specificity of diagnostic tests
- sample size – number of flocks to be sampled and number of birds to be sampled per flock.

Particular attention will need to be paid to selecting an appropriate design prevalence and statistical confidence level for surveys, because these parameters will have to be justified and withstand international scrutiny. Since no diagnostic tests are perfect, the survey design should anticipate the occurrence of false positive reactions and incorporate appropriate follow-up procedures.

Wild bird surveillance

Surveillance of wild bird populations will be commensurate with the level of assessed risk posed to domestic bird populations. Wild bird surveillance, if used, will be in accordance with Appendix 3.

Use of sentinel birds and restocking

Use of identifiable sentinel birds (see also Section 4.3.14) to confirm completion of appropriate decontamination on depopulated premises (resolved premises – RPs) is a valuable step in demonstrating freedom from AI. Birds placed should be tested by polymerase chain reaction (PCR) and serology less than 7 days before placement to demonstrate absence of infection when placed on the RP.

Sentinel birds must be a minimum of 7 weeks of age.

The number of sentinel birds should include an allowance for losses unrelated to AI that may occur over the 21-day observation period. This will ensure that sufficient birds are available for testing at the end of the period.

If sentinel birds show clinical signs following pre-placement sampling that could indicate AI, appropriate samples should be taken and test negative for AI before the birds are used as sentinels. Alternatively, substitute birds could be sourced.

It is essential that sentinel birds are exposed to areas that may have a risk of residual AI virus. For example, having sentinel birds roaming the floor of cage facilities is more desirable than having them caged; however, consideration must be given to the appropriateness of such action.

When restocking, the following three options can be considered; the sample sizes to test are as given in Table 7.1 for all options (plus extra birds – eg 3–5% of the sample size – to account for any mortality):

- Commercial restocking without sentinels.
    - Commercial restocking may be undertaken without the use of sentinels. Historically, full repopulation has not been allowed until at least 21 days following cleaning and disinfection. The risk of infection in restocked populations has been successfully reduced in overseas outbreaks by using an extended period of 60 days after cleaning and disinfection before repopulation.
    - If repopulation is undertaken without use of sentinels, dead bird sampling of repopulated flocks should be undertaken during the 21-day observation period to confirm the ongoing absence of AI.
- Commercial restocking plus sentinels.
    - Sentinel birds should be restocked within sheds at the appropriate stocking density in accordance with national animal welfare standards and, for free-range facilities, sufficient numbers to allow the birds to be exposed to all areas potentially contaminated with AI virus.
    - In a barn or litter shed, the sentinels can be placed in the penned area in the corner of a restocked commercial shed. For testing, the sentinels are sampled randomly within the penned area, with no requirement to retest the same birds.
    - In a caged bird shed, the sentinels can be placed randomly throughout the shed, with their cages identified at the appropriate stocking density in accordance with national animal welfare standards and to allow the birds to be exposed to all areas potentially contaminated with AI virus. For testing, the sentinels are chosen at random throughout the shed, with no requirement to retest the same birds.
    - In free-range facilities, sentinel birds should be allowed to roam all areas of the free-range production area. For testing, the sentinels are sampled, with no requirement to retest the same birds.
- Sentinels only.
    - An ideal minimum number of sentinel birds would be 200 birds per shed (eg maximum stocking density is 30 kg/m^2 for noncaged layers or pullets). For free-range facilities, sufficient numbers should be used to allow the birds to be exposed to all areas potentially contaminated with AI virus (CSIRO 2002).
    - In a barn or litter shed, the sentinels are allowed to roam on the floor. For testing, the sentinels are sampled, with no requirement to retest the same birds.
    - In a caged bird shed, the sentinels would ideally roam on the floor. If this is not logistically possible, they are placed randomly throughout the shed, with their cages identified.
    - In free-range facilities, sentinel birds should be allowed to roam all areas of the free-range production area. For testing, the sentinels are sampled, with no requirement to retest the same birds.

In some situations, restocking may take a number of days. If restocking takes more than 7 days, on the last day of restocking, birds on the source property are also to be sampled as described in Table 7.1. This will ensure that, if the RP breaks down, it is not due to contamination from the source property.

Tracheal and cloacal swabs will be taken from an appropriate sample size (see Table 7.1) for PCR, and blood samples will be taken for serological testing, 21 days after placing sentinels or restocking the

RP. During this time, any unusual health incidents are to be fully investigated, and subsequent resampling will occur to ensure meaningful serological results. If any live birds show clinical signs that indicate AI, they must immediately be tested for AI using PCR (tracheal and cloacal swab) and test negative, to support freedom from AI.

Table 7.1 Sample sizes for testing of sentinel birds

Barn/litter birds		**Caged birds**	
Population per shed	**Sample size per shed**	**Population per shed**	**Sample size per shed**
≤30	All	≤40	All
31–60	31	41–60	41
61–100	33	61–100	55
101–200	35	101–200	60
>200	40	>200	64

Note: Assumptions for barn/litter birds – sensitivity 98%, specificity 98%, design prevalence 15%; assumptions for caged birds – sensitivity 98%, specificity 98%, design prevalence 10%

a See also Sergeant ESG (2016). Epitools epidemiological calculators (http://epitools.ausvet.com.au).

Surveillance plan following depopulation, decontamination and sentinel bird program

The following surveillance plan may be considered for demonstrating the absence of AI. It has been successfully used in previous highly pathogenic avian influenza (HPAI) outbreaks in New South Wales (Maitland in 2012 and Young in 2013–14).[38]

Health monitoring

The owner or manager of poultry flocks should fax or email daily reports to a designated surveillance officer. The information to be provided, collated and assessed includes:

- any decline in feed and/or water consumption
- mortality rates and the relationship of daily mortality figures to normal figures
- production rates and any decline in egg production from normal
- any clinical signs attributable to AI
- a statement declaring the health or otherwise of the poultry.

Surveillance in the restricted area

Surveillance to be undertaken in the restricted area (RA) includes:

- identification and mapping of all commercial poultry premises in the RA; each owner is to be advised by the local control centre (LCC) to immediately report health issues to the state or territory department of agriculture and arrange
    - daily health monitoring, with a daily report to be submitted to the LCC
    - weekly sampling of flocks (cloacal swabs for PCR) on each commercial poultry premises, with final sampling 21 days following depopulation on all infected premises (IPs)

[38] Information provided by the NSW Department of Primary Industries

- identification and mapping of all backyard bird flocks in the RA; each owner is to be advised to immediately report health issues to the state or territory department of agriculture as soon as possible
- collection of cloacal swabs for PCR testing from all backyard poultry flocks as soon as possible after all birds on the IPs are destroyed and again 21 days later
- prompt investigation of any reports of disease in backyard poultry flocks, cage or zoo birds, or wild birds.

Surveillance in the control area

Surveillance to be undertaken in the control area (CA) includes:

- identification and mapping of all commercial poultry premises in the CA; each owner is to be advised by the LCC to immediately report health issues to the state or territory department of agriculture and arrange
    - daily health monitoring, with a daily report to be submitted to the LCC
    - weekly sampling of flocks (cloacal swabs) on each commercial poultry premises, with final sampling 21 days following depopulation on all IPs
- prompt investigation of any reports of disease in backyard poultry flocks, cage or zoo birds, or wild birds.

Surveillance in the outside area

In the outside area, poultry producers, veterinarians and members of the public are requested to report any suspicion of AI to the relevant state or territory department of agriculture as soon as possible.

Removal of restricted and control areas

The RA will be revoked following:

- investigation of any reports of disease consistent with AI with negative test results
- negative test results from backyard and commercial flocks within the RA 21 days following destruction of all birds on IPs.

The CA will be revoked following:

- investigation of any reports of disease consistent with AI with negative test results
- negative test results from flocks within the RA 42 days following destruction of all birds on IPs.

See also Section 5.4 for details on reclassifying previously declared areas.

Surveillance following removal of restricted and control areas

Surveillance in the 3 months following removal of the RA and CA should consist of passive surveillance in both previously infected and uninfected areas, including:

- review of laboratory submissions and reports that have features consistent with AI
- investigation of all suspect cases (reports of disease consistent with AI) in poultry and wild birds
- analysis of reports from routine wild bird surveillance in jurisdictions where wild bird surveillance projects are in place.

Active surveillance during this period should also include ongoing health monitoring on RPs. Jurisdictional departments of agriculture should be notified of the presence or absence of clinical disease on these premises.

7.2 Proof of freedom

The World Organisation for Animal Health (OIE) *Terrestrial animal health code* (Chapter 10.4, Article 10.4.3) lists the criteria (see also Section 4.1.4) for a previously AI-free country or zone to be recognised as free from AI following an outbreak (note that the OIE definition of poultry will apply). To underpin an official self-declaration of AI-free status following an outbreak, Australia would develop a formal report detailing the eradication procedures undertaken, the surveillance program (see Section 7.1) and the results reported. This report could be provided to trading partners to transparently document the basis for our self-declaration.

Trading partner acceptance of AI-free status following an outbreak will most likely have to be negotiated with individual trading partners. This may take considerably longer than the minimum periods prescribed in the OIE Terrestrial Code.

Appendix 1

AVIAN INFLUENZA FACT SHEET

Disease and cause

Avian influenza (AI) is caused by a number of influenza viruses, of which only influenza A viruses have been isolated from avian species; influenza viruses of the B, C and D types have never been isolated from birds.

AI viruses are classified into two pathotypes – highly pathogenic AI (HPAI) and low pathogenicity AI (LPAI) – based on either the lethality of the virus in experimentally inoculated chickens or molecular characteristics.

Species affected

Influenza A viruses have been isolated from most major bird families – at least 105 bird species of 26 families have been reported to date. Experimentally, AI virus can infect almost all commercial, domestic and wild avian species.

Humans are susceptible to infection with AI viruses.

Distribution

AI is widely distributed throughout the world, and outbreaks have occurred in Australia. Wild birds in Australia have been shown to actively carry and shed LPAI virus.

Potential pathways for introduction into commercial poultry in Australia

The most likely pathways of introduction of AI into commercial poultry in Australia are through:

- transfer from asymptomatic waterfowl to susceptible flocks via close contact
- migration of birds on established flyways
- contaminated poultry products, fomites or people.

Key signs

Key signs in chickens and turkeys include severe respiratory signs with excessively watery eyes and sinusitis; cyanosis of the combs, wattle and shanks; oedema of the head; ruffled feathers; loss of appetite; diarrhoea; nervous signs; and sudden death.

Eggs may be misshapen. The last eggs laid after the onset of illness frequently have no shells. Some severely affected hens may recover, but rarely come back into lay.

Spread

The principal means by which AI viruses initiate outbreaks is thought to be via wild birds contaminating range areas, water or food supplies for poultry. The infection subsequently spreads via movements of infected live birds, or faecally contaminated feed, equipment, materials, clothing and footwear. Infected backyard poultry and live bird markets can be a source of AI virus for commercial poultry.

Persistence of the virus

Influenza viruses are lipid-enveloped RNA viruses. Stability of AI viruses varies by subtype. In general, influenza viruses are fragile; however, some serotypes can persist longer under optimal conditions. The virus is stable over a pH range of 5.0–8.2.

Impacts for Australia

One of the biggest impacts of an outbreak of AI involving the poultry industry would be on the domestic economy. Chicken meat and eggs are produced very efficiently in Australia and provide the cheapest source of animal-based protein available to the population; this is reflected in the consumption rate. Loss of availability of these products would cause economic stress to the majority of the population and the domestic economy.

Appendix 2

INFECTIVE DOSE AND VIRUS SHEDDING

Infective dose

The infective dose for avian influenza (AI) virus under experimental conditions, for infection of chickens, turkeys, ducks, geese and quail, depends on the species of origin of the virus isolate, the virus subtype, the species infected, the age of individuals and the route of infection. Examples from studies of infective doses are shown in Table 9.1.

Table 0.1 Experimental infective dose of avian influenza virus by species

Species	**Infective dose**	
	LPAI (EID_{50})	**HPAI (EID_{50})**
Ducks	$10^{1.9}$–$10^{3.3}$	–
Chickens	$10^{5.8}$–$10^{7.7}$	$10^{1.2}$–$10^{4.7}$ (all virus origins) $10^{2.8}$–$10^{4.7}$ (turkey origin) $10^{1.2}$–$10^{3.0}$ (chicken origin)
Turkeys	$10^{4.2}$–$10^{6.0}$	–
Quail and geese	$10^{1.5}$–$10^{5.4}$	–

– = no data; EID_{50} = median egg infective dose; HPAI = highly pathogenic avian influenza; LPAI = low pathogenicity avian influenza

Source: Swayne & Slemons (2008)

Virus shedding

Virions are released from tissues within 16 hours after initial exposure. Virus shedding occurs before the onset of clinical signs and may last longer than apparent disease. The infectious period extends from the time the virus is first detected in the bird to the time it is no longer detected in oropharyngeal or cloacal swabs. Examples of concentrations of virus that may be detected in respiratory secretions or faeces are given in Table 9.2.

Table 0.2 Concentrations of avian influenza virus detected in respiratory secretions or faeces

	Virus concentration	
	Respiratory secretions (EID_{50}/mL)	**Faeces (EID_{50}/g)**
LPAI	$10^{1.1}$–$10^{5.5}$	$10^{1.0}$–$10^{4.3}$
HPAI	$10^{4.2}$–$10^{7.7}$	$10^{2.5}$–$10^{7.5}$

EID_{50} = median egg infective dose; HPAI = highly pathogenic avian influenza; LPAI = low pathogenicity avian influenza

Viruses with the potential to be highly pathogenic for chickens and turkeys can be carried by birds, and shed in faeces and from the respiratory tract for at least 14 days and up to 30 days after clinical

recovery from the disease (Webster et al 1978). Cloacal shedding can continue for longer than 30 days after infection in the presence of immunosuppressive diseases or other physical stresses.

Experimentally infected ducks have been shown to shed HPAI (H5N1) virus via both the cloaca and the respiratory tract for at least 17 days (Hulse-Post et al 2005).

In pheasants, partridges and guineafowl, virus was able to be recovered for up to 7 days after infection during the outbreak in the United States in 1983–84 (Pearson et al 2003).

Experimentally infected cats excreted H5N1 virus from the pharynx and nose, and in faeces for at least 7 days. Pharyngeal swabs contained $10^{4.5}$ tissue culture infective dose $(TCID)_{50}$/mL, and nasal swabs $10^{2.5}$–$10^{5.0}$ $TCID_{50}$/mL; virus titres in faeces varied widely, but were of similar magnitude (Rimmelzwaan et al 2006).

Appendix 3

SAMPLING, TESTING AND RESPONSE POLICY FOR HPAI IN WILD BIRDS

Sampling and testing

- Testing for avian influenza (AI) in wild birds will be performed only in laboratories that are competent and are using appropriate methods.
- Confirmation of highly pathogenic avian influenza (HPAI) virus infection is obtained from the isolation and typing of an HPAI virus, or the detection of genetic material from an HPAI virus. Seroevidence of H5 or H7 antibodies is not evidence of infection with HPAI virus, and response action will not be taken based on the results of serological testing only.
- As there is rarely an opportunity for follow-up sampling and testing of individual wild birds, sampling of wild birds for AI virus will include appropriate cloacal and/or tracheal samples for PCR testing and virus detection. Collection of serum samples only should generally be avoided, given the difficulties in interpretation and the inability to collect further meaningful samples from the sampled birds at a later time.
- At the time of sampling, all relevant details will be recorded, including date, location, species, circumstances and clinical status of the bird(s) sampled.
- If only blood samples are available, animal health authorities will consider all available information, including clinical signs and mortality reports, and determine the value and feasibility of further investigations, including the ability to identify the target population and collect further samples.

Response

The response to the detection of HPAI in wild birds will be based on risk assessment, and will include consideration of the circumstances under which sampling occurred, the possible source of the virus, the species involved, the clinical status of the sampled birds or population, the proximity to commercial and other significant bird establishments, and the proximity to public amenity areas.

Where evidence of infection with HPAI virus has been found in the situations described in Tables 10.1–10.3, the actions identified in the tables will be considered. The Consultative Committee on Emergency Animal Diseases (CCEAD) will decide on further action to be taken, based on consideration of the number of wild birds affected and how widespread the infection is. Tables 10.1–10.3 outline the actions to be considered in the following three situations:

- Table 10.1 – HPAI virus is detected in a wild bird.
- Table 10.2 – HPAI virus is detected in more than one wild bird.
- Table 10.3 – HPAI virus infection is widespread in wild birds only.

If HPAI virus is detected in one or more wild birds, the chief veterinary officer (CVO) of the state or territory of origin of the sampled bird(s) should immediately notify the Australian CVO, who will convene a CCEAD meeting.

No destruction of wild birds will occur other than for reasons of animal welfare. If HPAI virus is detected in wild birds, the response will be measured, and commensurate with the level of assessed risk posed to domestic and wild bird populations, and the risk posed to the community in general.

Table 0.1 Actions to consider if HPAI virus is detected in a wild bird

Action	Comments
Consult with, and provide appropriate advice to, relevant national and state/territory environment officials and peak industry bodies	–
Provide appropriate advice to relevant national and state/territory health authorities	–
Where practical, timely and appropriate, commence epidemiological investigations by personnel with wild bird expertise, in the area where the bird was found	–
Consult as necessary with expert groups (eg ornithologists) to consider the ecology of the avian species involved	Determine possible range of involvement
Consider possible source of the HPAI virus	From the ecology of the species involved
Consider the value of sampling live wild birds and, as necessary, sick and dead wild birds, for evidence of HPAI virus; sampling of dead birds may be particularly justified in the case of HPAI	Establish protocols and determine which agency will collect samples
Identify private or public enterprises holding birds that are in the vicinity and whether any risks require managing	Including zoos and public parks
Develop a communications strategy based on the findings of the risk assessment; relevant communication messages may include: • assurances concerning public health risks, in concert with public health authorities • advice on the importance of biosecurity to bird owners, poultry producers, veterinarians and wildlife carers • the need for vigilance by poultry and other bird owners for signs of unusual disease, and the requirement for reporting to animal health authorities	Including the public, bird owners and the wildlife community

– = no comments; HPAI = highly pathogenic avian influenza

If HPAI virus is detected in more than one wild bird in a group or area, the CCEAD may further consider the matters in Table 10.2.

Table 0.2 Actions to consider if HPAI virus is detected in more than one wild bird

Action	Comments
Undertake actions in Table 10.1	–
Consider sampling of identified wild and domesticated birds	Statistically based sampling regime may be considered, if practical
Provide targeted advice and warnings on biosecurity to:	
• veterinarians	–
• managers of public collections	Zoos and parks
• owners of pet and aviary birds	–
• owners of poultry	Backyard and all poultry industry sectors
• wildlife community	–
• duck hunters	–
• others with a vested interest	Wildlife carers, avian researchers, environment agencies, nature wardens, ornithologists, twitchers
• general public	–
Consider necessary actions for wetlands and sanctuaries	–

– = no comments; HPAI = highly pathogenic avian influenza

If further investigations prove that HPAI infection is widespread in wild birds only (ie not domestic birds), the CCEAD should consider whether further actions, set out in Table 10.3, may be justified.

Table 0.3 Actions to consider if HPAI infection is widespread in wild birds only

Action	Comments
Identify and proclaim restricted areas, if appropriate, including considering whether controls should be applied over movement and congregation of birds, and movement of fomites (eg at bird shows and pigeon races, and research activities)	Only likely to be of value in exceptional circumstances, when justified by assessed risk
Apply appropriate surveillance and biosecurity measures	Based on assessed risk
Apply enhanced biosecurity and control measures at local poultry holdings	Including disinfection of footwear or tyres and excluding contact with wild birds
Implement enhanced communications strategy, including advice to other agencies, industries, public	Including zoos and public parks, wildlife carers and wildlife researchers (based on assessed risk)
Consider use of vaccination in domestic poultry and for captive birds	Policy to be agreed by the CCEAD and in accordance with AUSVETPLAN (eg zoos, pets, public flocks)
Consult with health officials	Authoritative public health assurances will be essential
Consult with environment officials	

HPAI = highly pathogenic avian influenza

Appendix 4

MOVEMENT PERMIT CONDITIONS

Table 0.1 Movement permit conditions

Condition	Requirements
1	Day-old chicks come from source flock in CA.
2	Source premises must: • record mortalities and egg production daily • report abnormal levels of mortality or egg production to the LCC; dead/sick birds will be tested by PCR if indicated by increased mortality or reduced egg production. Triggers for reporting include: • severe flock depression • disease-related mortalities of 1% or more in 24 hours • severe respiratory disease symptoms • in a 7-day period, 20% or more drops in – egg production – feed consumption, or – water consumption.
3	Company declares (to the relevant state authority) that records of candling and hatchability meet breed, company and hatchery standards.
4	Transport truck, and transport and personnel have only operated in CA or OA.
5	Source premises has a biosecurity plan (especially transport; and movements of personnel, live birds, eggs and all associated equipment) that has been audited by an authorised government officer since onset of outbreak.
6	Travel is by approved route only, with no stopping en route.
7	Day-old chicks come from source flock in OA.
8	Vehicles and equipment, including empty crates, are decontaminated appropriately on entry to and exit from CA.
9	Vehicles and equipment are decontaminated appropriately.
10	Empty crates are cleaned and disinfected before return from CA to OA.
11	Absence of clinical signs of AI in the flock on the premises before and on day of travel.
12	Negative surveillance (by PCR) within 48 hours of slaughter.
13	Only to approved processing facility.

Condition	Requirements
14	Vehicles and crates are cleaned and disinfected before pick-up and after unloading.
15	Catching crew and vehicle drivers have dedicated clothing on farm, including boots, and are decontaminated off farm (including showering).
16	Catching machine is not to be used.
17	Designated processing plant in CA (movement of birds from RA to be avoided, if possible).
18	Birds originating in RA are processed last and identified. Processing facility is decontaminated following processing.
19	Movement for slaughter into the RA only if no other suitable processor.
20	Movement for slaughter into the CA only if no other suitable processor.
21	Transport is in a covered, leak-proof container and/or vehicle.
22	To transport sealed, closed containers, outside of bin is cleaned and disinfected before removal from premises.
23	Authorised method of disposal (eg composting) at an approved disposal site.
24	Vehicle and equipment are decontaminated after pick-up and delivery, and between declared areas.
25	Authorised dead bird pick-up organisation.
26	Dead birds must not be fed to, or brought into contact with, other birds or other susceptible species.
27	Multiple pick-up is permitted only if collection points are at farm perimeter within each designated declared area.
28	Vehicles and equipment are decontaminated appropriately on exit from disposal facility.
29	Movement records are kept of where the product is sold.
30	Vehicles and equipment are disinfected between premises.
31	Eggs are decontaminated on-farm (eg sprayed, fumigated, washed or disinfected). If there is no ability to decontaminate eggs on-farm, floor eggs, cracked eggs or eggs that have visual faecal contamination must not be used, and must be fumigated or washed before setting at the hatchery.
32	Vehicles and equipment, including egg fillers, are decontaminated appropriately on entry to and exit from CA.
33	Vehicles and equipment, including egg fillers, are decontaminated appropriately on entry to and exit from RA.
34	Dirty and cracked eggs are removed for safe disposal before movement.
35	Pulp produced is treated by validated heat treatment (eg pasteurisation).
36	Reuse of cardboard egg fillers and disposable packaging is prohibited; these should be disposed of safely (eg by incineration).
37	Plastic egg fillers are washed and disinfected adequately.

Condition	Requirements
38	Risk assessment is completed for individual properties before egg sales are permitted from individual premises.
39	Egg surfaces are washed or disinfected at source (eg farm or grading facility).
40	If eggs are washed on the farm, they can only be packed onto new cardboard fillers, or new or decontaminated plastic fillers.
41	Vehicles and equipment, including egg fillers, are decontaminated appropriately on entry to and exit from area.
42	No return of egg fillers and packaging from RA to CA or OA.
43	Plastic fillers must only be returned to originating farm.
44	No return of cardboard egg fillers and packaging within RA.
45	Authorised (by relevant state/territory authority) processors.
46	Approved (by relevant state/territory authority) method of processing (eg heat treatment).
47	Byproducts must not be fed to, or brought into contact with, other birds or other susceptible species.
48	To transport sealed, closed containers, outside of bin must be cleaned and disinfected before removal from premises and before return to processing plant, CA or OA.
49	Rendered product is separated from raw materials to avoid recontamination or cross-contamination.
50	Waste product must not be fed to, or brought into contact with, other birds.
51	To transport sealed, closed containers, outside of bin must be cleaned and disinfected before removal from premises and before return to processing plant.
52	Cannot be spread on land without prior processing or treatment.
53	Manure and litter must be moved to an approved disposal site.
54	Negative surveillance (by PCR) on birds within 48 hours of proposed movement.
55	Equipment to be moved is cleaned and disinfected, and records are kept.
56	If collected on plastic fillers, an adequate decontamination practice is used before return to the originating farm.
57	Trucks, including tyres, are washed on exit from property and on exit from any declared area.
58	Driver should not exit the cabin of the truck.
59	Driver should not contact poultry.
60	Driver and cab are decontaminated if driver exits the truck on declared premises (both cab and driver are decontaminated before the driver re-enters the cab).
61	Eggs are transported on cardboard egg fillers and using disposable packaging only.
62	Receiving premises must:

Condition	Requirements
	• record mortalities and egg production daily • report abnormal levels of mortality or egg production to the LCC; dead/sick birds will be tested by PCR if indicated by increased mortality or reduced egg production. Triggers for reporting include: • severe flock depression • disease-related mortalities of 1% or more in 24 hours • severe respiratory disease symptoms • in a 7-day period, 20% or more drops in – egg production – feed consumption, or – water consumption.
63	Travel is to an authorised hatchery only (by state/territory government authority).
64	Entire consignment comprises eggs from single premises only (no multiple pick-ups).
65	Entire consignment is for delivery to a single premises only (no multiple drop-offs).
66	Must have undergone approved method of processing (ie heat treatment for human consumption; rendering for animal food) before movement.

AI = avian influenza; CA = control area; LCC = local control centre; OA = outside area; PCR = polymerase chain reaction; RA = restricted area

Glossary

Disease-specific terms

Term	Definition
Cage birds	Birds that are confined within an enclosure and maintained for purposes other than food production.
Controlled marketing	Orderly marketing of birds through their normal production cycle after infection has died down or burnt out.
Cyanosis (adj: cyanotic)	Blueness of the skin and/or mucous membranes due to insufficient oxygenation of the blood.
Egg marketing premises	Premises where table eggs are graded and packed for the retail market. The premises may also contain a pulp plant and facilities for manufacture of egg-based products.
Egg pulp	A homogeneous liquid made from either whole liquid egg, egg albumen or egg yolk, pasteurised for marketing as a liquid or frozen product.
Further processing plant	A plant that receives fresh carcases from an abattoir for cutting up; processing into poultry nuggets, rolls and so on; and cooking or partial cooking for fast-food outlets and retail markets.
Galliformes (adj: gallinaceous)	The order of birds that includes poultry, turkey, pheasant and peafowl.
Haemagglutination	Agglutination of red blood cells by a specific antibody or other substance.
Haemagglutinin (vb: haemagglutinate)	Protein on the virus surface that agglutinates red blood cells.
Modified stamping out	Process in which stamping out is not implemented in full (eg the animals may be sent for process slaughter over a period of time; or some animals may be vaccinated, held under an appropriate level of biosecurity and killed at a later date). Details of the modifications should be provided. *See also* Process slaughter
Pathogenicity	Competence of an infectious agent to produce disease in the host species. The relative disease changes are described as highly, mildly or lowly pathogenic. Nonpathogenic describes the situation where infection produces no disease or clinical signs in a susceptible host. *See also* Virulence.
Peracute	Extremely acute form of a disease.
Poultry	For the purposes of this manual, 'poultry' means chickens, turkeys, guineafowl, ducks, geese, quail, pigeons, pheasants, partridges, emus and ostriches reared or kept in captivity.
Pre-emptive slaughter	Destruction of animals at high risk of infection but in which infection has not yet been demonstrated. Birds do not go for human consumption.

Term	Definition
Process slaughter	Slaughter of animals for human consumption after they have been transported, under movement controls, to a processing plant.
Processing plant	An abattoir for slaughtering animals for human consumption, with chilled and frozen storage facilities.
Proventriculus	The front (thin-walled) part of the stomach in birds.
Psittaciformes (adj: psittacine)	Parrots and related groups of birds.
Rendering	Processing by heat to inactivate infective agents. Rendered material may be used in various products according to particular disease circumstances.
Virulence	Capacity of an infectious agent to produce pathological changes. Agents that do not produce any disease signs are described as nonvirulent or avirulent. *See also* Pathogenicity
Zoo birds	Cage birds that are maintained at a zoo premises.

Standard AUSVETPLAN terms

Term	Definition
Animal byproducts	Products of animal origin that are not for consumption but are destined for industrial use (eg hides and skins, fur, wool, hair, feathers, hoofs, bones, fertiliser).
Animal Health Committee	A committee whose members are the chief veterinary officers of the Commonwealth, states and territories, along with representatives from the CSIRO Australian Centre for Disease Preparedness (CSIRO-ACDP) and the Australian Government Department of Agriculture, Water and the Environment. There are also observers from Animal Health Australia, Wildlife Health Australia, and the New Zealand Ministry for Primary Industries. The committee provides advice to the National Biosecurity Committee on animal health matters, focusing on technical issues and regulatory policy. *See also* National Biosecurity Committee
Animal products	Meat, meat products and other products of animal origin (eg eggs, milk) for human consumption or for use in animal feedstuff.
Approved disposal site	A premises that has zero susceptible livestock and has been approved as a disposal site for animal carcasses, or potentially contaminated animal products, wastes or things.
Approved processing facility	An abattoir, knackery, milk processing plant or other such facility that maintains increased biosecurity standards. Such a facility could have animals or animal products introduced from lower-risk premises under a permit for processing to an approved standard.

Term	Definition
At-risk premises	A premises in a restricted area that contains a live susceptible animal(s) but is not considered at the time of classification to be an infected premises, dangerous contact premises, dangerous contact processing facility, suspect premises or trace premises.
Australian Chief Veterinary Officer	The nominated senior veterinarian in the Australian Government Department of Agriculture, Water and the Environment who manages international animal health commitments and the Australian Government's response to an animal disease outbreak. *See also* Chief veterinary officer
AUSVETPLAN	*Aus*tralian *Vet*erinary Emergency *Plan*. Nationally agreed resources that guide decision making in the response to emergency animal diseases (EADs). It outlines Australia's preferred approach to responding to EADs of national significance, and supports efficient, effective and coherent responses to these diseases.
Carcase	The body of an animal slaughtered for food.
Carcass	The body of an animal that died in the field.
Chief veterinary officer (CVO)	The senior veterinarian of the animal health authority in each jurisdiction (national, state or territory) who has responsibility for animal disease control in that jurisdiction. *See also* Australian Chief Veterinary Officer
Compartmentalisation	The process of defining, implementing and maintaining one or more disease-free establishments under a common biosecurity management system in accordance with OIE guidelines, based on applied biosecurity measures and surveillance, to facilitate disease control and/or trade.
Compensation	The sum of money paid by government to an owner for livestock or property that are destroyed for the purpose of eradication or prevention of the spread of an emergency animal disease, and livestock that have died of the emergency animal disease. *See also* Cost-sharing arrangements, Emergency Animal Disease Response Agreement
Consultative Committee on Emergency Animal Diseases (CCEAD)	The key technical coordinating body for animal health emergencies. Members are state and territory chief veterinary officers, representatives of CSIRO-ACDP and the relevant industries, and the Australian Chief Veterinary Officer as chair.
Control area (CA)	A legally declared area where the disease controls, including surveillance and movement controls, applied are of lesser intensity than those in a restricted area (the limits of a control area and the conditions applying to it can be varied during an incident according to need).
Cost-sharing arrangements	Arrangements agreed between governments (national and state/territory) and livestock industries for sharing the costs of emergency animal disease responses. *See also* Compensation, Emergency Animal Disease Response Agreement

Term	Definition
Dangerous contact animal	A susceptible animal that has been designated as being exposed to other infected animals or potentially infectious products following tracing and epidemiological investigation.
Dangerous contact premises (DCP)	A premises, apart from an abattoir, knackery or milk processing plant (or other such facility) that, after investigation and based on a risk assessment, is considered to contain a susceptible animal(s) not showing clinical signs, but considered highly likely to contain an infected animal(s) and/or contaminated animal products, wastes or things that present an unacceptable risk to the response if the risk is not addressed, and that therefore requires action to address the risk.
Dangerous contact processing facility (DCPF)	An abattoir, knackery, milk processing plant or other such facility that, based on a risk assessment, appears highly likely to have received infected animals, or contaminated animal products, wastes or things, and that requires action to address the risk.
Declared area	A defined tract of land that is subjected to disease control restrictions under emergency animal disease legislation. There are two types of declared areas: restricted area and control area.
Decontamination	Includes all stages of cleaning and disinfection.
Depopulation	The removal of a host population from a particular area to control or prevent the spread of disease.
Destroy (animals)	To kill animals humanely.
Disease agent	A general term for a transmissible organism or other factor that causes an infectious disease.
Disease Watch Hotline	24-hour freecall service for reporting suspected incidences of exotic diseases – 1800 675 888.
Disinfectant	A chemical used to destroy disease agents outside a living animal.
Disinfection	The application, after thorough cleansing, of procedures intended to destroy the infectious or parasitic agents of animal diseases, including zoonoses; applies to premises, vehicles and different objects that may have been directly or indirectly contaminated.
Disinsectation	The destruction of insect pests, usually with a chemical agent.
Disposal	Sanitary removal of animal carcasses, animal products, materials and wastes by burial, burning or some other process so as to prevent the spread of disease.
Emergency animal disease	A disease that is (a) exotic to Australia or (b) a variant of an endemic disease or (c) a serious infectious disease of unknown or uncertain cause or (d) a severe outbreak of a known endemic disease, and that is considered to be of national significance with serious social or trade implications. *See also* Endemic animal disease, Exotic animal disease
Emergency Animal Disease Response Agreement	Agreement between the Australian and state/territory governments and livestock industries on the management of emergency animal disease responses. Provisions include

Term	Definition
	participatory decision making, risk management, cost sharing, the use of appropriately trained personnel and existing standards such as AUSVETPLAN. *See also* Compensation, Cost-sharing arrangements
Endemic animal disease	A disease affecting animals (which may include humans) that is known to occur in Australia. *See also* Emergency animal disease, Exotic animal disease
Enterprise	*See* Risk enterprise
Enzyme-linked immunosorbent assay (ELISA)	A serological test designed to detect and measure the presence of antibody or antigen in a sample. The test uses an enzyme reaction with a substrate to produce a colour change when antigen–antibody binding occurs.
Epidemiological investigation	An investigation to identify and qualify the risk factors associated with the disease. *See also* Veterinary investigation
Epidemiology	The study of disease in populations and of factors that determine its occurrence.
Exotic animal disease	A disease affecting animals (which may include humans) that does not normally occur in Australia. *See also* Emergency animal disease, Endemic animal disease
Exotic fauna/feral animals	*See* Wild animals
Fomites	Inanimate objects (eg boots, clothing, equipment, instruments, vehicles, crates, packaging) that can carry an infectious disease agent and may spread the disease through mechanical transmission.
General permit	A legal document that describes the requirements for movement of an animal (or group of animals), commodity or thing, for which permission may be granted without the need for direct interaction between the person moving the animal(s), commodity or thing and a government veterinarian or inspector. The permit may be completed via a webpage or in an approved place (such as a government office or commercial premises). A printed version of the permit must accompany the movement. The permit may impose preconditions and/or restrictions on movements. *See also* Special permit
In-contact animals	Animals that have had close contact with infected animals, such as noninfected animals in the same group as infected animals.
Incubation period	The period that elapses between the introduction of a pathogen into an animal and the first clinical signs of the disease.
Index case	The first case of the disease to be diagnosed in a disease outbreak. *See also* Index property
Index property	The property on which the index case is found. *See also* Index case
Infected premises (IP)	A defined area (which may be all or part of a property) on which animals meeting the case definition are or were present, or the

Term	Definition
	causative agent of the emergency animal disease is present, or there is a reasonable suspicion that either is present, and that the relevant chief veterinary officer or their delegate has declared to be an infected premises.
Local control centre (LCC)	An emergency operations centre responsible for the command and control of field operations in a defined area.
Monitoring	Routine collection of data for assessing the health status of a population or the level of contamination of a site for remediation purposes. *See also* Surveillance
Movement control	Restrictions placed on the movement of animals, people and other things to prevent the spread of disease.
National Biosecurity Committee (NBC)	A committee that was formally established under the Intergovernmental Agreement on Biosecurity (IGAB). The IGAB was signed on 13 January 2012, and signatories include all states and territories except Tasmania. The committee provides advice to the Agriculture Senior Officials Committee and the Agriculture Ministers' Forum on national biosecurity issues, and on the IGAB.
National Management Group (NMG)	A group established to approve (or not approve) the invoking of cost sharing under the Emergency Animal Disease Response Agreement. NMG members are the Secretary of the Australian Government Department of Agriculture, Water and the Environment as chair, the chief executive officers of the state and territory government parties, and the president (or analogous officer) of each of the relevant industry parties.
Native wildlife	*See* Wild animals
OIE Terrestrial Code	OIE *Terrestrial animal health code*. Describes standards for safe international trade in animals and animal products. Revised annually and published on the internet at: www.oie.int/en/what-we-do/standards/codes-and-manuals/terrestrial-code-online-access.
OIE Terrestrial Manual	OIE *Manual of diagnostic tests and vaccines for terrestrial animals*. Describes standards for laboratory diagnostic tests, and the production and control of biological products (principally vaccines). The current edition is published on the internet at: www.oie.int/en/what-we-do/standards/codes-and-manuals/terrestrial-manual-online-access.
Operational procedures	Detailed instructions for carrying out specific disease control activities, such as disposal, destruction, decontamination and valuation.
Outside area (OA)	The area of Australia outside the declared (control and restricted) areas.
Owner	Person responsible for a premises (includes an agent of the owner, such as a manager or other controlling officer).
Polymerase chain reaction (PCR)	A method of amplifying and analysing DNA sequences that can be used to detect the presence of viral DNA.

Term	Definition
Premises	A tract of land including its buildings, or a separate farm or facility that is maintained by a single set of services and personnel.
Premises of relevance (POR)	A premises in a control area that contains a live susceptible animal(s) but is not considered at the time of classification to be an infected premises, suspect premises, trace premises, dangerous contact premises or dangerous contact processing facility.
Prevalence	The proportion (or percentage) of animals in a particular population affected by a particular disease (or infection or positive antibody titre) at a given point in time.
Proof of freedom	Reaching a point following an outbreak and post-outbreak surveillance when freedom from the disease can be claimed with a reasonable level of statistical confidence.
Qualifiers	
– assessed negative	Assessed negative (AN) is a qualifier that may be applied to ARPs, PORs, SPs, TPs, DCPs or DCPFs. The qualifier may be applied following surveillance, epidemiological investigation, and/or laboratory assessment/diagnostic testing and indicates that the premises is assessed as negative at the time of classification.
– sentinels on site	Sentinels on site (SN) is a qualifier that may be applied to IPs and DCPs to indicate that sentinel animals are present on the premises as part of response activities (ie before it can be assessed as an RP).
– vaccinated	The vaccinated (VN) qualifier can be applied in a number of different ways. At its most basic level, it can be used to identify premises that contain susceptible animals that have been vaccinated against the EAD in question. However, depending on the legislation, objectives and processes within a jurisdiction, the VN qualifier may be used to track a range of criteria and parameters.
Quarantine	Legally enforceable requirement that prevents or minimises spread of pests and disease agents by controlling the movement of animals, persons or things.
Resolved premises (RP)	An infected premises, dangerous contact premises or dangerous contact processing facility that has completed the required control measures, and is subject to the procedures and restrictions appropriate to the area in which it is located.
Restricted area (RA)	A relatively small legally declared area around infected premises and dangerous contact premises that is subject to disease controls, including intense surveillance and movement controls.
Risk enterprise	A defined livestock or related enterprise that is potentially a major source of infection for many other premises. Includes intensive piggeries, feedlots, abattoirs, knackeries, saleyards, calf scales, milk factories, tanneries, skin sheds, game meat establishments, cold stores, artificial insemination centres, veterinary laboratories and hospitals, road and rail freight depots, showgrounds, field days, weighbridges and garbage depots.

Term	Definition
Sensitivity	The proportion of truly positive units that are correctly identified as positive by a test. *See also* Specificity
Sentinel animal	Animal of known health status that is monitored to detect the presence of a specific disease agent.
Seroconversion	The appearance in the blood serum of antibodies (as determined by a serology test) following vaccination or natural exposure to a disease agent.
Serosurveillance	Surveillance of an animal population by testing serum samples for the presence of antibodies to disease agents.
Serotype	A subgroup of microorganisms identified by the antigens carried (as determined by a serology test).
Serum neutralisation test	A serological test to detect and measure the presence of antibody in a sample. Antibody in serum is serially diluted to detect the highest dilution that neutralises a standard amount of antigen. The neutralising antibody titre is given as the reciprocal of this dilution.
Slaughter	The humane killing of an animal for meat for human consumption.
Special permit	A legal document that describes the requirements for movement of an animal (or group of animals), commodity or thing, for which the person moving the animal(s), commodity or thing must obtain prior written permission from the relevant government veterinarian or inspector. A printed version of the permit must accompany the movement. The permit may impose preconditions and/or restrictions on movements. *See also* General permit
Specificity	The proportion of truly negative units that are correctly identified as negative by a test. *See also* Sensitivity
Stamping out	The strategy of eliminating infection from premises through the destruction of animals in accordance with the particular AUSVETPLAN manual, and in a manner that permits appropriate disposal of carcasses and decontamination of the site.
State coordination centre (SCC)	The emergency operations centre that directs the disease control operations to be undertaken in a state or territory.
Surveillance	A systematic program of investigation designed to establish the presence, extent or absence of a disease, or of infection or contamination with the causative organism. It includes the examination of animals for clinical signs, antibodies or the causative organism.
Susceptible animals	Animals that can be infected with a particular disease.
Suspect animal	An animal that may have been exposed to an emergency disease such that its quarantine and intensive surveillance, but not pre-emptive slaughter, is warranted. or

Term	Definition
	An animal not known to have been exposed to a disease agent but showing clinical signs requiring differential diagnosis.
Suspect premises (SP)	Temporary classification of a premises that contains a susceptible animal(s) not known to have been exposed to the disease agent but showing clinical signs similar to the case definition, and that therefore requires investigation(s).
Swill	Also known as 'prohibited pig feed', means material of mammalian origin, or any substance that has come in contact with this material, but does not include: (i) Milk, milk products or milk by-products either of Australian provenance or legally imported for stockfeed use into Australia. (ii) Material containing flesh, bones, blood, offal or mammal carcases which is treated by an approved process.[1] (iii) A carcass or part of a domestic pig, born and raised on the property on which the pig or pigs that are administered the part are held, that is administered for therapeutic purposes in accordance with the written instructions of a veterinary practitioner. (iv) Material used under an individual and defined-period permit issued by a jurisdiction for the purposes of research or baiting. [1] In terms of (ii), approved processes are: 1. rendering in accordance with the 'Australian Standard for the Hygienic Rendering of Animal Products' 2. under jurisdictional permit, cooking processes subject to compliance verification that ensure that a core temperature of at least 100 °C for a minimum of 30 minutes, or equivalent, has been reached. 3. treatment of cooking oil, which has been used for cooking in Australia, in accordance with the 'National Standard for Recycling of Used Cooking Fats and Oils intended for Animal Feeds' 4. under jurisdictional permit, any other nationally agreed process approved by AHC for which an acceptable risk assessment has been undertaken and that is subject to compliance verification. The national definition is a minimum standard. Some jurisdictions have additional conditions for swill feeding that pig producers in those jurisdictions must comply with, over and above the requirements of the national definition.
Swill feeding	Also known as 'feeding prohibited pig feed', it includes: • feeding, or allowing or directing another person to feed, prohibited pig feed to a pig • allowing a pig to have access to prohibited pig feed

Term	Definition
	• the collection and storage or possession of prohibited pig feed on a premises where one or more pigs are kept • supplying to another person prohibited pig feed that the supplier knows is for feeding to any pig. This definition was endorsed by the Agriculture Ministers' Council through AGMIN OOS 04/2014.
Trace premises (TP)	Temporary classification of a premises that contains susceptible animal(s) that tracing indicates may have been exposed to the disease agent, or contains contaminated animal products, wastes or things, and that requires investigation(s).
Tracing	The process of locating animals, people or other items that may be implicated in the spread of disease, so that appropriate action can be taken.
Unknown status premises (UP)	A premises within a declared area where the current presence of susceptible animals and/or risk products, wastes or things is unknown.
Vaccination	Inoculation of individuals with a vaccine to provide active immunity.
Vaccine	A substance used to stimulate immunity against one or several disease-causing agents to provide protection or to reduce the effects of the disease. A vaccine is prepared from the causative agent of a disease, its products or a synthetic substitute, which is treated to act as an antigen without inducing the disease.
– adjuvanted	A vaccine in which one or several disease-causing agents are combined with an adjuvant (a substance that increases the immune response).
– attenuated	A vaccine prepared from infective or 'live' microbes that are less pathogenic but retain their ability to induce protective immunity.
– gene deleted	An attenuated or inactivated vaccine in which genes for non-essential surface glycoproteins have been removed by genetic engineering. This provides a useful immunological marker for the vaccine virus compared with the wild virus.
– inactivated	A vaccine prepared from a virus that has been inactivated ('killed') by chemical or physical treatment.
– recombinant	A vaccine produced from virus that has been genetically engineered to contain only selected genes, including those causing the immunogenic effect.
Vector	A living organism (frequently an arthropod) that transmits an infectious agent from one host to another. A *biological* vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. A *mechanical* vector is one that transmits an infectious agent from one host to another but is not essential to the life cycle of the agent.

Term	Definition
Veterinary investigation	An investigation of the diagnosis, pathology and epidemiology of the disease. *See also* Epidemiological investigation
Viraemia	The presence of viruses in the blood.
Wild animals	
– native wildlife	Animals that are indigenous to Australia and may be susceptible to emergency animal diseases (eg bats, dingoes, marsupials).
– feral animals	Animals of domestic species that are not confined or under control (eg cats, horses, pigs).
– exotic fauna	Nondomestic animal species that are not indigenous to Australia (eg foxes).
Wool	Sheep wool.
Zero susceptible species premises (ZP)	A premises that does not contain any susceptible animals or risk products, wastes or things.
Zoning	The process of defining, implementing and maintaining a disease-free or infected area in accordance with OIE guidelines, based on geopolitical and/or physical boundaries and surveillance, to facilitate disease control and/or trade.
Zoonosis	A disease of animals that can be transmitted to humans.

Abbreviations

Disease-specific abbreviations

Abbreviation	Full title
AI	avian influenza
EID	egg infectious dose
HA	haemagglutinin
HPAI	highly pathogenic avian influenza
LPAI	low pathogenicity avian influenza
NA	neuraminidase
PPE	personal protective equipment
TCID	tissue culture infective dose

Standard AUSVETPLAN abbreviations

Abbreviation	Full title
ACDP	Australian Centre for Disease Preparedness
AN	assessed negative
APF	approved processing facility
ARP	at-risk premises
AUSVETPLAN	Australian Veterinary Emergency Plan
CA	control area
CCEAD	Consultative Committee on Emergency Animal Diseases
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CVO	chief veterinary officer
DCP	dangerous contact premises
DCPF	dangerous contact processing facility
EAD	emergency animal disease
EADRA	Emergency Animal Disease Response Agreement
EADRP	Emergency Animal Disease Response Plan
EDTA	ethylenediaminetetraacetic acid (anticoagulant for whole blood)
ELISA	enzyme-linked immunosorbent assay

Abbreviation	Full title
GP	general permit
IETS	International Embryo Technology Society
IP	infected premises
LCC	local control centre
NASOP	nationally agreed standard operating procedure
NMG	National Management Group
OA	outside area
OIE	World Organisation for Animal Health
PCR	polymerase chain reaction
POR	premises of relevance
RA	restricted area
RP	resolved premises
SCC	state coordination centre
SP	suspect premises
SpP	special permit
TP	trace premises
UP	unknown status premises
ZP	zero susceptible species premises

References

Abdel-Moneim AS, Abdel-Ghany AE & Shany SAS (2010). Isolation and characterization of highly pathogenic avian influenza virus subtype H5N1 from donkeys. *Journal of Biomedical Science* 17: art 25, www.jbiomedsci.com/content/17/1/25.

Achenbach JE & Bowen RA (2011). Transmission of avian influenza A viruses among species in an artificial barnyard. PLoS One 6(3):e17643.

Alarcon P, Brouwer A, Venkatesh D, Duncan D, Dovas CI, Georgiades G, Monne I, Fusaro A, Dan A, Śmietanka K, Ragias V, Breed AC, Chassalevris T, Goujgoulova G, Hjulsager CK, Ryan E, Sánchez A, Niqueux E, Tammiranta N, Zohari S, Stroud DA, Savić V, Lewis NS & Brown IH (2018). Comparison of 2016–17 and previous epizootics of highly pathogenic avian influenza H5 Guangdong lineage in Europe. *Emerging Infectious Diseases* 24(12):2270–2283.

Alfonso CP, Cowen BS & Campen HV (1995). Influenza A viruses isolated from waterfowl in two wildlife management areas of Pennsylvania. *Journal of Wildlife Diseases* 31(2):179–185.

Allwright DM, Burger EP, Geyer A & Terblanche AW (1993). Isolation of an influenza A virus from ostriches (*Struthio camelus*). *Avian Pathology* 22:59–65.

Arzey G (2004). The role of wild aquatic birds in the epidemiology of avian influenza in Australia. *Australian Veterinary Journal* 82:277–388.

Arzey GG, Kirkland PD, Arzey KE, Frost M, Maywood P, Conaty S, Hurt AC, Deng Y-M, Iannello P, Barr I, Dwyer DE, Ratnamohan M, McPhie K & Selleck P (2012). Influenza virus A (H10N7) in chickens and poultry abattoir workers, Australia. *Emerging Infectious Diseases* 18(5):814–816.

Barbazan P, Thitithanyanont A, Misse D, Dubot A, Bosc P, Luangsri N, Gonzalez JP & Kittayapong P (2008). Detection of H5N1 avian influenza virus from mosquitoes collected in an infected poultry farm in Thailand. *Vector-borne and Zoonotic Diseases* 8(1):105–110.

Barber MRW, Aldridge Jr JR, Webster RG & Magor KE (2010) Association of RIG-I with innate immunity of ducks to influenza. *Proceedings of the National Academy of Sciences USA* 107(13):5913–5918.

Beato MS & Capua I (2011). Transboundary spread of highly pathogenic avian influenza through poultry commodities and wild birds: a review. *OIE Scientific and Technical Review* 30(1):51–61.

Becker W & Uys C (1967). Experimental infection of chicken with influenza A/Tern/South Africa/1961 and chicken/Scotland/1959 viruses. 1. Clinical picture and virology. *Journal of Comparative Pathology* 77:159–165.

Bowes, VA, Ritchie SJ, Byrne S, Sojonky K, Bidulka JJ & Robinson JH (2004). Virus characterisation, clinical presentation, and pathology associated with H7N3 avian influenza in British Columbia broiler breeder chickens in 2004. *Avian Diseases* 48:928–934.

Brown I, Mulatti P, Smietanka K, Staubach C, Willeberg P, Adlhoch C, Candiani D, Fabris C, Zancanaro G, Morgado J & Verdonc F (2017). Avian influenza overview October 2016–August 2017. *EFSA Journal* 15(10): e05018.

Brown JD, Stallknecht DE, Beck JR, Suarez DL & Swayne DE (2006). Susceptibility of North American ducks and gulls to H5N1 highly pathogenic avian influenza viruses. *Emerging Infectious Diseases* 12(11):1663–1670.

Brown JD, Swayne DE, Cooper RJ, Burns RE & Stallknecht DE (2007). Persistence of H5 and H7 avian influenza viruses in water. *Avian Diseases* 51:285–289.

Brown JD, Goekjian G, Poulson R, Valeika S & Stallknecht DE (2008a). Avian influenza virus in water: infectivity is dependent on pH, salinity and temperature. *Veterinary Microbiology* 136(1–2):20–26.

Brown JD, Stallknecht DE & Swayne DE (2008b). Experimental infection of swans and geese with highly pathogenic avian influenza virus (H5N1) of Asian lineage. *Emerging Infectious Diseases* 14(1):136–142.

Bublot M, Pritchard N, Swayne DE, Selleck P, Karaca K, Suarez DL, Audonnet JC & Mickle TR (2006). Development and uses of fowlpox vectored vaccines for avian influenza. *Annals of the New York Academy of Sciences* 1081:193–201.

Bulach D, Halpin R, Spiro D, Pomeroy L, Janies D & Boyle DB (2010). Molecular analysis of H7 avian influenza viruses from Australia and New Zealand: genetic diversity and relationships from 1976 to 2007. *Journal of Virology* 84(19):9957–9966.

Cappucci DT, Johnson DC, Brugh M, Smith TM, Jackson CF, Pearson JE & Senne DA (1985). Isolation of AI virus (subtype H5N2) from chicken eggs during a natural outbreak. *Avian Diseases* 29(4):1195–1200.

Capua I, Mutinelli F, Bozza MA, Terregino C & Cattoli G (2000a). Highly pathogenic avian influenza (H7N1) in ostriches (*Struthio camelus*). *Avian Pathology* 29:643–646.

Capua I, Grossele B, Bertoli E & Cordioli P (2000b). Monitoring for highly pathogenic avian influenza in wild birds in Italy. *Veterinary Record* 147:640.

Capua I, Marangon S, Dalla Pozza M, Terregino C & Cattoli G (2003). Avian influenza in Italy 1997–2001. *Avian Diseases* 47:839–843.

Chen Q, Wang H, Zhao L, Ma L, Wang R, Lei Y, Li Y, Yang G, Chen J, Chen G, Li L, Jin T, Li J, Liu X, Xu X, Wong G, Liu L, Liu Y, Shi W, Bi Y & Gao GF (2016). First documented case of avian influenza (H5N1) virus infection in a lion. *Emerging Microbes & Infections* 5(12):e125.

Chen Y, Ge W & Huang C (2008). Serological survey of antibody to H9 and H6 subtypes of bird flu virus in healthy youths in Guangxi. *China Tropical Medicine* 8:985–986.

Chumpolbanchorn K, Suemanotham N, Siripara N, Puyati B & Chaichoune K (2006). The effect of temperature and UV light on infectivity of avian influenza virus (H5N1, Thai field strain) in chicken fecal manure. *Southeast Asian Journal of Tropical Medicine and Public Health* 37(1):102–105.

Clavijo A, Riva J, Copps J, Robinson Y & Zhou E-M (2001). Assessment of the pathogenicity of an emu-origin influenza AH5 virus in ostriches (*Struthio camelus*). *Avian Pathology* 30:83–89.

Curran J (2012). *The surveillance and risk assessment of wild birds in northern Australia for highly pathogenic avian influenza H5N1 virus*, PhD thesis, Murdoch University.

De Vriese J, Steensels M, Palya V, Gardin Y, Dorsey KM, Lambrecht B, Van Borm S & van den Berg T (2010). Passive protection afforded by maternally-derived antibodies in chickens and the antibodies' interference with the protection elicited by avian influenza-inactivated vaccines in progeny. *Avian Diseases* 54:246–252.

Easterday BC, Hinshaw VL & Halvorson DA (1997). Influenza. In: Calnek BW (ed), *Diseases of poultry,* 10th edn, Iowa State University Press, 583–605.

EFSA Scientific Panel (European Food Safety Authority Scientific Panel on Animal Health and Animal Welfare) (2005). Animal health and welfare aspects of avian influenza. Annex to *EFSA Journal* 266:1021.

FAO (Food and Agriculture Organization of the United Nations) & SPC (Secretariat of the Pacific Community) (nd). *Reference guide for animal health staff*, FAO & SPC, http://lrd.spc.int/ext/Disease_Manual_Final/index.html.

Ferenczi M, Beckmann C, Warner S, Loyn R, O'Riley K, Wang X & Klaassen M (2016). Avian influenza infection dynamics under variable climatic conditions, viral prevalence is rainfall driven in waterfowl from temperate, south-east Australia. *Veterinary Research* 47:23.

Fichtner GJ (2003). The Pennsylvania/Virginia experience in eradication of avian influenza (H5N2). *Avian Diseases* 47, special issue, Proceedings of the Second International Symposium on Avian Influenza, 1986, 33–38.

Gavier-Widen D, Meredith A & Duff JP (eds) (2012). *Infectious diseases of wild mammals and birds in Europe*, Wiley-Blackwell.

Germeraad EA, Elbers ARW, de Bruijn ND, Heutink R, van Voorst W, Hakze-van der Honing R, Bergervoet SA, Engelsma MY, van der Poel WHM & Beerens N (2020). Detection of low pathogenic avian influenza virus subtype H10N7 in poultry and environmental water samples during a clinical outbreak in commercial free-range layers, Netherlands 2017. *Frontiers in Veterinary Science*, https://doi.org/10.3389/fvets.2020.00237.

Graiver DA, Topliff CL, Kelling CL & Bartelt-Hunt SL (2009). Survival of avian influenza virus (H6N2) after land disposal. *Environmental Science and Technology* 43:4063–4067.

Grillo VL, Arzey KE, Hansbro PM, Hurt AC, Warner S, Bergfeld J, Burgess GW, Cookson B, Dickason CJ, Ferenczi M, Hollingsworth T, Hoque MDA, Jackson RB, Klaassen M, Kirkland PD, Kung N, Lisovski S, O'Dea MA, O'Riley K, Roshier D, Skerratt LF, Tracey JP, Wang X, Woods R & Post L (2015). Avian influenza in Australia: a summary of 5 years of wild bird surveillance. *Australian Veterinary Journal* 93(11):387–393.

Halvorson D (2008). Control of low pathogenicity avian influenza. In: Swayne DE (ed), *Avian influenza*, Blackwell Publishing, 513–536.

Hansbro PM, Warner S, Tracey JP, Arzey KE, Selleck P, O'Riley K, Beckett EL, Bunn C, Kirkland PD, Vijaykrishna D, Olsen B & Hurt AC (2010). Surveillance and analysis of avian influenza viruses, Australia. *Emerging Infectious Diseases* 16(12):1896–1904.

Haynes L, Arzey E, Bell C, Buchanan N, Burgess G, Cronan V, Dickason C, Field H, Gibbs S, Hansbro P, Hollingsworth T, Hurt A, Kirkland P, McCracken H, O'Connor J, Tracey J, Wallner J, Warner S, Woods R & Bunn C (2009). Australian surveillance for avian influenza viruses in wild birds between July 2005 and June 2007. *Australian Veterinary Journal* 87(7):266–272.

Henzler DJ, Kradel DC, Davison S, Ziegler AF, Singletary D, DeBok P, Castro AE, Lu H, Eckroade R, Swayne D, Lagoda W, Schmucker B & Nesselrodt A (2003). Epidemiology, production losses and control measures associated with an outbreak of avian influenza virus subtype H7N2 in Pennsylvania (1996–98). *Avian Diseases* 47:1022–1036.

Hinshaw VS, Webster RG & Turner B (1979). Waterborne transmission of influenza A viruses? *Intervirology* 11:66–68.

Hulse-Post DJ, Sturm-Ramirez KM, Humberd J, Seiler P, Govorkkova EA, Krauss S, Scholtissek C, Puthvanthana P, Buranathai C, Nguyen TD, Long HT, Naipospos TA, Chen H, Ellis TM, Guan Y, Peiris JS

& Webster RG (2005). Role of domestic ducks in the propagation and biological evolution of highly pathogenic H5N1 influenza viruses in Asia. *Proceedings of the National Academy of Sciences USA* 102(30):10682–10687.

Jorgensen PH, Nielsen OL, Hansen HC, Manvell RJ, Banks J & Alexander DJ (1998). Isolation of influenza A virus, subtype H5N2, and avian paramyxovirus type 1 from a flock of ostriches in Europe. *Avian Pathology* 27:15–20.

Kalthoff D, Hoffmann B, Harder T, Durban M & Beer M (2008). Experimental infection of cattle with highly pathogenic avian influenza virus (H5N1). *Emerging Infectious Diseases* 14(7):1132–1134.

Keawcharoen J, Oraveerakul K, Kuiken T, Fouchier RA, Amonsin A, Payungporn S, Noppornpanth S, Wattanodorn S, Theamboonlers A, Tantilertcharoen R & Pattanarangsan R (2004). Avian influenza H5N1 in tigers and leopards. *Emerging Infectious Diseases* 10(12):2189–2191.

Khomenko S, Abolnik C, Roberts L, Waller L, Shaw K, Monne I, Taylor J, Dhingra M, Pittiglio C, Mugyeom M, Roche X, Kivaria F, Akiko K, Okuthe S, Kone P, Wiersma L, Von Dobschuetz S, Soumare B, Makonnen Y, Morzaria S & Lubroth J (2018). 2016–2018 spread of H5N8 highly pathogenic avian influenza (HPAI) in sub-Saharan Africa. *FOCUS ON* no. 12, www.fao.org/3/CA1209EN/ca1209en.pdf.

Klaassen M, Hoye BJ & Roshier DA (2011). Identifying crucial gaps in our knowledge of the life-history of avian influenza viruses: an Australian perspective. *Emu* 111:103–112.

Kleyheeg E, Slaterus R, Bodewes R, Rijks JM, Spierenburg MAH, Beerens N, Kelder L, Poen MJ, Stegeman JA, Fouchier RAM, Kuiken T & van der Jeugd HP (2017). Deaths among wild birds during highly pathogenic avian influenza A(H5N8) virus outbreak, the Netherlands. *Emerging Infectious Diseases* 23(12):2050–2054.

Kuiken T, Rimmelzwaan G, van Riel D, van Amerongen G, Baars M, Fouchier R & Osterhaus A (2004). Avian influenza in cats. *Science* 306:241.

Lang AS, Kelly A & Runstadler JA (2008). Prevalence and diversity of avian influenza viruses in environmental reservoirs. *Journal of General Virology* 89(2):509–519.

Lang V, Marjuki H, Krauss SL, Webby RJ & Webster RG (2011). Different incubation temperatures affect viral polymerase activity and yields of low-pathogenic avian influenza viruses in embryonated chicken eggs. *Archives of Virology* 156(6):987–994.

Latorre-Margalef N, Gunnarsson G, Munster VJ, Fouchier RAM, Osterhaus ADME, Elmberg J, Olsen B, Wallensten A, Fransson T, Brudin L & Waldenström J (2009). Does influenza A affect body condition of wild mallard ducks, or vice versa? A reply to Flint and Franson. *Proceedings of the Royal Society B: Biological Sciences* 276(1666):2347–2349.

Loeffen W, de Boer E & Koch G (2004). Transmission of a highly pathogenic avian influenza virus to swine in the Netherlands. In: *Proceedings of the In-between Congress of the International Society for Animal Hygiene*, St Malo, France, 329–330.

Maas R, Rosema S, van Zoelen D & Venema S (2011). Maternal immunity against avian influenza H5N1 in chickens: limited protection and interference with vaccine efficacy. *Avian Pathology* 40(1):87–92.

Magor KE (2011). Immunoglobulin genetics and antibody responses to influenza in ducks. *Developmental and Comparative Immunology* 35:1008–1017.

Markwell DD & Shortridge KF (1982). Possible waterborne transmission and maintenance of influenza viruses in domestic ducks. *Applied and Environmental Microbiology* 43(1):110–116.

Mase M, Imada T, Nakamura K, Tanimura N, Imai K, Tskuamoto K & Yamaguchi S (2005). Experimental assessment of the pathogenicity of H5N1 influenza A viruses isolated in Japan. *Avian Diseases* 49(4):582–584.

McQuinston JH, Garber LP, Porter-Spalding BA, Hahn JW, Pierson FW, Wainwright SH, Senne DA, Brignole TJ, Akey BL & Holt TJ (2005). Evaluation of risk factors for the spread of low pathogenicity H7N2 avian influenza virus among commercial poultry farms. *Journal of the American Veterinary Medical Association* 226(5):767–772.

Mostafa A, Abdelwhab EM, Mettenleiter TC & Pleschka S (2018). Zoonotic potential of influenza A viruses: a comprehensive overview. *Viruses* 10(9):497.

Mutinelli F, Capua I, Terregino C & Cattoli G (2003). Clinical, gross and microscopic findings in different avian species naturally infected during the H7N1 low and high pathogenicity avian influenza epidemics in Italy during 1999 and 2000. *Avian Diseases* 47(S3):844–848.

Nettles VF, Wood JM & Webster RG (1985). Wildlife surveillance associated with an outbreak of lethal H5N2 avian influenza in domestic poultry. *Avian Diseases* 29(3):733–741.

Nidom CA, Takano R, Yamada S, Sakai-Tagawa Y, Daulay S, Aswadi D, Suzuki T, Suzuki Y, Shinya K, Iwatsuki-Horimoto K, Muramoto Y & Kawaoka Y (2010). Influenza A (H5N1) viruses from pigs, Indonesia. *Emerging Infectious Diseases* 16(10):1515–1523.

Nielsen AA, Sovgard H, Stockmarr A, Handberg KJ & Jorgensen PH (2011). Persistence of low-pathogenic avian influenza H5N7 and H7N1 subtypes in house flies (Diptera: Muscidae). *Journal of Medical Entomology* 48(3):608–614.

OCVO (Office of the Chief Veterinary Officer) (2010). *National avian influenza surveillance dossier*, Australian Government Department of Agriculture, Fisheries and Forestry, Canberra, www.agriculture.gov.au/sites/default/files/sitecollectiondocuments/animal-plant/animal-health/livestock-movement/national-avian-influenza-sept-2010.pdf.

Panigrahy B & Senne DA (1998). Subtypes of avian influenza virus isolated from exotic birds and ratites in the United States 1992–1996. *Avian Diseases* 47 (2003), special edition, Proceedings of the 4th International Symposium on Avian Influenza, 1997, 70–75.

Panigrahy B, Senne DA & Pearson JE (1995). Presence of avian influenza virus (AIV) subtypes H5N2 and H7N1 in emus (*Dromaius novaehollandiae*) and rheas (*Rhea americana*): virus isolation and serologic findings. *Avian Diseases* 39(1):64–67.

Pearson JE, Senne DA, Carbrey EA, Gustafson GA, Landgraf JG, Cassidy DR & Erickson GA (2003). Laboratory support for the Pennsylvania/Virginia avian influenza outbreak. *Avian Diseases* 47, special issue, Proceedings of the Second International Symposium on Avian Influenza, 1986, 39–50.

Peiris JSM (2009). Avian influenza viruses in humans. *OIE Scientific and Technical Review* 28(1):161–174.

Power CA (2005a). An investigation into the potential role of aerosol dispersion of dust from poultry barns as a mode of disease transmission during an outbreak of avian influenza (H7:N3) in Abbotsford, BC in 2004. *Bulletin of the Aquaculture Association of Canada* 105(1):7–14.

Power C (2005b). *The source and means of spread of the avian influenza virus in the lower Fraser Valley of British Columbia during an outbreak in the winter of 2004*, interim report, Canadian Food Inspection Agency.

ProMED-mail (2003). Avian influenza – Netherlands (24): pig involvement, 21 May, *ProMED-mail*, www.promedmail.org.

ProMED-mail (2004). Avian influenza – eastern Asia (128): Thailand, tigers, 21 Oct, *ProMED-mail*, www.promedmail.org.

ProMED-mail (2005). Avian influenza – Asia (12): Vietnam, civets, H5N1, *ProMED-mail* 20050826.2527, www.promedmail.org.

ProMED-mail (2006). Avian influenza – worldwide (47): Europe, *ProMED-mail* 20060309.0752, www.promedmail.org.

ProMED-mail (2014). Avian influenza (44) – Mexico (Nuevo Leon), imported parakeets, LPAI H7N3, *ProMED-mail,* www.promedmail.org.

Promkuntod N, Antarasena C, Prommuang P & Prommuang P (2006). Isolation of avian influenza virus A subtype H5N1 from internal contents (albumen and allantoic fluid) of Japanese quail (*Cournix coturnix japonica*) eggs and oviduct during a natural outbreak. *Annals of the New York Academy of Sciences* 1081(1):171–173.

Purchase HS (1931). Experiments on the viability of the virus of fowl-plague under trade conditions. *Veterinary Record* 11:644–648.

QDAF (Queensland Department of Agriculture and Fisheries) (2020). Outbreaks of avian influenza, www.daf.qld.gov.au/business-priorities/biosecurity/animal-biosecurity-welfare/animal-health-pests-diseases/notifiable/outbreaks.

Qiao CL,Yu KZ, Jiang YP, Jia YQ, Tian GB, Liu M, Deng GH, Wang XR, Meng QW & Tang XY (2003). Protection of chickens against highly lethal H5N1 and H7N1 avian influenza viruses with a recombinant fowlpox virus co-expressing H5 haemagglutinin and N1 neuraminidase genes. *Avian Pathology* 32:25–32.

Reid AH, Fanning TG, Slemons RD, Janczewski TA, Dean J & Taubenberger JK (2003). Relationship of pre-1918 avian influenza HA and NP sequences to subsequent avian influenza strains. *Avian Diseases* 47:921–925.

Rimmelzwaan GF, van Riel D, Baars M, Bestbroer TM, van Amerongen G, Fouchier RAM, Osterhaus ADME & Kuiken T (2006). Influenza A virus (H5N1) infection in cats causes systemic disease with potential novel routes of virus spread within and between hosts. *American Journal of Pathology* 168(1):176–183.

Roshier DA, Robertson AI & Kingsford RT (2002). Responses of waterbirds to flooding in an arid region of Australia and implications for conservation. *Biological Conservation* 106(3):399–411.

Roshier DA, Asmus M & Klaasen M (2008). What drives long-distance movements in the nomadic grey teal *Anas gracilis* in Australia? *Ibis* 150:474–484.

Samadieh B & Bankowski RA (1971). Transmissibility of avian influenza A viruses. *American Journal of Veterinary Research* 32(6):939–945.

Savill NJ, St Rose SG & Woolhouse MEJ (2008). Detection of mortality clusters associated with highly pathogenic avian influenza in poultry: a theoretical analysis. *Journal of the Royal Society Interface* 5: 409–1419.

Sawabe K, Hoshino K, Isawa H & Sasaki T (2009). Detection and isolation of highly pathogenic H5N1 avian influenza A viruses from blow flies collected in the vicinity of an infected poultry farm in Kyoto, Japan, 2004. *American Journal of Tropical Medicine and Hygiene* 75(2):327–332.

Scott A, Hernandez-Jover M, Groves P & Toribio J-A (2020). An overview of avian influenza in the context of the Australian commercial poultry industry. *One Health* 10:100139.

Selleck PW, Arzey G, Kirkland PD, Reece RL, Gould AR, Daniels PW & Westbury HA (2003). An outbreak of highly pathogenic avian influenza in Australia in 1997 caused by an H7N4 virus. *Avian Diseases* 47:806–811.

Seo SH & Webster RG (2001). Cross-reactive, cell-mediated immunity and protection of chickens from lethal H5N1 influenza virus infection in Hong Kong poultry markets. *Journal of Virology* 75:2516–2525.

Sommer D, Heffels-Redmann U & Köhler K (2016). Rolle der Roten Vogelmilbe (*Dermanyssus gallinae*) bei der Übertragung von aviärem Influenza-A-Virus [Role of the poultry red mite (*Dermanyssus gallinae*) in the transmission of avian influenza A virus]. *Tierärtliche Praxis Ausgabe Grosstiere Nutztiere* 44(1):26–33.

Songserm T, Amonsin A, Jam-on R, Sae-Heng N, Pariyothorn N, Payungporn S, Theamboonlers A, Chutinimitkul S, Thanawongnuwech R & Poovoranwan Y (2006). Fatal avian influenza A H5N1 in a dog. *Emerging Infectious Diseases* 12:1744–1747.

Stallknecht DE, Kearney MT, Shane SM & Zwank PJ (1990). Effects of pH, temperature, and salinity on persistence of avian influenza viruses in water. *Avian Diseases* 334:412–418.

Suarez DL (2000). Evolution of avian influenza viruses. *Veterinary Microbiology* 74:15–27.

Suarez DL (2008). Influenza A virus. In: Swayne DE (ed), *Avian influenza*, Blackwell Publishing, Iowa, 3–22.

Suarez DL & Shultz-Cherry S (2000). Immunology of avian influenza virus: a review. *Developmental and Comparative Immunology* 24:269–283.

Sutton D, Aldous EW, Warren CJ, Fuller CM, Alexander DJ & Brown IH (2013). Inactivation of the infectivity of two highly pathogenic avian influenza viruses and a virulent Newcastle disease virus by ultraviolet radiation. *Avian Pathol*ogy 42(6):566–568.

Swayne DE (2008). Epidemiology of avian influenza in agricultural and other man-made systems. In: Swayne DE (ed), *Avian influenza*, Blackwell Publishing, Iowa, 59–85.

Swayne DE & Halvorson DA (2003). Influenza. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR & Swayne DE (eds), *Diseases of poultry*, 11th edn, Iowa State Press, 135–160.

Swayne DE & Halvorson DA (2008). Influenza. In: Saif YM, Fadly AM, Glisson JR, McDougald IR, Nolan LK & Swayne DE (eds), *Diseases of poultry*, 12th ed, Blackwell Publishing, 153–184.

Swayne DE & Kapczynski D (2008). Strategies and challenges for eliciting immunity against avian influenza virus in birds. *Immunological Reviews* 225:314–331.

Swayne DE & Slemons RD (2008). Using mean infectious dose of high and low pathogenicity avian influenza viruses originating from wild duck and poultry as one measure of infectivity and adaptation to poultry. *Avian Diseases* 52:455–460.

Swayne DE & Suarez DL (2000). Highly pathogenic avian influenza. *OIE Scientific and Technical Review* 19:463–482.

Swayne DE, Suarez DL & Sims LD (2013). Influenza. In: Swayne DE, Glisson JR, McDougald LR, Nolan LK, Suarez DL & Nair VL (eds), *Diseases of poultry*, 13th edn, Blackwell Publishing, Iowa, 181–218.

Tiensin T, Chaitaweesub P, Songserm T, Chaisingh A, Hoonsuwan W, Buranathai C, Parakamawongsa T, Premashthira S, Amonsin A, Gilbert M, Nielen M & Stegeman A (2005). Highly pathogenic avian influenza H5N1, Thailand, 2004. *Emerging Infectious Diseases* 11:1664–1672.

Tong S, Zhu X, Li Y, Shi M, Zhang J, Bourgeois M, Yang H, Chen X, Recuenco S, Gomez J, Chen L-M, Johnson A, Tao Y, Dreyfus C, Yu W, McBride R, Carney PJ, Gilbert AT, Chang J, Guo Z, Davis CT, Paulson JC, Stevens J, Rupprecht CE, Holmes EC, Wilson IA & Donis RO (2013). New World bats harbor diverse influenza A viruses. *PLOS Pathogens* 9(10):e1003657.

Torremorell M, Alonso C, Davies PR, Patnayak D, Torchetti M & McCluskey B (2016). Investigation into the airborne dissemination of H5N2 highly pathogenic avian influenza virus during the 2015 spring outbreaks in the midwestern United States. *Avian Diseases* 60(3):637–643.

Tracey JP (2010). Risk-based surveillance of avian influenza in Australia's wild birds. *Wildlife Research* 37(2):134–144.

Tracey JP, Woods R, Roshier D, West P & Saunders GR (2004). The role of wild birds in the transmission of avian influenza for Australia: an ecological perspective. *Emu* 104(2):109–124.

Tyasasmaya T, Wuryastuty H, Wasito & Sievert K (2016). Avian influenza virus H5N1 remained exist in gastrointestinal tracts of house flies 24 hours post-infection. *Jurnal Veteriner* 17(2):205–210.

USAHA (United States Animal Health Association) (2008). *Foreign animal diseases*, 7th edn, USAHA, St Joseph, Missouri, www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf.

Vrtiak OJ & Kapitancik B (1967). Study of fowl plague virus resistance in biological and technical material. *Bulletin Office International Des Epizooties* 67:969–688.

Wang M, Fu C & Zheng B (2009). Antibodies against H5 and H9 avian influenza among poultry workers in China. *New England Journal of Medicine* 360:2583–2584.

Webster RG, Yakhno M, Hinshaw VS, Bean WJ & Murti KG (1978). Intestinal influenza: replication and characterisation of influenza viruses in ducks. *Virology* 84:268–276.

Wei J, Zhoub J, Cheng K, Wu J, Zhong Z, Song Y, Ke C, Yen H-L & Li Y (2018). Assessing the risk of downwind spread of avian influenza virus via airborne particles from an urban wholesale poultry market. *Building and Environment* 127:120–126.

Westbury HA (1998). History of highly pathogenic avian influenza in Australia. In: Swayne DM & Slemons VA (eds), *Proceedings of the Fourth International Symposium on Avian Influenza*, US Animal Health Association, Richmond, Virginia, 23–30.

WHA (Wildlife Health Australia) (2018). *Wild Bird News: National Avian Influenza Wild Bird Surveillance Newsletter*, December 2018, https://wildlifehealthaustralia.com.au/Portals/0/Documents/ProgramProjects/N1_Wild_Bird_News_Dec_2018.pdf.

WHO (World Health Organization) (2007a). *Questions and answers on potential transmission of avian influenza (H5N1) through water, sanitation and hygiene and ways to reduce the risks to human health*, WHO Department of Public Health and Environment & WASH Inter Agency Group, WHO, Geneva, www.who.int/water_sanitation_health/emerging/AI_WASH_working_group_qas_april_2007.pdf.

WHO (World Health Organization) (2007b). *Control of avian influenza A (H5N1): public health concerns*, WHO, Geneva, www.who.int/influenza/resources/avian_influenza/en.

Wu Y, Wu Y, Tefsen B, Shi Y & Gao GF (2014). Bat-derived influenza-like viruses H17N10 and H18N11. *Trends in Microbiology* 22(4):183–191.

Yamamoto Y, Nakamura K, Yamada M & Mase M (2010). Persistence of avian influenza virus (H5N1) in feathers detached from bodies of infected domestic ducks. *Applied and Environmental Microbiology* 76(16):5496–5499.

Yee KS, Cardona CJ & Carpenter TE (2009). Transmission of low-pathogenicity avian influenza virus of subtype H6N2 from chickens to Pekin ducks and Japanese quail (*Coturnix coturnix japonica*). *Avian Pathology* 38(1):59–64.

Zhou N, He S, Zhang T, Zou W, Shu L, Sharp GB & Webster RG (1996). Influenza infection in humans and pigs in southeastern China. *Archives of Virology* 141:649–661.