

**Open Government Status:  
Partially Closed (Annexes  
1, 2 and 3 closed)**

## **Advisory Committee on Dangerous Pathogens**

### **Update on Avian Influenza**

#### **Issue**

1. To consider recent developments regarding avian influenza and comment on HPA proposals for delivering public health precautions.

#### **Background**

2. At the last ACDP meeting, members requested that the committee should consider Avian Influenza (AI), in particular non- H5N1 viruses, and appropriate public health measures. It is approximately a year since an *ad hoc* ACDP meeting was held to discuss the public health measures needed in the event of either low pathogenic AI (LPAI) or highly pathogenic AI (HPAI) being found in poultry or wild birds in the UK. Therefore, it would seem timely to review the recommendations made.

#### **Recent Developments**

##### LPAI and HPAI – risks to the UK

3. Members are provided with the following documents:

Annex 1 – HPA paper, “Proposed changes to HPA operational approach to avian influenza incidents”

Annex 2 – HPA paper, “Addressing the quantitative Microbiological Risk Assessment (QMRA) of the risks posed to the UK public health by highly pathogenic avian influenza H5N1”

Annex 3 – HPA final report on the Norfolk avian influenza incident

Annex 4 - Defra summary report of the Norfolk avian influenza outbreak

Annex 5 – Summary of the Defra wild bird survey (latest figures to be tabled on the 21<sup>st</sup>)

Annex 6 – Statement from ad-hoc ACDP meeting held on Friday 28<sup>th</sup> October (included for ease of reference).

## **Risk from pigs**

4. As members are aware, last year ACDP considered the precautions that should be taken by those working with pigs, given that pigs can be infected by some avian influenza viruses and are susceptible to both human and porcine influenza viruses.

5. ACDP agreed that the hypothesis that pigs act as the “mixing vessels” for influenza viruses from which a new pandemic human strain arises is no longer widely held to be correct, as there was little evidence that a flu strain dangerous to public health has been produced in this way. However, there is a theoretical risk that co-infection of the pig with avian influenza and human influenza could lead to re-assortment to produce a new strain of public health significance and a precautionary approach should be adopted. Members concluded that the Joint Committee on Vaccination and Immunisation (JCVI) should be asked to consider the use of seasonal flu vaccine for those in close direct contact with pigs as a public health measure to minimise the chance of recombination between seasonal flu, porcine or avian flu in pigs with transmission back to people.

6. The JCVI discussed this issue in June 2006 and the minutes recording the committee’s decision will either be tabled or verbally reported on the 21<sup>st</sup> September.

## **ACTION**

7. ACDP is asked to note the information given, and discuss as appropriate. In particular, ACDP is asked to consider the proposals for delivering health protection measures given in Annex 1.

ACDP/84/P5 Annex 1 (Closed)  
ACDP/84/P5 Annex 2 (Partially Closed)  
ACDP/84/P5 Annex 3 (Closed)

# **LOW PATHOGENIC AVIAN INFLUENZA H7N3 OUTBREAK IN NORFOLK, ENGLAND, APRIL – MAY 2006**

## **FINAL EPIDEMIOLOGY REPORT**

**THE NATIONAL EMERGENCY EPIDEMIOLOGY GROUP  
ANIMAL HEALTH AND WELFARE DIRECTORATE GENERAL,  
DEPARTMENT FOR ENVIRONMENT, FOOD AND RURAL AFFAIRS,  
1a PAGE STREET, LONDON, SW1P 4PQ**

## INTRODUCTION

1. This report describes the epidemiology of the outbreak of low pathogenicity avian influenza (LPAI) caused by an H7N3 strain, and the detailed investigations surrounding it. It is based on data available up to 22 May 2006. Restrictions applying to the LPAI 'Restricted Areas' were lifted on 26 May 2006.

2. Epidemiology is the study of the distribution, the determinants (risk factors) and causes of disease outbreaks. Epidemiologists, using sophisticated statistical analyses, field investigations, and complex laboratory techniques, investigate the cause of a disease, its distribution and method of spread, and provide advice on measures for control and prevention. It draws conclusions based on the evidence available and the balance of probabilities.

## EXECUTIVE SUMMARY

3. The following summary presents the best estimates for the timing and distribution of the LPAI cases detected in Norfolk, UK in April 2006. These estimates are based on the detailed epidemiological investigations that are described in the following report.

- Three holdings, consisting of two free range layer chicken flocks and one broiler breeder chicken flock were infected with LPAI type H7N3.
- No other evidence of LPAI infection was found in any associated premises, or any other potentially exposed premises within 3km of these holdings (206 holdings assessed, 169 visits carried out, 45 premises restricted and investigated further.)
- No evidence of H7N3 LPAI infection was found in any other domestic holdings or wild birds in the UK, that were investigated as a result of heightened surveillance during this period, or in the pre-existing targeted surveillance that has taken place over previous months.
- Infection is believed to have arrived at the first infected premises, LPAI 2006/02, between 17-20 March 2006. The source is believed to be wild birds, which have direct access to this outdoor flock.
- Infection is believed to have arrived at the second infected premises, LPAI 2006/03 between 2-5 April 2006. The source is believed to be LPAI 2006/02, with fomite transmission occurring through shared personnel or equipment.
- Infection is believed to have arrived at the third infected premises, and the first to be reported, LPAI 2006/01, between 15-18 April 2006. Based on the available evidence the source is believed to be LPAI 2006/03, with transmission possibly occurring by fox carriage of carcasses from LPAI 2006/03 to LPAI 2006/01, and introduction of virus to the housed birds through fomites spread on contaminated footwear.

- The results of the investigations suggest that awareness of the clinical signs associated with any AI virus by poultry keepers and the veterinary profession is paramount.
- The results of this investigation, together with those from a recent survey of poultry keepers, indicate that biosecurity measures should include preventing the predation of dead birds by free-living species such as foxes and advocate not practicing the deliberate feeding of foxes with poultry carcasses.

## **OUTBREAK - INFECTED PREMISES**

4. This section presents summary information about each of the premises identified as infected by virus isolation or serology.

### **Descriptive Overview**

5. Three commercial poultry premises were detected as infected in this outbreak. The index case is presumed, from the available epidemiological findings, to be the free range laying flock which was on LPAI 2006/02. The working hypothesis, based on the fact that other sources of infection were ruled out, is that infection was from wild birds, which are considered to be the natural reservoir for low pathogenic avian influenza (LPAI) viruses.

6. The second infected flock was at LPAI 2006/03, which was under the same ownership as LPAI 2006/02. This flock became infected from its sister flock at the peak of the clinical episode on LPAI 2006/02.

7. The third infected farm was LPAI 2006/01, on which a housed broiler breeder flock was maintained. The most plausible explanation, from the extensive epidemiological investigations, is that this flock was infected from the LPAI 2006/03 flock at the peak of its clinical outbreak. The working hypothesis as to the method of introduction, again having ruled out other possible means of between flock transmission, is the introduction of the virus via infected poultry carcasses or tissues from LPAI 2006/03 by the foxes resident within the boundary of LPAI 2006/01. Infection was most probably introduced into the poultry houses by contaminated footwear worn by the staff and most likely by the egg collecting team.

8. The epidemiological findings of this small outbreak have provided some valuable insights into the clinical disease associated with this H7N3 strain, the dynamics of infection within flocks and indications for improved control measures to minimise transmission. The details of the infected farms and the conclusions drawn are provided in the following sections.

**LPAI 2006/02**General

9. This farm, a component of a number of contiguous holdings under the same private ownership, is located in the centre of Norfolk, to the east of the town of Dereham. A free range layer flock is maintained on this premises and poultry have been kept here for over 20 years. The other holdings comprise a dairy herd, an arable unit and another free range layer flock of the same size (LPAI 2006/03, see below). The owner maintains seasonal (from June/July, for the shooting season starting in August) pheasant (1000) and partridge (250) populations, but in this respect the farms were unstocked with these species at the time of the outbreak, and subsequent epidemiological investigations in May.

Flock details and management

10. This free range layer flock was stocked with 8000 birds. It comprises a split slatted floor barn for housing with daytime access to an adjacent grassland area. The flock is housed at night. Water was provided by hanging rainbow drinkers inside the barn. At the time of the epidemiological investigation the chickens were 47 weeks of age, the site having been stocked when the chickens were about 18 weeks of age. These point of lay (POL) chickens were derived from chicks hatched in Great Britain. Although under private ownership, the POL chickens were derived from a layer-rearer associated with a large national company and egg collection was by this company.

Clinical History and Laboratory Findings

11. Clinical signs of disease were first observed in this flock on or around 25 March, and included a drop in egg production and a two-fold increase in mortality. There was also evidence of cannibalism.

12. The owner's private veterinary surgeon was consulted on 29 March and carcasses of affected birds were submitted for autopsy at the veterinary practice's laboratory. The post mortem examination revealed evidence of egg peritonitis and the clinical signs were attributed to a power failure in the preceding week.

13. Suspect avian notifiable disease was reported on this farm, retrospectively, on 27 April as LPAI infection had recently been detected on a farm (LPAI 2006/01 see below) some 3.5km away. As a result, the flock was investigated by the State Veterinary Service (SVS), receiving a visit by a Veterinary Officer on 28 April. Cannibalism was the only clinical abnormality observed. Twenty blood samples, for serology, and 20 cloacal swabs for virological examination were taken.

14. An examination of the production and mortality records indicated that there was a coincident reduction in egg production and an increased mortality rate in the week beginning 22 March (Table 1). The peak in the mortality occurred in the "week" commencing 29 March when it was approximately double the normal rate. Egg

production was at the lowest level during this week. Both egg production and the mortality rates returned to near pre-clinical episode levels in the week beginning 5 April.

**Table 1: Weekly egg production (%) and mortality rates at LPAI 2006/02 from 1 March to 2 May 2004**

"WEEK" COMMENCING	EGG PRODUCTION (%)	MORTALITY RATE (birds/1000/week)
01/03/2006	91.2	3.8
08/03/2006	89.5	2.8
15/03/2006	90.9	4.2
22/03/2006	85.5	6.3
29/03/2006	81.4	7.9
05/04/2006	84.5	3.4
12/04/2006	85.6	3.2
19/04/2006	85.6	2.2
26/04/2006	85.5	0.4

15. The M-gene PCR test which detects influenza type A virus, conducted on the cloacal swabs, was negative.

16. Serological testing revealed a prevalence of 75% (15 positives out of 20) chickens serologically positive to H7 strain avian influenza (Table 2).

17. Further samples were obtained on 30 April when the culling of the flock commenced. Ten carcasses and 100 blood samples were collected. Fifty-seven per cent of the birds sampled were seropositive (Table 2). Virological examination of tissue from the ten carcasses, by M-gene PCR and inoculation of embryonated eggs, failed to isolate any AI virus.

INTERPRETATION	Avian Influenza HI TITRE	No. SAMPLES with titre (taken on 28 April)	No. SAMPLES with titre (taken on 30 April)
NEGATIVE	0	0	0
	1	0	4
	2	1	13
	3	4	26
POSITIVE	4	5	20
	5	6	17
	6	3	6
	7	0	8
	8	0	2
	9	1	2
	10	0	2
	11	0	0
	12	0	0
TOTAL		20 (75%)*	100 (57%)*

\* % samples positive

**Table 2: Results of serological testing at LPAI 2006/02 on 28 and 30 April**

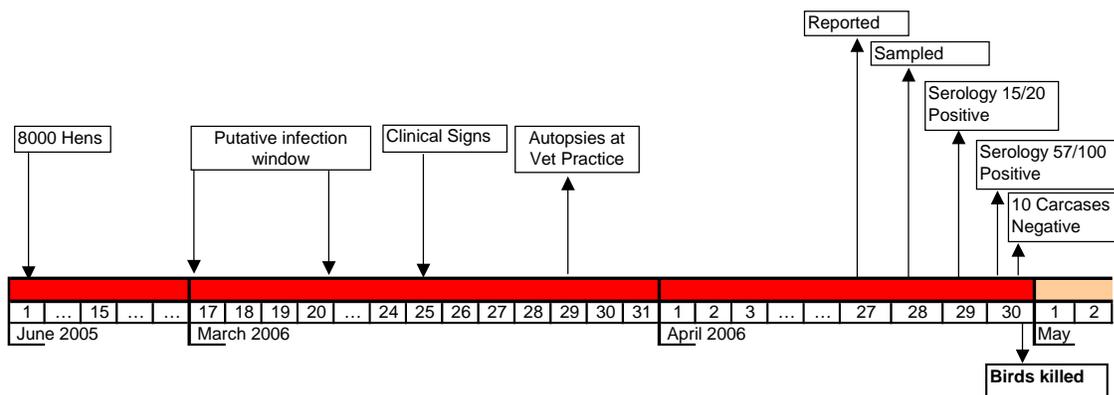
18. There was no statistically significant difference between the distribution of seroconversion titres observed at the two sampling dates (Fishers Exact  $\chi^2$   $p = 0.53$ ). Similarly there was no statistically significant difference between the prevalence of seropositive birds observed at the two sampling dates (Fishers Exact  $\chi^2$   $p = 0.34$ ).

### Estimated Date of Infection

19. Assuming that clinical signs were associated with infection and the incubation period ranges from 2 – 5 days then the likely date of infection was between 17 and 20 March. A summary of the events on the farm is given as a timeline in Figure 1.

**Figure 1**

**TIMELINE FOR LPAI 2006/02**



### **Investigations for potential source and/or spread**

20. Investigations for source and spread of infection are described as ‘tracings’, and are based on the estimated incubation period, statutory requirements and period over which virus is excreted by infected birds.

21. Source tracings were assessed from 2 - 21 days prior to first clinical signs; spread tracings were assessed from 5 days prior to first clinical signs until the imposition of movement restrictions, OR where there was suspicion of risk arising outside this window. Tracings visits were carried out for contacts considered significant risk (for example where personnel associated with infected farms were likely to have entered other poultry premises), and restrictions imposed and samples taken on premises at which the visit confirmed the possibility of exposure of farm poultry to infection.

22. Because of their common ownership and shared personnel, vehicles and equipment, LPAI 2006/02 and LPAI 2006/03 were treated as a single unit for tracings

purposes. *For the two premises together*, a total of 102 tracings were assessed, 58 tracings visits carried out, and five premises placed under restriction and sampled.

#### Description of source and spread risk factors

23. The usual epidemiological investigations on source and spread factors revealed in particular:

- abundant wildlife (avian and mammalian) in the area (see Paragraphs 52 and 53 for more detail)
- movement of personnel, vehicles and equipment between this and the nearby LPAI 2006/03 (also a free-range poultry unit, see below) in the same ownership
- the owner carries out work for other farmers, typically removing manure and used bedding to be spread on his own land or elsewhere
- regular visits to the premises by contractors, some with apparently sub-optimal record-keeping and/or biosecurity standards and some of whom frequently visit other livestock premises in the area to deliver feed, collect eggs and animal by products, or to carry out routine maintenance or pest control

#### **LPAI 2006/03**

##### General

24. As indicated above this farm is under the same ownership as LPAI 2006/02 (see paragraph 6), and lies approximately 1km to the east.

##### Flock details and management

25. This is the same as on LPAI 2006/02, described in paragraph 7.

##### Clinical History and Laboratory Findings

26. Suspect avian notifiable disease was reported on this farm at the same time, 27 April, as the report of disease on LPAI 2006/02 (see paragraph 10). At the time of the Veterinary Officer visit on 28 April egg production was reduced and there was evidence of cannibalism.

27. An examination of the production and mortality records indicated that the mortality rate increased in the “week” beginning 4 April, some two weeks after the onset of clinical signs on LPAI 2006/02. Egg production was reduced in the “week” beginning 11 April (Table 3). During this week the peak in the mortality rate occurred resulting in a threefold increase of the normal rate. The maximum mortality rate was greater (12.9 deaths per 1000 birds per week) than that observed at LPAI 2006/02 (7.9 deaths per 1000 birds per week).

"WEEK" COMMENCING	EGG PRODUCTION (%)	MORTALITY RATE (birds/1000/week)
28/03/2006	93.0	4.0
04/04/2006	92.4	7.1
11/04/2006	79.8	12.9
18/04/2006	79.9	6.7
25/04/2006	81.1	3.0

**Table 3: Weekly egg production (%) and mortality rates at LPAI 2006/03 from 28 March to 1 May 2004**

28. Twenty blood samples, for serology, were taken on 28 April together with 20 cloacal swabs and four carcasses for virological examination.

29. The M-gene PCR, conducted on 29 April, revealed evidence of AI infection in samples from the four carcasses, but the cloacal swabs were negative. The intravenous pathogenicity index (IVPI), using 6 week old poults was 0.0.

30. Serological testing revealed a prevalence of 45% (9 positives out of 20, Table 4)

31. Further samples were obtained on 30 April when the culling of the flock commenced. Ten carcasses and 100 blood samples were collected.

32. The tissues from the ten carcasses were negative on examination by the M-gene PCR and virus was not isolated by egg inoculation.

33. The prevalence of seropositive birds was 96% (96 out of 100) (Table 4). The distribution of seroconversion titres between the two farm visits was significantly different (Fishers Exact  $\chi^2$   $p < 0.001$ ).

OUTCOME	TITRE	No. SAMPLES (taken on 28 April)	No. SAMPLES (taken on 30 April)
NEGATIVE	0	0	0
	1	1	0
	2	1	1
	3	9	3
POSITIVE	4	4	17
	5	4	30
	6	1	23
	7	0	8
	8	0	6
	9	1	6
	10	0	5
	11	0	0
	12	0	1
TOTAL		20 (45%)*	100 (96%)*

\* % samples positive

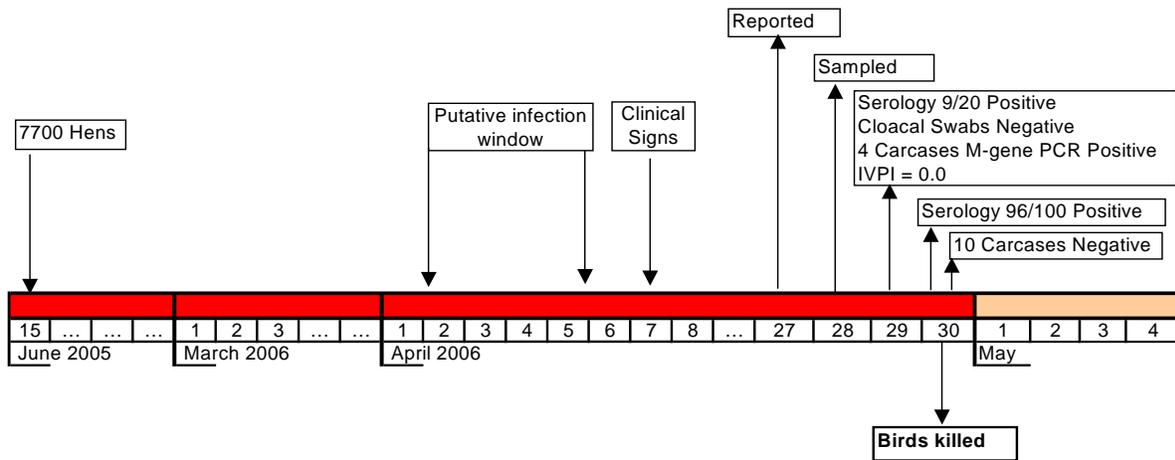
**Table 4: Results of serological testing at LPAI 2006/03 on 28 and 30 April**

Estimated Date of Infection

34. Assuming that clinical signs were associated with infection and the incubation period ranges from 2 – 5 days then the likely dates of infection are between 2 and 5 April. A summary of the events on the farm is given as a timeline in Figure 2.

**Figure 2**

**TIMELINE FOR LPAI 2006/03**



Investigations for potential source and/or spread

35. As described above, LPAI 2006/02 and LPAI 2006/03 were treated as a single unit for tracings purposes. *For the two premises together*, a total of 102 tracings were assessed, 58 tracings visits carried out, and five premises placed under restriction and sampled.

36. The usual epidemiological investigations on source and spread factors revealed, the same risk factors as for LPAI 2006/02, see Paragraph 19.

**LPAI 2006/01**

General

37. This farm, one of 34 poultry premises belonging to a partly integrated company based in Norfolk, lies in the centre of Norfolk, to the east of the town of Dereham. The sheds housing the broiler breeder flock were established between 7 and 8 years ago. No other livestock are kept on the site.

### Flock Details and Management

38. The flock comprises some 31,500 broiler breeders that are housed in four sheds linked by a common egg collection passage. Each shed provides a partly slatted/deep litter environment in which birds can move throughout, and has approximately 800 nipple drinkers. The affected birds were 32 weeks old at the time of the report, and were imported as day old chicks from France in September 2005.

39. Of note is that the egg store is situated between Sheds 2 and 3 (where clinical signs first appeared); egg collections occur every 4 days (14 and 18 April in the high risk period). These involve the doors to the egg store (and so common corridor) being open for about an hour allowing potential fomite access from the apron by wind, birds or personnel.

### Clinical History and Laboratory Findings

40. LPAI 2006/01 was the first farm on which evidence of avian influenza was detected in this outbreak. Clinical signs were first noted on 20 April 2006 in Shed 2, on the 21 April in Shed 3, on the 22/23 April in Shed 1, and on 24/25 April in Shed 4. These signs included a drop in egg production and quality, closely followed by marked anorexia (evidenced by a drop in feed consumption), and a fourfold increase in the mortality rate.

41. Suspect avian notifiable disease was reported on this farm in the evening of 25 April 2006 by the private veterinarian, after their investigations to date have proved negative for others causes. As a result, the flock was visited by a Veterinary Officer on 26 April. At that time the increased mortality and reduced egg production were still evident in Sheds 1 and 4, however birds in Sheds 2 and 3 were recovering. Twenty blood samples, for serology, and 20 cloacal swabs for virological examination, were taken.

42. Examination of the production and mortality records indicated that there was a reduction in egg production in Sheds 2 and 3 from the 20 April, with a peak in mortality in these two houses on the 23 and 24 April respectively. There was no clear evidence of a recovery in either production or mortality rates by the time of slaughter on the 27 April (Figures 3 and 4).

Figure 3 Daily mortality rate in each house at LPAI 2006/01, 19 to 26 April 2006

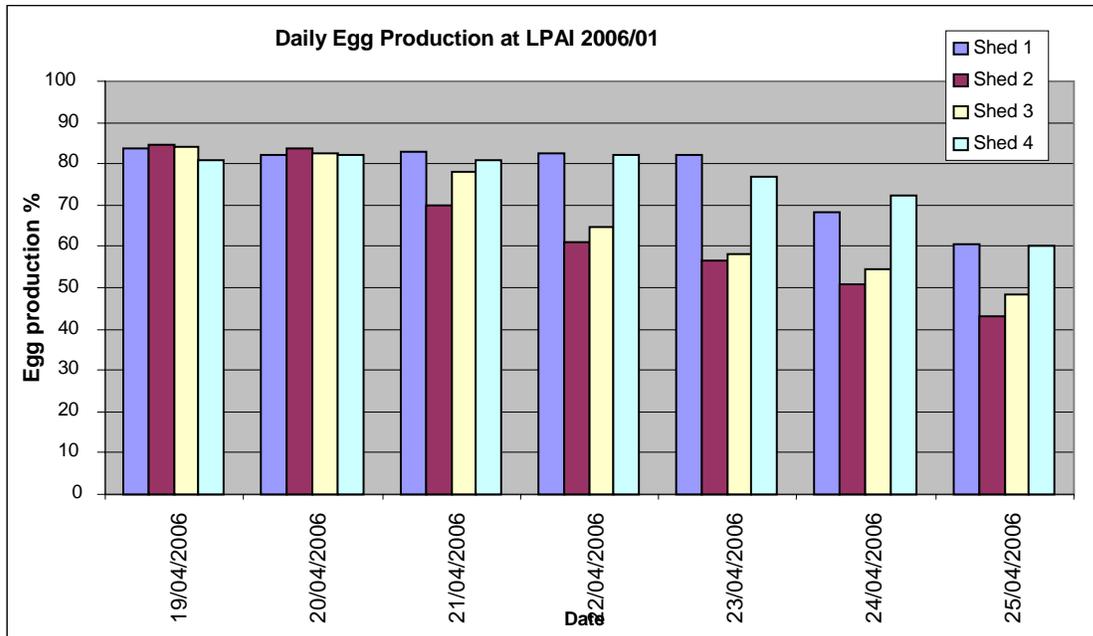
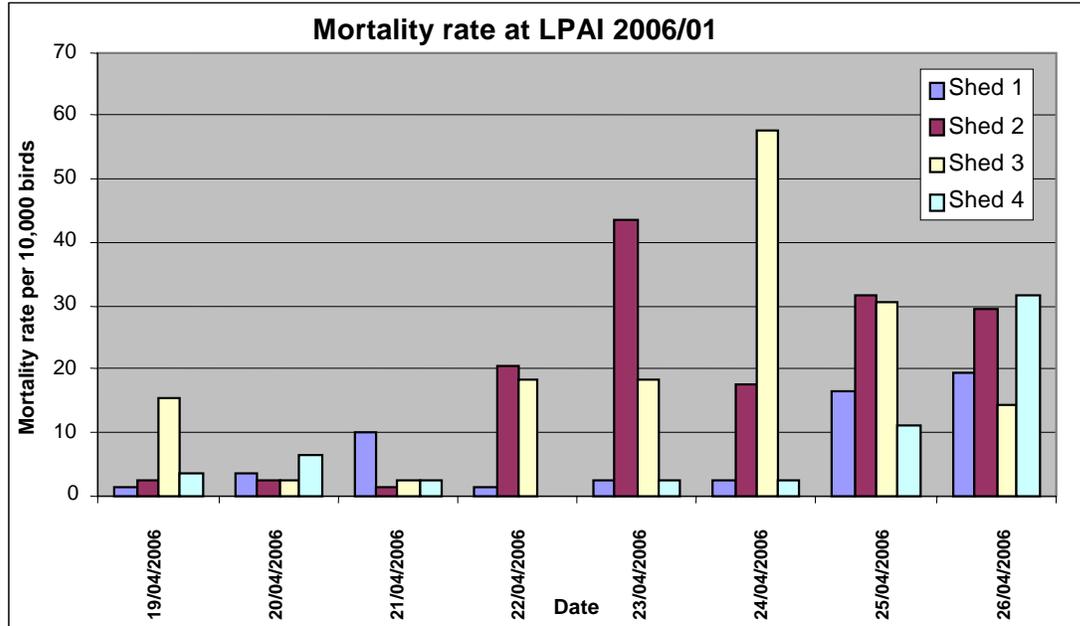


Figure 4 Egg production (%) at LPAI 2006/01, 19 to 25 April

43. Initial evidence of viral infection with the H7 strain was found in tissue samples from carcasses and cloacal swabs from birds in Sheds 1 and 4 taken on 26 April. At this time birds in these sheds were serologically negative for H5 and H7, however three serum samples from 20 birds in Shed 2 were positive for H7 antibodies.

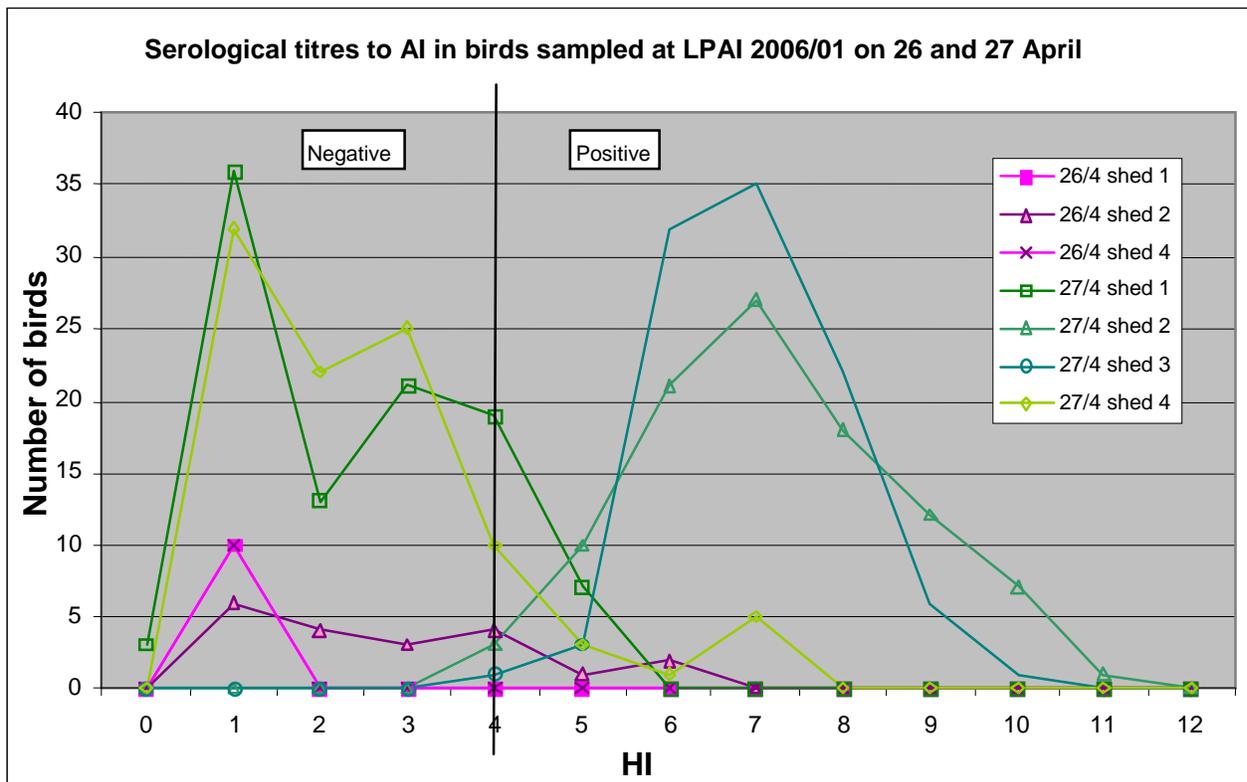
44. Further laboratory examinations revealed the presence of LPAI virus H7N3. The pathotype was assessed as LP on the basis of genetic sequencing, and the intravenous pathogenicity tests confirmed this as a low pathogenic strain with an IVPI of 0.22.

45. At the time of culling, on 27 April, ten carcasses and blood samples from 100 birds from each Shed, were taken for laboratory examinations.

46. The results of the serological (HI) examinations are summarised in Figure 5 and show that by 27 April the prevalence of seropositives in the first two Sheds affected (2 & 3) was 100%, whereas in Sheds 1 & 4, which were most likely infected some 3 to 5 days later, the sero-prevalences were 25.3% and 19.4%, respectively. The high (100%) prevalences were associated with a greater proportion of birds with greatest titres. These results provide strong evidence for the observed clinical signs being associated with LPAI infection.

47. There was a significant difference in the distribution of seroconversion titres in House 2 between the first and second farm visits (Fishers Exact  $\chi^2 < 0.001$ ), this was the only house sampled that was positive at both visits (see Figure 5). The proportion of high titre samples was much greater at the second visit. Samples from Shed 3 were tested by HI on 27 April when all were positive.

**Figure 5: Serological titres to AI in birds sampled at LPAI 2006/01 on 26 and 27 April (titres of 4 and above are considered positive)**



48. Experimental evidence indicates that once exposed seroconversion by HI is generally not detected until 6 or more days post-exposure, with all birds becoming positive by 10 days. The evidence presented therefore suggests that following infection the transmission throughout the flock in Sheds 2 and 3 continued so that by 7 days post- onset of clinical disease 100% of birds had become infected and were seropositive.

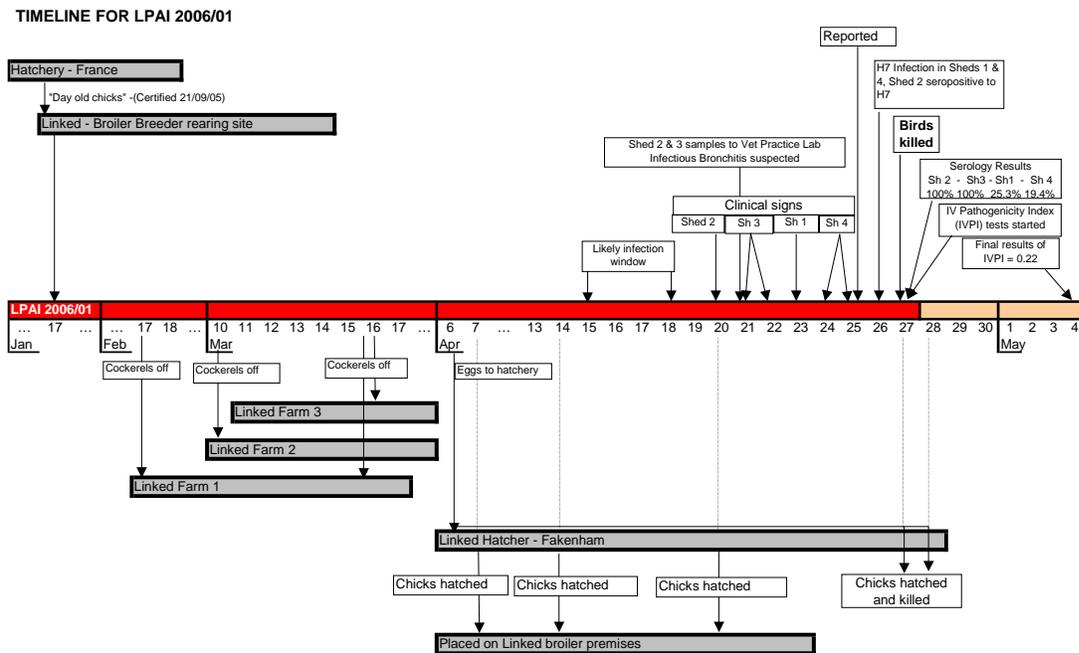
49. This would suggest that Sheds 2 and 3 were infected on or around 18 April. It is not possible to say whether Sheds 2 or 3 was infected first. Excretion of virus from birds in these houses would likely be greatest between 21– 25 April.

50. Seroconversion appeared to start between the two visits in Sheds 1 and 4 as no seroconversion was detected in the 20 samples collected from each at the visit on 26 April, indicating a maximum possible prevalence below 15%. However 26% and 19% of the 100 samples from each of these flocks respectively were positive on 27 April. These findings indicate that these houses became infected on or around 21 April, which would be consistent with transmission of virus from Shed 2 or 3.

Estimated Date of Infection:

51. Assuming that clinical signs were associated with infection and the incubation period ranges from 2 – 5 days then the likely dates of infection are between 15 and 18 April. A summary of the events on the farm is given as a timeline in Figure 7.

**Figure 6**



Investigations for potential source and/or spread:

52. A total of 104 tracings were assessed for LPAI 2006/01, 111 tracings visits carried out, and 40 premises placed under restriction and sampled. This number of tracing visits is greater than the number of tracings assessed due the linked sites within the pyramid being visited more than once.

Description of source and spread risk factors

53. Detailed epidemiological investigations on the site revealed the following potential risk factors:

- there is an active fox earth within the perimeter of the site, close to the poultry houses
- while wildlife are not as much in evidence as in the area around the free-range units at LPAI 2006/02 and LPAI 2006/03, and wild birds are seen on the premises relatively infrequently, contamination of the concrete apron by infected droppings from wild birds is still a possibility
- regular visits to the premises by contractors to deliver feed, carry out pest control, or carry out repairs
- regular visits by staff to carry out routine maintenance, and lorries to take hatching eggs from the site to the company hatchery
- while employees are normally exclusive for the site and footbaths are in evidence, the office/changing room has a single entrance/exit, giving the possibility of cross-contamination between visitors from the outside and staff exiting the poultry houses
- a neighbour's dogs regularly enter the site via the earth bank on the north side and have been known to remove carcasses

**Description of wildlife on/around IPs**

54. All three IPs are situated in a rural lowland area with abundant wildlife. Many avian species are in evidence, especially on LPAI 2006/02 and LPAI 2006/03, where the numerous small ponds and free-range nature of the enterprise attract significant numbers of waterfowl (especially mallards and Egyptian geese); there are also large active rookeries on both of these premises, doves (and possibly swallows) nesting in the farm buildings at LPAI 2006/02, and a variety of other wild species (moorhens, sparrowhawks, owls, skylarks, game species, etc.) to be seen. While LPAI 2006/01 abuts an area of scrub woodland, and mallards and pheasants can be seen in close proximity to the site, wild birds are rarely seen near the houses.

55. There is abundant evidence of badger, deer and fox activity in the area. Of particular interest is the active badger sett and runs close to LPAI 2006/03, the finding of fox droppings and evidence of bird fox kills around the farm buildings at LPAI 2006/02, and the active fox earth inside the perimeter of the LPAI 2006/01 site close to the poultry houses, on the inside of the earth bank surrounding the premises. Information received from colleagues at the Central Science laboratory (CSL), York suggests that adult foxes are likely to be feeding six week-old cubs at the time of this outbreak and to range across approx 2 km<sup>2</sup> in a lowland rural setting like this. Whilst the nearest of the infected free-range units is (at 3km from LPAI 2006/01) outside this range, the same report advised that foxes 'will be likely to repeatedly visit reliable sources of food', and therefore the possibility that the LPAI 2006/01 foxes may have taken birds from the LPAI 2006/03 unit cannot be ruled out.

## **SURVEILLANCE ASSOCIATED DIRECTLY WITH THE OUTBREAK**

### **Surveillance Activities Associated with LPAI 2006/01**

56. This farm was part of a medium sized (in the context of GB poultry companies) poultry company comprising 37 premises owned by the company or contracted farmers:

- 1 hatchery producing ~170,000 broiler chicks per day
- 11 broiler breeder sites (10 stocked), each containing up to 35,000 chickens
- 4 rearing sites (3 stocked), each containing ~35,000 birds, reared from day old to 18 weeks of age
- 21 broiler sites

### **Broilers**

57. The birds on each broiler site were subject to a clinical examination, involving a veterinary inspection of the birds and the production and mortality records. No abnormalities were identified. One site reported abnormal mortality and this flock was formally reported as a suspect case of avian notifiable disease. Samples were taken for serological and virological examination, according the protocol indicated in the following paragraph. The results were negative for AI virus

### **Broiler Breeders**

58. These premises were identified for the initial targeted surveillance involving the collection of samples for laboratory testing. This was because it was considered that the greatest frequency of movement of company staff, mainly egg collectors and farm management staff, occurred between these flock types. There was therefore a greater risk of transmission having occurred between these premises. The surveillance consisted of:

- A clinical inspection of the chickens at each site for signs of disease typical of avian influenza
- An examination of the egg production records, water and feed intake and daily mortality rates at each site
- Blood sampling of 20 birds, from each epidemiological group within the flock(s) at each site, for serological (HI) testing
- Cloacal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR
- Oro-pharyngeal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR

59. This surveillance was completed on 30 April. One farm had experienced an egg drop problem but this had been attributed to infection with Infectious Bronchitis virus. These chickens and those on the other nine farms were all negative on serological testing and virological examination.

### **Rearing farms**

60. The three populated premises were subject to surveillance visits by State Veterinary Service (SVS) veterinary officers. This surveillance comprised:

- A clinical inspection of the chickens at each site for signs of disease typical of avian influenza
- An examination of the records of water and feed intake and daily mortality rates at each site
- Blood sampling of 20 birds, from each epidemiological group within the flock(s) at each site, for serological (HI) testing
- Cloacal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR
- Oro-pharyngeal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR

61. No evidence of infection with any AI virus was detected on these premises.

### **Commercial Premises in the 1km Statutory Restricted Zone around LPAI 2006/01**

62. In accordance with national and EU statutory requirements commercial poultry premises were sought by interrogation of the Great Britain Poultry Registry (GBPR) (the GBPR requires the statutory registration of premises containing 50 or more birds kept for commercial purposes) and by “foot patrols” by local staff of the SVS.

63. As a result one premises, containing 100 laying chickens was identified within the statutory restricted zone. The surveillance on this premises consisted of:

- A clinical inspection of the chickens at the premises for signs of disease typical of avian influenza
- Blood sampling of 20 birds for serological testing
- Cloacal swabbing of 20 birds for testing by the M-gene RT-PCR
- Oro-pharyngeal swabbing of 20 birds for testing by the M-gene RT-PCR

64. This flock was subject to two veterinary visits at which samples were taken, the last visit and sampling was on 18 May. All findings, clinical and laboratory tests, on the chickens were negative for AI virus.

### **Surveillance Activities Associated with LPAI 2006/02**

65. A consideration of what further surveillance was appropriate, whilst the results were awaited from the surveillance of broiler breeder farms owned by the parent company of LPAI 2006/01, was made by the National Expert Group. This Group recommended surveillance of all premises containing > 50 birds kept outdoors comprising one or more of the following species: chickens, turkeys, geese and ducks within 3Km of LPAI 2006/02. LPAI 2006/02 was chosen as the epicentre for this surveillance as at this time one of the working hypotheses was that it was the index case and that there was a high probability that it had become infected from wild birds. The objective was therefore to determine whether other potentially at risk (*i.e.* outdoor maintained flocks) had also become infected without a notable incident of clinical disease.

66. A search of the GBPR database identified 12 premises which maintained chickens, turkeys, ducks or geese outdoors. Nine of these premises were found to be stocked when visited, and the poultry were inspected and sampled. An additional relatively large outdoor flock outside the 3km radius, to the south-west of LPAI 2006/02 was also sampled. A summary of the species present on these premises is:

- Four premises had only chickens. The average size of these premises was 2148 but only two of the premises had over 1000 birds.
- Two premises had geese and chickens only; the average number of birds in these premises was 47.
- Two premises had ducks only (one is a duck hatchery). The average number of ducks in these premises was 6600 and the smallest one had 3000 ducks.
- One premises had only chickens and ducks, with a total of 77 birds on the premises.
- One premises had chickens, geese, ducks and aviary birds, with a total of 220 birds on the premises.

67. The predominant kept species at the time of this surveillance (May) within a 3km radius of LPAI 2006/02 were ducks (13254), chickens (8787), game birds (100),

geese (83) and aviary birds (60). (This confirms the relatively low density of premises keeping birds, and of the low number of birds in this area.)

68. These ten premises were subjected to the following surveillance:

- A clinical inspection of the birds at each site for signs of disease typical of avian influenza
- Blood sampling of 20 birds, from each epidemiological group within the flock(s) at each site, for serological testing
- Cloacal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR
- Oro-pharyngeal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR

69. There was no evidence of infection with AI virus on any of these premises.

Surveillance, involving sampling, as a result of tracings associated with the three infected premises

70. As indicated in the earlier sections tracing activities resulted in 45 premises being restricted and examined and sampled using the following protocol:

- A clinical inspection of the chickens at each site for signs of disease typical of avian influenza
- Blood sampling of 20 birds, from each epidemiological group within the flock(s) at each site, for serological testing
- Cloacal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR
- Oro-pharyngeal swabbing of 20 birds, from each epidemiological group within the flock(s) at each site, for testing by the M-gene RT-PCR

71. No evidence of AI virus infection was found on these 45 premises.

Wild Bird Surveillance on and around the three infected farms

72. Local veterinary staff and scientific (including mammalian and avian ecologists) have been investigating these three farms. They were instructed to submit any dead wild birds found in the area for laboratory examination to supplement the national ongoing surveillance of wild bird surveillance. This only resulted in the submission of one moorhen, one pheasant and a domestic cockerel. The last two of these were road traffic fatalities. All have proved negative for infection with AI virus. (The relatively small number of birds obtained probably reflects fox activity in this area, see Paragraph 53)

**OTHER SURVEILLANCE FOR AI VIRUS INFECTION IN WILD AND DOMESTIC BIRDS IN THE COUNTY OF NORFOLK**

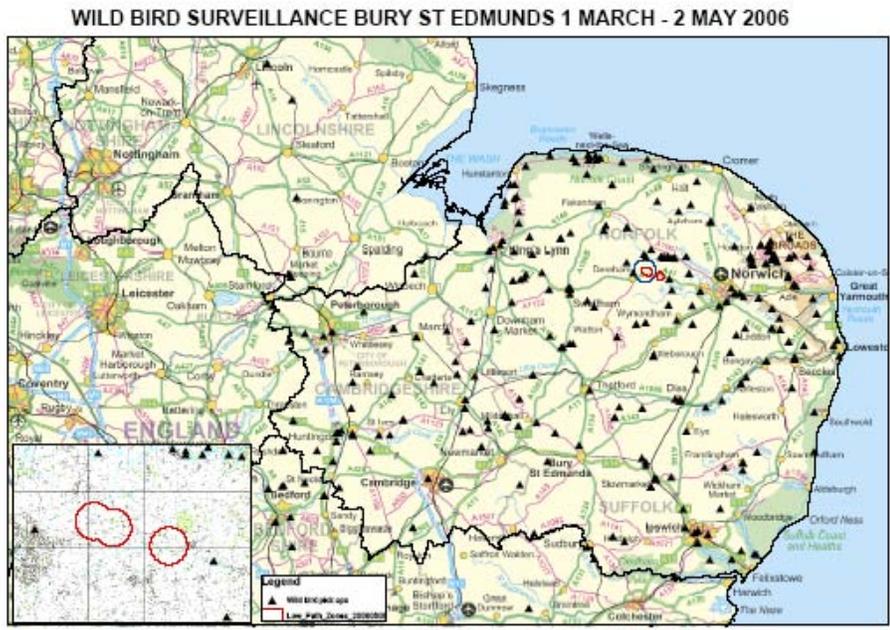
**Wild Birds**

73. As part of the national wild bird surveillance in operation in the UK, and other EU Member States, dead birds are submitted for laboratory examination. In the area under the jurisdiction of the Bury St Edmunds Animal Health Divisional Office 411 birds were examined from 1 January 2006 to 30 April 2006 (Table 5).

SPECIES GROUP	NUMBER SAMPLED
Cormorant	1
Duck	192
Goose	35
Gull	25
Other	22
Partridge	1
Pigeon	14
Rook	3
Swan	118
Total	411

**Table 5: Number and species group of dead wild birds examined for AI virus in East Anglia, 1 January 2006 to 30 April 2006**

74. The geographical distribution of these birds is shown in Figure 7. All were negative for AI virus.



**Figure 7: Source location of wild bird carcass submissions examined for the presence of avian influenza virus (inset shows locality immediately around IPs on a larger scale)**

## Domestic Birds

### Abattoir surveillance

75. The possibility of detecting undisclosed disease through abattoir surveillance was explored. LPAI can cause grossly visible kidney lesions in affected birds, so the possibility of raising awareness among abattoir inspectors, to establish an alert system for possible AI through the detection of flocks affected in this way, was considered. However on discussion with poultry specialist vets and pathologists this was ruled out. The kidney lesions are non-specific, and nephrotoxicity is common in older birds at slaughter, from a range of conditions. In the case of LPAI 2006/01, on necropsy few of the birds examined had the lesions. Thus such surveillance is unlikely to help find undisclosed disease, as the lack of specificity may lead to false alerts, and the lack of sensitivity could give a false reassurance.

76. As a possible alternative, the use virological examinations in abattoirs was considered. Details of the throughputs, species and source premises were available for the abattoirs in Norfolk in the network database. This has been assembled for epidemiological analyses for contingency planning and during the course of an outbreak of avian notifiable disease. Analysis of these data indicated that sampling at these abattoirs would not be efficient, as at most only 5 premises in Norfolk would be represented per week at the largest abattoir. Sampling at abattoirs was not therefore selected as a means of surveillance.

### Annual LPAI survey of domestic poultry

77. The possibility of undisclosed disease in Norfolk or elsewhere in the country, prior to the detected outbreak was explored. The results from the annual countrywide (UK) survey, required by the EU, for evidence of LPAI in poultry flocks that was completed in January were examined. This showed that all 450 holdings surveyed gave negative results in tests for the presence of avian influenza. Forty-three of these holdings, which included both breeding and layer fowl, breeding and fattening turkeys, ducks and geese were in Norfolk.

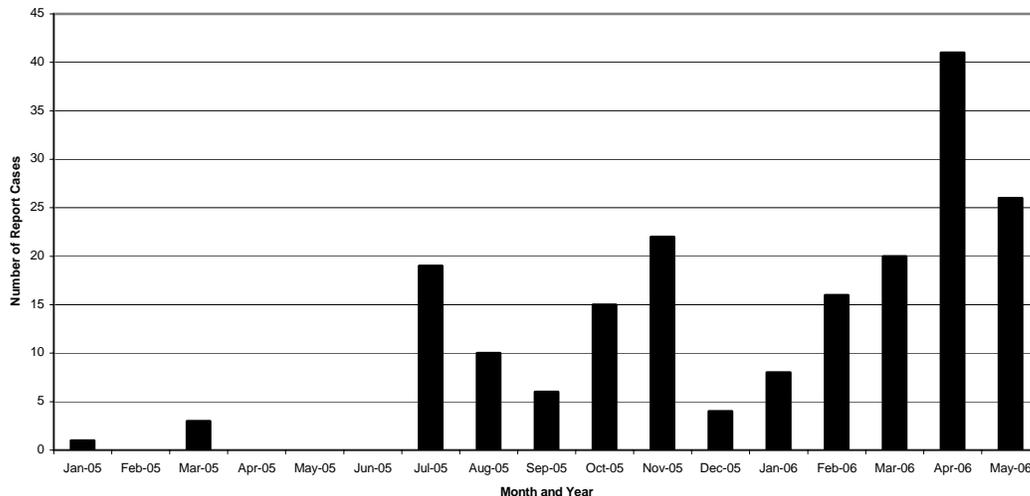
## Human Surveillance

78. The possibility that clinical illness in people might act as an indicator of undisclosed disease was considered. Communications were established with the medical authorities to ensure any such cases were brought to Defra attention and investigated (Paragraphs 98 to 108 describe this in more detail).

## AVIAN NOTIFIABLE DISEASE SURVEILLANCE IN GREAT BRITAIN

79. Figure 8 shows the number of report cases of notifiable diseases of poultry, recorded by Defra, that were received between January 2005 and the end of April

2006. These cases were reported in domestic birds; surveillance for notifiable diseases in wild birds is described in paragraphs 71 and 72.



**Figure 8: Monthly number of report cases of notifiable poultry diseases in GB, from 1 January 2005 to 19 May 2006.**

Source: DEFRA website accessed 19 May 2006

80. The graph presented in Figure 8 shows a considerable monthly increase in report cases following the confirmation of Newcastle disease (ND) in pheasants on one estate in July 2005, the first report case since March of that year.

81. It can be suggested that this phenomenon is, in part, due to increased vigilance following the ND outbreak and increased public concern surrounding a possible pandemic of influenza potentially originating from AI. The possibility of increased vigilance as a cause is supported by the initial observations that many of the report cases following the outbreak were recorded in game-birds and that the numbers of game known to be kept increases substantially from June onwards in preparation for the shooting season beginning on 12 August. (Further analyses of these data, stratified by species, is not currently feasible due to the structure and format in which the data is collated.)

82. In addition to the three infected premises described in this report, during the period of the outbreak (26 April to 8 May) there were 10 report cases of suspect notifiable avian disease in Norfolk. Seven of these were negated on clinical inspection and the remaining three were negated following restriction, inspection and laboratory testing.

## ANALYSIS OF EPIDEMIOLOGICAL FINDINGS

**Table 6: Summary information about affected and at risk holdings**

Infected Premises	LPAI 2006/01	LPAI 2006/02	LPAI 2006/03
Flock type	Housed broiler breeder	Free range layer	Free range layer
Report date	25 April 2006	27 April 2006	27 April 2006
Estimated infection date	15-18 April 06	17-20 March 06	06-09 April 06
Total risk period for tracings ( <i>earliest infection date to date of slaughter</i> )	30 Mar - 29 Apr 06	01 Mar - 01 May 06	21 Mar - 01 May 06
Total Susceptible Stock	31,500	7,572	7,260
Number of tracings assessed	104	102	
Number of tracing visits carried out	111*	58	
Number of holdings on which suspicion of possible exposure lead to imposition of restrictions + sampling	40	5	
Number of holdings estimated infected by this premises	0	1	1

\*some sites visited more than once

### THE SOURCE OF INFECTION

83. The results of the tracings and associated targeted surveillance involving laboratory examinations failed to identify any infected domestic flocks other than the three flocks described above.

84. Our conclusion is therefore that the flock at LPAI 2006/02 was the first to be infected, given the clinical and laboratory findings on all three farms. In the absence of another source of infection, the birds in this flock are most likely to have acquired infection from local wild birds. The natural reservoir of LPAI virus strains is wild birds and the area surrounding the farm was populated by a wide range of species. No evidence of infection was found in the three free living birds examined, but there was apparently a high level of fox predation which reduced the number of birds available for examination

85. There were no other similarly managed domestic flocks in the area to formally examine potential risk factors that could have predisposed the flock at LPAI 2006/02 to infection. It is possible that this infection was an unfortunate, rare and random event.

86. How infection gained access to the domestic birds remains unknown and will remain so.

#### TRANSMISSION OF INFECTION BETWEEN THE FLOCKS

87. The most likely source of infection for the LPAI 2006/03 flock is the LPAI 2006/02 flock, which was under the same management, rather than another infection from wild birds. The flocks had common personnel and therefore there was opportunity for transmission between the two flocks. There are two observations from the investigations that support this inter-flock transmission.

88. The first is that the most likely period when the LPAI 2006/03 became infected was from 2 April – 5 April. This coincided with the peak mortality in the LPAI 2006/02 flock when the virus load on the premises was likely to be at a peak.

89. The second observation is that the mortality rate and the reduction in egg production were greater in the LPAI 2006/03 flock suggesting the virus had gained in virulence after its passage in chickens.

90. There is no evidence of transmission of infection from the other two farms to the LPAI 2006/01 flock by personnel, vehicles or other fomites. It is likely that at the time of infection of the LPAI 2006/01 flock (15 – 18 April), virus excretion in the flock at LPAI 2006/02 was at a very low level or had ceased altogether. However at this time (15 – 18 April) the mortality rate was at a peak at LPAI 2006/03, when the virus load on the premises was greatest.

91. Epidemiology is a science which draws conclusions based on the available evidence, and the balance of probabilities. By ruling out other options and in the absence of any other findings, we have concluded that the most likely series of events in the transmission of infection from the LPAI 2006/03 flock is as follows.

92. The adult foxes resident at the LPAI 2006/01 site carried dead chickens from LPAI 2006/03 to their earth as a feed supply for the cubs and themselves. This would have resulted in the environment within the site, especially around the fox earth, becoming contaminated. The coincidence with the peak in the mortality rate at LPAI 2006/03 and most likely time of infection is notable. The number of birds dying may simply have provided a plentiful and easily obtained feed supply.

93. With infection having been introduced to the site the normal biosecurity measures are not likely to have prevented the introduction of infection into the poultry houses. An examination of the records of visitors to the site within the period the flock became infected suggest a number of possibilities for infection being introduced to the building. A maintenance visit, by a company member of staff, was made on 17 April. The person would have entered the building using the normal entrance used by the staff on site. This was opposite Shed 2, in which clinical signs were first observed. Two further possibilities involve the egg collectors. Egg collection is every

four days and during the period of interest occurred on 14 and 18 April. This involves opening the double doors to the central part of the building (between Sheds 2 and 3) where the egg store is located. This is to facilitate access for the egg trolleys. The process takes an hour to complete.

94. These visits therefore seem to have provided a means of introduction of the virus on footwear. Transmission to other premises should have been prevented by the biosecurity measures in place. This is clearly the case as the extensive surveillance conducted indicates that none of the other premises within the company became infected.

95. The size of the ultimate outbreak was undoubtedly limited because of the relatively low density of flocks in the area. This would have reduced the chances of transmission by the local, fox mediated transmission suggested. Also, normal biosecurity measures are likely to have prevented more distant transmission of infection to other flocks from LPAI 2006/02 and LPAI 2006/03.

#### OBSERVATIONS ON THE CLINICAL FINDINGS AND THE DYNAMICS OF INFECTION WITHIN FLOCKS

96. It is well recognised in the published literature that the clinical signs associated with LPAI infection vary greatly from none to a markedly high mortality rate. This variation is dependent on many factors, including age, species, the virulence of the virus, concurrent infections and husbandry.

97. The clinical signs in the first infected flock were sufficiently marked for the owner to seek veterinary attention. However, problems with the husbandry of the flock caused by a power failure clearly complicated the differential diagnosis. In the event, the predominant clinical signs were restricted to a drop in egg production and an elevated mortality rate. Both were relatively small and the mortality rate was increased for some 14 days.

98. Seroconversion of experimentally infected turkeys has been found to be dependent on the exposure dose (Capua *et al.* 2004). There is also some evidence of an effect of strain on the proportion of birds that seroconvert. Serological positivity has been reported to persist in chickens for 5 months post infection with LPAI. It is notable that the prevalence of seropositive birds observed in the first infected flock did not reach 100%, compared to the other flocks infected. The most likely explanation for this finding is that the virus increased in virulence with its transmission between birds and between flocks. There are number of findings which support this hypothesis:

- The severity of the clinical signs, as adjudged by the mortality rate, was increased on the second infected farm that was kept under the same management conditions. The peak mortality rate on the second infected farm was twice that of the first infected farm, both flocks were managed identically,

for all intents and purposes; they each had the same underlying, pre-outbreak mortality rate.

- The severity of clinical signs, as adjudged by the mortality rate(s) and egg production rate(s), was further increased on the third farm infected. The underlying, pre-outbreak mortality rate was somewhat greater in the LPAI 2006/01 (third infected) flock than the other two farms, but the peak mortality rate in the flock in House 3 was more than four times (at 58 deaths per 10,000 birds) that experienced by the LPAI 2006/03 flock. Similarly, the egg production rate was severely reduced, by approximately 50%, compared to the reduction experienced in the LPAI 2006/03 flock, which was 8.5%.
- No virus was isolated from the first infected flock. Relevant published studies on the duration of excretion of virus are exclusively concerned with experimentally infected poultry. One study involving the Dutch HPAI H7N7 strain indicated that infection was no longer detectable 20 days post exposure. Our findings from the investigations of the first infected flock are therefore in agreement with these findings. Our investigations commenced 39 days after the last most likely date of infection. The main consequence was that no IVPI index was established for the virus infecting this first infected farm. However, the IVPI for the virus isolate from the second infected flock, at LPAI 2006/03, was 0.0. Therefore, no information was lost, but the IVPI for the virus isolate from the LPAI 2006/01 flock was 0.22 suggesting an increase in virulence.

99. There are no published reports on the dynamics of seroconversion, involving sequential sampling of infected birds, as a result of natural infection with the H7 strain (LPAI or HPAI) of the AI virus. Experimental studies indicate that 100% of infected birds seroconvert some 10 – 14 days post infection. Our results suggest that in natural infections a 100% seroconversion in a flock will take longer, perhaps not unexpectedly because the conditions of experimental studies will be conducive to transmission. The findings in the LPAI 2006/03 flock indicate that at 24 days post infection the prevalence of seropositive birds had only reached 45.

100. Experimental infection studies, involving serological testing, indicate that seroconversion occurs 5 – 6 days post-exposure. The results of our investigations support these findings, particularly those from the LPAI 2006/03 flock, and in particular the findings from Sheds 1 and 4.

## MEDICAL EPIDEMIOLOGY

101. Links were established between the medical and veterinary epidemiology teams dealing with suspect human and poultry cases respectively, on 29 April. The veterinary objectives were:

- to determine the likely source of infection for any human case(s) to inform veterinary inquiries, both to confirm the temporal and spatial distribution of

virus in the poultry population, and to validate the assumption that virus is passing from poultry to humans and not vice versa

- to advise the medical team as to the type of work done in the poultry industry, and the different ways exposures can occur.

102. Data on the single human case were shared with Defra (while respecting confidentiality) and details of numbers of people exposed and treated were captured.

### **Human case investigations**

103. A single human case was confirmed, in a member of the permanent staff on LPAI 2006/01. This person developed conjunctivitis on 24 April, 5-8 days after infection is believed to have reached the poultry on the premises. Their symptoms coincided with clinical disease already being evident in two of the four poultry sheds on the farm and was the day that chickens in the third shed started to show evidence of disease.

104. Typing of the avian influenza virus recovered from the human case showed it to be the same strain as was recovered from the poultry on the farm, H7N3, which together with the timing of disease provides epidemiological evidence that the person was infected from the poultry. This person had made a complete recovery by 2<sup>nd</sup> May.

105. The case also worked at 4 other poultry farms in the area on a regular basis (weekly); these farms have been investigated and to date there is no evidence of avian flu affecting the birds there, indicating that disease was not transmitted between the human case and the poultry on these premises.

106. Public health surveillance was enhanced by asking GPs to report all cases of conjunctivitis if linked to a farm or to birds, and public health interventions were provided from a number of sites. This included an emergency response centre in Dereham, with arrangements for rapid submission of samples for examination in Cambridge. People who suspected they were infected were subject to clinical examination and assessment against a case definition for conjunctivitis (hyperaemia with discharge). Those that fitted the case definition were given treatment while samples were examined.

107. By 15 May a total of 7 people who sought advice as regards the possibility that they had contracted avian flu had been sampled for the presence of influenza virus as a cause of conjunctivitis. This was confirmed in the individual case reported above; no evidence of avian flu virus was found in the other six. However an interesting finding was the presence of a vaccine strain of paramyxovirus type 1 in an abattoir worker who would have been exposed to vaccinated birds in the course of his work.

## Human exposure

108. People will have been exposed to virus on all the infected premises for some time before disease was disclosed. Once disease was suspected, additional measures to protect human health were adopted and any potential risk would have reduced. Assessment of exposure is challenging, however Table 7 below summarises the number of people who would have been exposed during the period **before** disease was disclosed, with an indication of timing and frequency.

Premises ID	LPAI 2006/01	LPAI2 2006/02	LPAI 2006/03
Earliest exposure date range*	16-19 April	21-24 March	3-6 April
Earliest maximum exposure date**	21 April	26 March	8 April
Date of report of disease	25 April	27 April	27 April
Number of people with daily direct contact with poultry	5	4	
Number of people with less frequent direct contact with poultry	4	1	
Number of people with less frequent, indirect contact with poultry	4	8	
Estimate of number of person days at risk, among those with direct daily contact <sup>^</sup>	35	140	
Attack rate for human clinical infection	1/35	0/140	
Number of birds on premises	34,500	8000	7700
Housing	4 deep litter sheds	Barn with daylight access to pasture	

\* excretion can start within one day of infection

\*\* maximum excretion from infected individual chicken occurs 5-7 days after infection

<sup>^</sup>estimate takes mid-point of exposure period and does not consider level of exposure

**Table 7: Exposure of personnel on IPs before disease was disclosed and preventive measures adopted**

109. The figures show a very approximate summary estimate of one clinical case per approximately 175 person days at risk. (This excludes consideration of people who visited the poultry less frequently, such as the veterinarians and maintenance staff, or who had indirect contact with the poultry such as feed lorry drivers.) However the premises had very different attack rates and it is of note that the single human case was among the 5 people with daily direct dealings with the poultry on LPAI 2006/01. This premises holds a much larger number of birds, and in closer, and very dusty, confinement, than the other two premises.

110. The epidemiological study to be carried out (see next paragraph) should help to assess different exposure groups more accurately, taking into account such factors as level of exposure related to occupation and type of poultry contact, age, etc. Estimation of risk is difficult, as there was only one clinical case and very few people with symptoms of AI infection, however the serological surveillance in this study may help.

## Human prophylaxis and treatment

111. Oseltamivir prophylaxis (Tamiflu) was offered to all 119 people who worked with the poultry on any of the three affected farms and individuals involved in the control effort. 109 individuals received oseltamivir, one person refused it and information about nine people is unclear. All were also offered seasonal flu vaccination and 73% accepted it. Time of first exposure was recorded for 62 individuals, out of which 46 (74%) received treatment on the same day of first exposure, two received it in advance of exposure and 14 individuals between 1 and 8 days after exposure.

112. These data are still being compiled as part of the HPA's ongoing response to the incident and further epidemiological investigation is being undertaken by the Health Protection Agency to fully assess exposures, use of prophylaxis, symptoms and serological evidence of LPAI H7N3 infection, and occupational risk in the human population associated with the poultry outbreak.

## CONCLUSIONS

113. This was an unexpected outbreak of LPAI infection, particularly because commercial poultry flocks in Great Britain had remained free of LPAI infection and increased surveillance for LPAI and HPAI had proved negative over some years. This is despite wild birds being regarded as a natural endemically infected population.

114. The spectrum and severity of the clinical signs associated with LPAI and HPAI can overlap. The vigilance of flock owners and their attendant veterinary surgeons therefore need to be raised of the possibility of infection with avian influenza, whether due to an LPAI or HPAI strain, and include this infection in the differential diagnosis of reduced egg production, an increased mortality rate, reduced feed and water intake and an absence of conclusive evidence of an alternative diagnosis. This suggests that poultry keepers and the veterinary profession should be made aware of the findings to optimise the identification of such cases of infection.

115. We support the record keeping by poultry keepers of daily egg production and mortality statistics, together with data on feed and water intake. This has been crucially important in the current investigation and provides a unique degree of precision to estimate, for example, dates of infection so that control measures can be correctly and efficiently targeted.

116. The findings suggest that free range flocks may be at most risk from the novel introduction of infection with LPAI, and HPAI, virus. Owners of such flocks should be most vigilant, especially in minimising direct and indirect contact with free-living birds.

117. The results indicate that the established and normal biosecurity measures adopted by poultry keepers are adequate to prevent the transmission of infection

between separately managed flocks. For free range flock owners there is a need to stress the need to identify and remove dead birds so that they cannot be predated.

118. Owners of flocks housed within sites subject to biosecurity measures should be aware of the potential dangers of species such as foxes that can introduce infection into an otherwise biosecure site.

### **Acknowledgements**

We are indebted to the owners of the farms concerned, their staff, and colleagues in the attendant veterinary practice for their time in providing production records and information concerning their flocks and farms for the epidemiological investigations. We would also like to acknowledge the extensive and thorough work of colleagues in the State Veterinary Service (SVS) Local Disease Control Centre (LDCC), the avian virology unit at the Veterinary Laboratories Agency (VLA) Weybridge, the Health Protection Agency (HPA), and in the Animal Health and Welfare Directorate General at Page Street, which made these analyses possible.

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## Defra Wild Bird Survey

1. The wild bird survey provides an early warning if highly pathogenic avian influenza H5N1 viruses are introduced into the UK in wild birds and an assessment of the risk of introduction into domestic poultry.
2. In collaboration with ornithologists and epidemiologists Defra has developed a more targeted approach to wild bird surveillance following an analysis of poultry register data and wetland bird survey data.
3. The survey focuses on species of wild birds that experts believe to have a greater potential role in the spread of avian influenza viruses. There is a comprehensive list which generally includes ducks, geese, swans, gulls and waders. Sampling is targeted as much as possible to areas where there is an abundance of these migratory water bird species and domestic poultry. The aim of this is to focus on areas where an introduction of HPAI H5N1 would be more likely to be detected if it had occurred and to areas where an introduction may have more significance to poultry health.
4. There are three components to the wild bird survey:
  - Sampling of live caught wild birds
  - Sampling of birds shot during normal wildfowling activities ( no birds are shot in addition to those that would normally be shot by hunters)
  - Screening of wild birds found dead

Defra will announce the details of the targeted autumn strategy in the near future. Further information, and results of the survey to date, may be found at:

[www.defra.gov.uk/animalh/diseases/notifiable/disease/ai/wildbirds/survey.htm](http://www.defra.gov.uk/animalh/diseases/notifiable/disease/ai/wildbirds/survey.htm)

## **Advisory Committee on Dangerous Pathogens**

### **Statement from ad-hoc ACDP meeting held on Friday 28<sup>th</sup> October at the Department of Health, Skipton House, SE1 6LH**

An urgent meeting of the Advisory Committee on Dangerous Pathogens (ACDP) was convened to consider the public health measures that would be needed in the event of avian influenza (AI), either low pathogenic AI (LPAI) or highly pathogenic AI (HPAI), being found in poultry or wild birds in the UK. Members were asked to consider the precautions to be taken by those working with poultry or wild birds, those undertaking surveillance of avian species and advice to be given to the wider public. Precautions to be taken by those working with pigs were also considered, given that pigs can be infected by some avian influenza viruses and are susceptible to both human and porcine influenza viruses.

#### **Background**

There is currently no evidence of AI in poultry or wild birds in the UK. However, the widespread outbreaks of H5N1 in poultry in SE Asia, the continuing findings of spread of H5N1 viruses amongst wild birds in Northern Europe and the recent finding of an H5N1 positive bird in quarantine in the UK has increased the need to be prepared against AI infection. The Government needs to provide clear guidance on the public health implications of AI infection in poultry and wild birds to all who are potentially at risk.

Members were provided with background papers as set out in Annex 1.

Members were specifically asked for their views on the draft guidance, prepared by a cross-Government and Industry Working Group, on measures to be taken to protect the health of those working with poultry, and on the protocol prepared by Defra covering the submission of samples from wild birds for laboratory investigation.

This ACDP meeting comes at a time when sample surveillance of poultry flocks is underway and two new surveillance studies of wild birds have recently commenced in accordance with EC requirements. A positive finding of low pathogenic strains of AI amongst wild birds is considered likely. The public health implications of such findings need to be addressed.

#### **Summary of current findings and recommendations for public health**

- Avian influenza viruses do not appear to cross the species barrier from birds to people easily.
- Though data on transmissibility is limited, there is evidence that both LPAI and HPAI can pass from poultry to people, and the risks of such transmission is likely to be the same for both LPAI and HPAI. The severity of any illness that arises may vary according to the particular strain of virus and the person's susceptibility, including whether they have any co-infection.

- Current evidence from the H5N1 outbreaks in Asia suggests that the H5N1 strain does not pass easily from poultry to people, though data may be lacking as to the total number of people who have been infected, particularly in areas of poor surveillance and those infected without showing signs of serious disease.
- Evidence to date indicates that the risks to people of acquiring infection are almost entirely confined to those who have close contact with infected poultry and there is so far no evidence of transmission from wild birds to people.
- Poultry, wild birds and game birds can present a range of zoonotic disease risks to those who work with or handle them, and precautions should always be taken to prevent the risk of disease transmission.
- Wild birds can be infected with a range of avian influenza viruses the majority of which are species-specific in causing disease. Wild birds, particularly ducks, can be infected with both LPAI and HPAI strains without showing outward signs of disease, even when they have high viraemia.
- LPAI viruses do not appear to mutate into HPAI strains within wild birds.
- LPAI viruses have been shown to have the potential to mutate into HPAI within poultry, and these can then infect wild birds.
- There is a theoretical risk of influenza virus re-assortment if people with seasonal influenza become co-infected with avian influenza. Vaccination against seasonal flu of those people most at risk of avian influenza will reduce the risk of such influenza virus re-assortment.
- Pigs can be infected with some strains of avian influenza virus, and are susceptible to both porcine and human influenza viruses. Human influenza viruses are circulating between pigs all the time. However, there remains a theoretical risk of virus re-assortment within pigs between human and avian influenza viruses in the event of pigs being co-infected.
- Overall, the public health risks posed by avian influenza, in the absence of outbreaks of H5N1 in poultry in the UK remain low. Appropriate hygiene precautions to prevent any infection from handling wild birds, game birds or poultry should always be taken by the public and appropriate zoonotic disease prevention measures as recommended by the HSE and as indicated by risk assessment should be taken by those handling poultry and wild birds in the workplace. Such preventive measures may need to be enhanced if AI is confirmed, depending upon local risk assessment.

### **ACDP Advice and Recommendations for Public Health**

#### **Poultry workers**

ACDP Members emphasised that there are many zoonotic diseases other than avian influenza that pose a threat to the health of poultry workers and others in direct contact with poultry. There was agreement that the precautions necessary to protect against these other zoonotic health risks also offered protection against infection with avian influenza. Members agreed that in some circumstances, such protection would need to be increased,

based upon local risk assessments. The ACDP endorsed guidance drafted by a cross-Government and Industry Working Group on precautions to be taken by the poultry industry to protect workers from avian influenza infection. Members recommended that this guidance be supplemented by posters and/or leaflets that presented the key points in language appropriate for the intended audience. The Working Group was comprised of representatives from the National Farmers Union, British Egg Industry Council, British Poultry Council, Health and Safety Executive (HSE), Health Protection Agency, Department of Health, Department for Environment, Food and Rural Affairs (Defra) and the State Veterinary Service. This guidance has been published on both the Defra website [<http://www.defra.gov.uk/animalh/diseases/notifiable/disease/ai/poultrykeepers.htm>] and the HSE website [<http://www.hse.gov.uk/biosafety/diseases/avianflu.htm>].

Members were reminded of the current advice, which is that in a confirmed outbreak of avian influenza, all those who might have been exposed to highly pathogenic avian influenza through direct contact with infected poultry, or who are at risk of such exposure, should be offered antiviral therapy (oseltamivir) for the prevention of disease, and the Joint Committee on Vaccination and Immunisation has recommended that seasonal flu vaccine be offered to all those at risk of infection as a public health measure to prevent risk of virus re-assortment. ACDP Members were aware of the diversity amongst the workforce involved across the whole of the poultry and game industry and of the difficulties this presented in practice.

In view of the discussions around the equal risks of transmissibility to people of LPAI and HPAI from poultry, and the time taken for flu vaccine to produce an immune response, Members were of the view that JCVI should be made aware of ACDP's recommendation for routine use of seasonal flu vaccine for poultry workers and others in the light of the current risk assessment by Defra of a high risk of further global spread of avian influenza amongst wild birds.

Additionally, the ACDP considered that serological surveys of workers in the different sectors of the UK poultry industry (e.g. poultry cullers, husbandry staff, vets etc) could provide useful information about infection risks and exposures and should be carried out if possible. It was felt that such studies were urgently needed to further evaluate the potential for avian-to-human transmission of avian influenza (both LPAI and HPAI) within the UK. The Chair was asked to write to the Health Protection Agency to express the ACDP's strongly held views on the value of such studies. Data were presented indicating a 3.8% seroprevalence to LPAI in Italian poultry workers, none of whom experienced a flu-like syndrome.

### **Game Birds**

The ACDP noted that people in close contact with game birds, would have a similar risk of exposure to AI, and therefore should have the same precautions offered to them as poultry workers. The Health and Safety Executive agreed to look into providing specific advice for this group of workers.

### **Wild birds**

The risk of transmission of either LPAI or HPAI from wild birds to the general public was considered to be small. The ACDP were of the view that although the general public should not in general handle dead wild birds, there were those who would need to for a variety of reasons, as well as those who would wish to. Members considered that the general public need only to adopt common-sense precautions, and that these should be more clearly set out for them so as to be easily followed.

The ACDP noted that the Defra surveillance programme of wild birds for AI involved both an ongoing, established survey of dead birds in which there is public participation by bird enthusiasts, and a survey of wild birds in which samples are taken from live birds (cloacal swabs). A protocol for the submission of dead birds and the taking of samples from live birds have been produced by Defra. ACDP agreed with the advice given but recommended making it clear that the protocols for sampling are intended for individuals who have received appropriate training in the methods to be used and who are handling carcasses or live animals as part of their job, and are not aimed at the general public. It is intended that the amended protocol will be published on the Defra website.

### **Pigs**

ACDP noted that pigs can be infected by some avian influenza viruses and are susceptible to both human and porcine influenza viruses. It was noted that the hypothesis that pigs act as the “mixing vessels” for influenza viruses from which a new pandemic human strain arises is no longer widely held to be correct, as there was little evidence that a flu strain dangerous to public health has been produced in this way. Human flu viruses of the H1 and H3 strains are established in pigs and circulate between pigs all the time. There is, however, the theoretical risk that co-infection of the pig with avian influenza and human influenza could lead to re-assortment to produce new strain of public health significance and a precautionary approach should be adopted. Members were of the view that JCVI should be asked to consider the use of seasonal flu vaccine for those in close direct contact with pigs as a public health measure to minimise the chance of recombination between seasonal flu, porcine or avian flu in pigs with transmission back to people.

## Annex 1 – Background open papers provided to ACDP Members

- ECDC Papers – Interim ECDC Risk Assessment "[The Public Health Risk from Highly Pathogenic Avian Influenza Viruses Emerging in Europe with Specific Reference to type A/H5N1](#)" (version 19<sup>th</sup> October 2005) and "[Interim Guidance for Workers Protection](#)" (version 19<sup>th</sup> October 2005), including the 3 references from the latter document.
- EFSA Report - "Opinion of the AHAW Panel related to animal health and welfare aspects of Avian Influenza"  
[http://www.efsa.eu.int/science/ahaw/ahaw\\_opinions/1145\\_en.html](http://www.efsa.eu.int/science/ahaw/ahaw_opinions/1145_en.html)
- [Pro-med summary](#) from 6th Jan 05 about Netherlands Avian Influenza Outbreaks
- Puzelli, S. *et al.* Serological Analysis of Serum Samples from Humans Exposed to Avian H7 Influenza Viruses in Italy between 1999 and 2003 (2005) *JID* 2005:192 (15 October) p-1318 - 1322
- [Defra Exotic Animal Disease Generic Contingency Plan](#) (especially pages AI 12-13 in Part III)
- JCVI Meeting summaries - [JCVI website](#)