

Prevention and management of avian influenza outbreaks: experiences from the United States of America

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Summary

The epidemiology and control of avian influenza (AI) are complex. The virus is transported in nature by the activities of wild birds and in commercial poultry by the activities of people. In general, all the outbreaks of AI in the United States of America (USA) have involved AI virus spread by the movement of poultry and manure and objects contaminated by poultry and manure, but the specific cause of spread has been different for most outbreaks. The 1924 highly pathogenic AI (HPAI) outbreak was spread halfway across the USA by contaminated rail cars and poultry crates; the 1983 HPAI outbreak was spread by the movement of people between farms and transport of live and dead poultry, including depopulation efforts; whereas low pathogenicity AI (LPAI) outbreaks in different states were spread by people and equipment, partial flock removal, transport of spent hens and/or manure, and transport of dead birds for rendering.

There is a dichotomy surrounding AI control methods in the USA. Large LPAI outbreaks have mainly affected turkeys in the western part of the country and have been controlled by vaccination and controlled marketing – strategies developed prior to the 1983 HPAI outbreak. By contrast, in the eastern part of the country, the AI control strategy has been modelled on the successful stamping-out programme that was used during the HPAI outbreak in 1983. The author presents a summary of the costs and control strategies in table form.

Keywords

Avian influenza – Control – Controlled marketing – Epidemiology – Lessons learned – Prevention – Stamping out – United States of America.

Introduction

Highly pathogenic avian influenza (AI) outbreaks in the United States of America (USA) have been recently reviewed by Swayne (25). Large outbreaks of low pathogenicity AI (LPAI) in the USA are partially documented, and their inclusion here contributes to the understanding of transmission dynamics and control of AI.

First highly pathogenic avian influenza (fowl plague) outbreak in the United States of America

The first AI outbreak in the USA was the fowl plague outbreak – highly pathogenic AI (HPAI) – that affected

poultry in nine states during 1924 and 1925. An undiagnosed mortality problem was first seen as early as June 1924 (5) in the live poultry marketing system in New York and high mortality was reported in several large markets in August. The disease spread to New Jersey and Pennsylvania markets. By late September it was spreading to all parts of the city and became very destructive (18). At that time, the New York City live poultry market system comprised 307 permit-holding wholesale and retail businesses whose annual sales were approximately 49 million chickens per year. Losses were proportional to the amount of time that fowl were in the markets rather than to the number of chickens handled.

Observations at the time indicated that the disease was restricted to chickens and that heavier breeds were more susceptible. A continuous cycle of infection was reported in the markets and this infection was introduced into these markets and private flocks by the purchase of diseased and exposed chickens. Other sources of infection were reportedly associated with chickens foraging in areas where chicken carcasses or offal had been dumped. Wild bird infection apparently played no role as a source or means of spread.

Investigations conducted by the Bureau of Animal Industry, later part of the United States Department of Agriculture (USDA), revealed that an unnamed investigator at a 'well-known eastern educational institution' imported some virus of European fowl pest from the Pasteur Institute in the summer of 1923. Prior to this, the disease was not known to exist in the USA. This same investigator was also reported to have shared virus with other investigators and to have provided it for infection studies on a farm. An epidemiological link was found between at least one of the institutions conducting such investigations and New York City poultry dealers (3).

The economic impact of this outbreak was estimated to be US\$1 million, and it was estimated conservatively that 500,000 to 600,000 chickens died in New York City alone. In December, New York (wrongly believing that incoming fowl were infected) embargoed chickens from several Midwestern states. This also had significant economic effects on Midwestern farmers, as well as introducing disease into new areas as rejected chickens were sent from New York to other states (25, 27).

After the embargo, infected poultry were ultimately found on farms or at markets in Connecticut, Illinois, Indiana, Michigan, Missouri and West Virginia, beginning in January and February of 1925 (25). Authorities documented the lack of transmission through the air and the role of contaminated crates and rail cars in spreading the disease (18). Ultimately it is clear that the outbreak began in the live poultry market system and spread to other locations via the railway system (25).

Lessons learned in the 1924 to 1925 outbreak

In the absence of knowledge of the aetiologic agent, and without modern diagnostic tools, such as virus isolation, antigen capture, polymerase chain reaction (PCR), agar gel immunodiffusion, enzyme-linked immunosorbent assay, computers and global information systems, the multi-state outbreak was eradicated by the spring of 1925, following the application of quarantine, depopulation and disease control procedures.

New Jersey incident

In May of 1929, a small outbreak of fowl plague was detected in New Jersey, involving four infected flocks. Clinical signs and lesions were similar to those reported in the 1924 to 1925 fowl plague outbreak. The New Jersey outbreak was declared eradicated on 15 August 1929. The source of this infection is unknown (25).

The lessons of the two fowl plague outbreaks in the early 20th Century were lost to avian health specialists during the next 40 years.

The second large outbreak of highly pathogenic avian influenza in the United States of America

Low pathogenicity AI H5N2 infection was first detected in Pennsylvania on 22 April 1983, in chickens experiencing respiratory signs. By 24 October, additional flocks were affected (in total: twelve broiler flocks, ten layer flocks, two broiler-breeder flocks and one pullet flock) (11). A serological survey in late August and early September did not detect any additional cases. Chickens experienced mild-to-moderate respiratory signs, low mortality and declines in egg production. Virus isolates pathotyped at the National Veterinary Services Laboratories (NVSL) were found to be characteristic of non-pathogenic AI, as described in the recommendations from the First International Symposium on Avian Influenza in Beltsville, Maryland, 1981. Mortality ranged from 0.5% to 11% (mean: 2.3%), and in egg production flocks the mean decline in egg production was 23%, with recovery to near normal levels within six weeks (17).

On 8 October, layers were presented to the diagnostic laboratory with reported declines in feed and water intake and egg production within a few days. Within ten days, mortality was 89%. The predominant signs were listlessness and dehydration, and lesions included

oedematous, cyanotic or necrotic comb and wattles, and peri-orbital oedema. In some birds, oedema and subcutaneous haemorrhages were observed on the feet and shanks. The isolated virus was confirmed by NVSL to be HPAI H5N2. A federal emergency was declared on 4 November 1983 and eradication efforts began.

This conversion of an LPAI virus to an HPAI virus is the first documented case of HPAI virus emerging from LPAI virus. Dr Bob Eckroade stated, 'Dr Charles Beard spoke loudest about the situation, saying, "You better watch it," and so we did. We had literally watched this H5N2 AI virus change from a non-pathogenic agent into a pathogenic one' (11).

The presence of the HPAI virus did not mean that the LPAI virus had disappeared, and the complex flock infections that ensued caused unanticipated regulatory, diagnostic and control nightmares. First, regulations were either not interpreted or not written to allow the destruction of flocks infected with non-pathogenic AI. Secondly, attempts to confirm HPAI in flocks where both viruses existed required multiple diagnostic procedures, because sometimes an HPAI virus was detected and, at other times, the non-pathogenic virus was found. Sometimes, flocks that clinically appeared to have HPAI yielded only LPAI viruses and vice versa. This contributed greatly to the large testing volume. (Between October 1983 and April 1985, 174, 114 samples from 41,471 submissions were received at NVSL.) Thirdly, flocks previously infected with LPAI were considered to be potentially infected with HPAI and their

status was questionable. Finally, in February 1984, a decision was made to eradicate all flocks with evidence of any H5N2 infection. This outbreak ultimately resulted in the destruction of 17 million birds in 448 flocks in Pennsylvania (HPAI and LPAI), Virginia (LPAI) and New Jersey (LPAI), at an estimated cost of US\$63 million to the government, and US\$15 million in non-indemnified losses to producers, or a total of US\$156 million, when adjusted to 2007 dollars (Table I) (26).

Lessons learned in the 1983 to 1984 outbreak

The source of this outbreak was not detected at the time, but the spread of infection was later attributed to contaminated transport trucks and coops used to haul poultry, movement of live and dead birds (including depopulation efforts), and contaminated feed trucks, eggs, feed, water, people and equipment. Many of these lessons could be described as rediscoveries of lessons from the 1924 to 1925 outbreak.

Related outbreaks

Although both the HPAI and LPAI H5N2 viruses were eradicated from affected states in 1983 and 1984, a related outbreak of LPAI H5N2 occurred in 1986. It started in Pennsylvania and New Jersey and spread to New York, Massachusetts and Ohio, involving 21 flocks of broilers, roasters, turkeys, layers and other poultry (12). This outbreak was controlled by destruction, disposal,

Table I
Outbreaks of highly pathogenic avian influenza and low pathogenicity avian influenza in the United States of America: locations, economic losses and control measures

Year	Virus	Location	Cost in 2007 US\$	Control measures	Cost per flock or farm US\$
1924 to 1925	HPAI	New York	\$12.2 m	Stamping out	Unknown
1978	LPAI	Minnesota	\$16 m	Controlled marketing	\$113,000/flock
1983 to 1984	HPAI	Pennsylvania	\$156 m ^(a)	Stamping out	\$346,000/flock
1988	LPAI	Minnesota	\$5.8 m	Controlled marketing/Vaccination	\$23,000/flock
1991	LPAI	Minnesota	\$1.5 m	Controlled marketing	\$14,000/flock
1995	LPAI	Utah	\$2.7 m	Controlled marketing/Vaccination	\$45,000/flock
1995	LPAI	Minnesota	\$8.2 m	Controlled marketing/Vaccination	\$46,000/flock
1986 to 1988	LPAI	Pennsylvania	\$6 m	Stamping out/Controlled marketing	\$128,000/flock
2000 to 2003	LPAI	California	Unknown	Controlled marketing/Vaccination	Unknown
2002	LPAI	Virginia	\$166 m	Stamping out	\$842,000/farm
2003	LPAI	Connecticut	\$5.6 m	Vaccination	\$800,000/farm ^(b)

HPAI: highly pathogenic avian influenza

m: million

LPAI: low pathogenicity avian influenza

a) This outbreak resulted in a nationwide shortage of eggs that drove egg prices up and increased the costs to consumers by an additional \$700 million

b) Although the Virginia and Connecticut outbreaks cost the same amount per farm, each outbreak involved approximately the same number of birds – 4.7 million – making the cost per bird \$35 in Virginia and \$1.19 in Connecticut

Adapted from Swayne and Halvorson (26)

quarantine, surveillance, disease control measures and education, and is the first LPAI outbreak in the USA controlled by stamping out. Kradel observed the link between the index case of this outbreak and the New York live poultry marketing system (another rediscovery of the experience from 1924 to 1925) (17). An epidemiological investigation clearly incriminated crates from live poultry markets being unloaded directly into the poultry house at the index farm and the practice of 'topping off' a flock – taking the larger birds from the flock and leaving susceptible birds behind (12). Bean *et al.* demonstrated the molecular link between virus isolates from the live poultry markets, from the 1986 outbreak, and from the 1983 to 1984 outbreak (6). Intense efforts to eliminate the H5N2 virus from the live poultry marketing system were successful, and the last H5N2 virus related to the HPAI 1983 outbreak was recorded in 1989.

The Texas incident

On 16 February 2004, H5N2 was diagnosed in a small broiler flock near Gonzalez, Texas. The flocks experienced mortality of 3% and respiratory signs. On inoculation of chickens with this H5N2 virus, no mortality occurred but, based on molecular characteristics, the infection was declared to be HPAI. The amino acid sequence at the haemagglutinin cleavage site was identical to the HPAI A/chicken/Scotland/1959 strain. Epidemiological observations pointed to infection occurring after birds were returned from the live poultry market in the area. The virus was detected in two retail live poultry marketing establishments. All birds in affected and at-risk premises were depopulated and the outbreak was declared over on 1 April 2004. There was no evidence of a wild bird role and, based on a deletion from the neuraminidase (NA) gene encoding the stalk of the protein, it was suggested that the virus had been circulating in poultry for some time (25).

Low pathogenicity avian influenza in the United States of America

Following the fowl plague outbreaks in the 1920s, AI was not recognised again in the USA until the mid-1960s, in turkeys in California, Massachusetts, Wisconsin and Minnesota. Over the next four decades, small outbreaks of LPAI were detected each year by virus isolation or serological test. From the mid-1970s to the present, there have been nine large outbreaks (arbitrarily characterised here as involving over 100 flocks or one million birds) of LPAI in the USA. Four of these outbreaks were in Minnesota.

Minnesota outbreaks

In 1978, turkey growers in Minnesota experienced a large outbreak of LPAI, due largely to H6N1, but H4N8, H6N2, H6N8 and H9N2 infections were also detected. This outbreak involved:

- 130 market turkey flocks, containing over two million turkeys
- 11 turkey breeder flocks, containing 24,000 breeders
- 3 egg-production chicken flocks, containing 165,000 hens.

The source of the outbreak in 1978 and subsequent years was the exposure of free-range turkeys to wild bird populations. Once free-range turkeys became infected, traffic on the farm would move the AI virus into confined flocks. A management practice of growing hens and toms on the same farm, and marketing the hens to provide more space for the toms, contributed to the outbreak. This partial flock removal practice, driven by economic pressure to maximise the use of floor space, brings potentially contaminated load-out equipment and crews into close contact with turkeys that remain on the farm (4).

The highest mortality recorded was over 75%, due largely to secondary bacterial infection with *Escherichia coli* or live *Pasteurella multocida* vaccine, and the highest condemnation at processing was 73% of birds marketed. Losses due to mortality, condemnation, weight loss, egg loss, medication costs, clean up costs and loss of profits totalled US\$5 million in direct costs or approximately US\$2 per bird (US\$16 million, when adjusted to 2007 dollars) (26). The additional costs associated with 'down time', caused from the interrupted schedule on a three-age farm, were shown to be another US\$2.50 per bird (US\$7.86 in 2007 dollars) (22).

After turkeys were removed from ranges, due to the onset of winter, this outbreak was ended by improved biosecurity and controlled marketing. Controlled marketing refers to the practice of marketing birds after clinical signs have abated and when viral shedding is at a low level. The magnitude of this outbreak caused growers and veterinarians to develop co-operative plans for the prevention and control of AI, through:

- education
- prevention of exposure
- monitoring
- reporting
- responsible response (13, 21).

Low pathogenicity avian influenza continued to be seen each year in Minnesota for the next nine years, involving between 2 and 73 flocks each year and the detection of H1, H2, H3, H4, H5, H6, H7, H8, H9 and H10 subtypes. In 1979, and for a few years after that, some turkey growers embarked on a programme of prophylactic vaccination for AI. The subtype selected for vaccination was based on farm history. This practice proved to be ineffective and, after 1982, prophylactic vaccination was used only in turkey breeders and consisted primarily of H1N1 vaccine directed against swine influenza.

In 1988, Minnesota turkey growers again experienced a large LPAI outbreak, involving 258 flocks of turkeys and one flock of broiler-breeders. Subtypes H2N2, H4N6, H5N6, H7N9, H8N4 and H9N2 were detected. The outbreak was controlled by elevated biosecurity, controlled marketing and vaccine use. Economic losses associated with the outbreak were US\$5.8 million (in 2007 dollars).

Three years later, in 1991, in an outbreak involving 110 flocks of turkeys, H1, H4, H5, H6 and H7 influenza viruses were detected. Economic losses associated with the outbreak were US\$1.5 million (in 2007 dollars).

The last large LPAI outbreak in Minnesota began on 1 August 1995, and did not end until March of 1996. When it was over, 178 flocks of market turkeys had been infected – most with H9N2 – but three other subtypes were also detected: H1N1, H6N8 and H10N7. Factors that contributed to this outbreak included:

- large wild bird populations
- partial flock removal
- marketing actively infected birds
- inadequate supervision of a load-out crew
- in two cases, failure to clean out barns (15).

The presence of feed and water on turkey ranges during a drought attracted large numbers of wild birds to these ranges, and some observers felt that infected free-range turkeys were infecting large numbers and species of wild birds, changing the ecology of the virus in nature and contributing to further spread within the turkey industry (M.C. Kumar, personal communication, August 2008). After the 1978 outbreak, the practice of growing hens and toms on the same farm had ceased, but partial flock removal returned, as portions of tom flocks (consumer toms) were marketed to allow additional space for other birds (heavy toms). Control measures included biosecurity, controlled marketing and vaccination, and a total of 1.6 million doses of vaccine were administered. Economic losses associated with the outbreak reached US\$8.2 million, in 2007 dollars (26).

Lessons learned in low pathogenicity avian influenza outbreaks

Minnesota

All introductions of AI into the state of Minnesota were considered to be from wild birds. After the 1997 outbreak of HPAI H5N1 in Hong Kong, with resulting human infection, the Minnesota industry collectively decided to cease growing turkeys on the range. Today, less than 0.5% of Minnesota turkey flocks are range reared, and introductions of AI into turkey populations have declined from an average of more than five per year to less than one. Minnesota turkey growers learned that the spread of infection was associated with the movement of live and dead birds, manure and contaminated people and equipment. During the last 30 years, it has been noted that decisions that were formerly made at the farm level are now made in corporate offices, and these decisions may drastically affect disease transmission and control.

Utah

On 1 April 1995, a clinical outbreak of AI began in free-range turkeys in Utah, resulting in the isolation and identification of H7N3 by NVSL on 25 April 1995. The source of infection was thought to be wild birds which had contact with free-range turkeys. Control measures included:

- informing growers of the outbreak
- controlled marketing of flocks after recovery
- cleaning out and disinfection of barns.

Permission to use H7N3 vaccine was granted by the USDA Animal and Plant Health Inspection Service (APHIS) and the first flock was vaccinated on 20 June, 11 weeks after the onset of the outbreak. Non-vaccinated sentinel turkeys in vaccinated flocks were monitored serologically and did not seroconvert. Within six weeks after the vaccination programme began, over 150 flocks were vaccinated and no additional infected flocks were detected (15). The economic losses associated with this outbreak were US\$2.7 million, in 2007 dollars.

Pennsylvania

An outbreak of LPAI H7N2 occurred in Pennsylvania from December of 1996 to April of 1998. Over 2.6 million commercial poultry in 47 flocks on 21 premises were involved. Egg-laying chickens were predominantly affected but one flock each of pullets, quail, turkeys and mixed

backyard birds were also involved. No broilers were affected. The index case of this infection was the flock of a live-poultry market dealer. Traceback investigations cooperatively conducted by the New York Department of Food and Agriculture and the Pennsylvania Department of Agriculture revealed that this dealer had visited a total of 405 farm premises in the three-month period before the outbreak. The influenza spread from the farm of the dealer to layers in the same general vicinity. Subsequent spread was attributed to:

- area spread (unknown biosecurity breeches and practices) (16)
- bird depopulation activities
- association with live bird markets
- manure spread
- movement out of the quarantine zone
- contact with contaminated equipment.

Once it was determined that depopulation activities were contributing to the spread of the outbreak, these activities were abruptly terminated. Affected birds were allowed to remain on the farm, eggs were allowed to move to further processing and pasteurisation, and flock movement was delayed until viral shedding could no longer be detected (9, 16). A unique feature in this outbreak was the apparent interruption of infection followed by recrudescence or reinfection observed in two flocks of laying hens. Whether this was related to incomplete flock immunity, loss of immunity or the unusual virus predilection for oviduct tissue is not clear. Losses during this outbreak were estimated at US\$3 to US\$4 million (US\$6 million in 2007 dollars).

California

In early February 2000, LPAI H6N2 was detected in backyard chickens and a commercial egg production flock in Southern California. From analysis of the viruses isolated from both cases, it was concluded that they were closely related to a probable wild bird source (28), but no epidemiological link between the premises could be found. Evidence of infection was found in eight egg-layer facilities during 2000. During 2001, LPAI H6N2 was detected on six premises, including one in Northern California. In February 2002, LPAI H6N2 was detected in Southern California and, by mid-March, it had been disseminated to Northern California meat bird premises. The spread of the virus from Southern California to Northern California was attributed to the movement of spent hens (7). The 2002 outbreak involved:

- 37 layer flocks
- at least 43 broiler farms
- nine turkey farms

- two broiler-breeder farms
- one turkey breeder farm.

Although not all flocks of broilers on an affected farm were shown to be infected, the total number of broilers on affected farms exceeded 20 million, making this the largest LPAI outbreak in the USA, in terms of bird numbers (8) (R. O'Connor, personal communication, September 2008). The direct and indirect cost to the layer industry is estimated at US\$40 million (G. Cutler, personal communication, September 2008), but no estimate of economic loss to the meat-bird industry is available.

Although wild birds were observed on farms, there was no evidence of their role as a source of virus or means of spread. Transmission from layer flock to layer flock was attributed to the circulation of contaminated egg flats and racks or pallets between farms, particularly on farms where eggs were hand gathered. Practices that contributed to transmission from flock to flock in Northern California included manure handling, moving live birds to slaughter, dead bird pick-up, shared equipment and common transporters and service crews (7) (R. O'Connor, personal communication).

California poultry producers developed a comprehensive voluntary control plan that required surveillance, biosecurity and movement controls (7). The first part of the plan was to minimise the risk of exposure by increasing communication with neighbours and controlling the:

- transport of birds
- movement of manure
- marketing of eggs
- feed delivery
- movement of crews
- disposal of dead birds
- use of shared employees, shared equipment and common vendors.

The second part of the plan was to increase surveillance with targeted serological testing and daily observation for clinical signs. The third part of the plan was a 'responsible response' to AI virus infection, in which negative (never infected) flocks had no restrictions on movement; virus negative flocks (previously infected) could move to slaughter or onto a positive farm; and suspect flocks and positive flocks (currently shedding) were self-quarantined. The final step in the California plan was to prevent infection in future flocks by a cleaning and disinfection programme and vaccination of at-risk flocks, many of which were on multiple-age layer farms (7). Eight million doses of inactivated oil emulsion vaccine with a heterologous NA were administered. As a result of this

comprehensive programme, H6N2 was eliminated from the California commercial poultry industry in 2003.

Although the source of the H6N2 virus remains unknown, H6N2 was first detected in live poultry markets in 2004. An H6N2-positive quail flock, which was a source flock for these markets, had a connection to one of the layer farms that had previously tested positive in 2002. An AI control plan was created to prevent the movement of infectious agents throughout the marketing system. The plan incorporated the:

- prevention of infection in supply flocks
- prevention of introduction into the live bird markets
- prevention of spread
- prevention of maintenance of infection.

As a result, H6N2 was eradicated from the California live poultry markets in 2005 (20).

Lessons learned

Eliminating the influenza virus requires rigorous application of farm and regional biosecurity measures, combined with vaccination and monitoring. Poultry companies recognised the need for improved communication between and within industries and the existence of unanticipated threats and biosecurity gaps. After observing evidence of AI transmission associated with the rapid removal of manure from facilities housing infected birds, the cleaning out of such farms was delayed for seven to ten days to allow for virus inactivation. Regional biosecurity must be systematic in approach and involve all production systems:

- layers
- meat birds
- allied industries
- backyard birds
- live poultry markets.

Virginia

On 7 March 2002, signs of respiratory disease and a drop in egg production (24) were observed in a turkey breeder flock in Virginia. A diagnosis of LPAI H7N2 was confirmed on 12 March. Within the next few days, clinical signs were seen at several additional turkey breeder facilities owned by the same company. On 20 March, commercial turkeys 30 miles away and owned by a different company were diagnosed as positive. By 28 March, 20 positive flocks had been identified, and by 12 April the total was more than 60 flocks (2).

After embarking on a programme of destruction and disposal, Virginia officials were unable to destroy and

dispose of infected flocks fast enough and requested federal assistance. On 14 April, a joint taskforce of state, federal and industry representatives was formed. This LPAI outbreak marks the first time that the federal government participated in stamping out LPAI. Dead bird surveillance was instituted during this outbreak, and all poultry producers in the quarantine zone were required to place a sample of dead birds in sealed containers at the end of their driveway for weekly sampling by the taskforce. This practice facilitated sampling without compromising biosecurity and proved very effective in detecting infected flocks that were not exhibiting clinical signs. Initially, swab specimens were tested by three methods: antigen capture, virus isolation and real-time reverse transcription PCR (RRT-PCR). After comparison of these methods, it was decided to use only the antigen capture test in Virginia and RRT-PCR at NVSL, making this the first disease outbreak in the USA for which the primary diagnostic test was a molecular method (24).

By the time the last positive flock was identified on 2 July 2002, 197 flocks had been infected (24). Approximately 4.7 million birds were destroyed, at a total cost of US\$166 million (in 2007 dollars).

The source of infection was never established, but the strain responsible was shown to be genetically similar to the strain causing outbreaks in Pennsylvania and the strain that was found in the live bird markets in the northeast of the country from 1994 to 2006. A North Carolina turkey flock processed in Virginia was reported to North Carolina officials as testing seropositive to H7N2 AI on 6 March 2002. Follow-up surveillance detected one additional infected turkey flock and one quail flock. The owner of one of the traceback farms made regular trips to the markets in Pennsylvania, and this activity could have been the source of the outbreak. Surveillance activities failed to find any evidence of a wild bird source (24).

A case-control study involved surveying 151 affected premises and 199 non-affected premises. The most significant risk factor was the transport of dead birds for rendering. Other significant risk factors included:

- birds aged more than ten weeks
- use of non-family (hired) labour
- mammalian wildlife on the farm.

Factors that were not significantly associated with infection were:

- types of biosecurity measures employed
- feed and litter sources
- types of domestic animals on the farm
- the presence of wild birds (19).

Lessons learned

This outbreak pointed out the need for greater barriers between live poultry markets and the poultry industries. Disease spread occurred primarily through people, fomites and contaminated equipment. The methods used for sample collection, euthanasia and disposal did not contribute to transmission within or outside the control area. Arguments for allowing inactivated vaccine use had been proposed (14), and in this outbreak USDA gave permission, but Virginia decided not to use it. The need for criteria for vaccine use became apparent. Thus, APHIS requested that the United States Animal Health Association (USAHA) convene a special meeting, in association with the 2002 National Poultry Improvement Plan meeting, to address the issues surrounding AI vaccine use. Informal recommendations from this meeting led to the passage of a resolution at the 2002 USAHA meeting, acceptance by USDA and the basis for allowing vaccine use in Connecticut (10).

Connecticut

In February 2003, an outbreak of LPAI H7N2 was detected on four of seven farms under the same ownership. Clinical signs included a dramatic decline in feed consumption, followed by a drop in egg production of 10% to 20%. Respiratory distress was observed and mortality was slightly elevated. When the infection was diagnosed, three of the seven farms were already infected. There was no evidence that wild birds had played a role in introducing the virus, which was genetically linked to the LPAI H7N2 virus that had been circulating in the live bird marketing system in the northeast of the country for nearly a decade (1).

The Connecticut Department of Economic and Community Development estimated that depopulation costs alone would exceed US\$30 million, and the projected benefit-to-cost ratio of vaccination versus

depopulation was approximately 10:1 (23). Based on the projected business and social costs associated with depopulating 3.5 million layers from multi-age farms, and taking into account recent successes in Utah and Italy, a programme of vaccination and enhanced biosecurity was recommended. After nearly a month of meetings and negotiations between the owner and government officials, USDA APHIS authorised vaccination with certain caveats:

- a) pullets would be vaccinated twice, at six weeks and thirteen weeks of age
- b) egg production flocks that had been infected would be vaccinated once
- c) authorisation would be withdrawn if:
 - there was evidence of virus mutation to HPAI
 - protocols were not followed
 - there were indications of failure after six months
 - there was spread of the virus to new premises
 - significant trade bans were imposed on the USA (1).

Lessons learned

The administration of inactivated oil emulsion vaccine, in conjunction with enhanced biosecurity, aided in the elimination (eradication) of LPAI H7N2 without depopulating a large commercial layer operation. Vaccination began in April 2003, and no AI virus was isolated from vaccinated or sentinel birds or from the environment from that time until the quarantine was lifted, in September 2004 (1). The estimated cost to the producer of the disease and the control programme was US\$5 million in 2003 (N. Adriatico, personal communication, March 2003).



Prévention et gestion des foyers d'influenza aviaire : l'expérience des États-Unis d'Amérique

D.A. Halvorson

Résumé

L'épidémiologie et la prophylaxie de l'influenza aviaire sont des domaines complexes. Dans la nature, la circulation virale est déterminée par les activités de l'avifaune, tandis que chez les volailles commerciales elle est due aux activités humaines. En général, dans tous les foyers d'influenza aviaire survenus aux États-Unis d'Amérique, le virus s'est propagé suite à des mouvements de volailles, de fumiers, ou d'objets contaminés par des volailles ou des fumiers infectés ; néanmoins, dans la plupart des cas, la propagation virale avait aussi une cause spécifique et différente à chaque fois : le foyer d'influenza aviaire hautement pathogène (IAHP) de 1924 s'est propagé sur la moitié du territoire des États-Unis par l'intermédiaire de wagons et de cages à volailles infectés ; le foyer d'IAHP de 1983 s'est propagé suite aux mouvements de personnes entre exploitations aviaires et au transport de volailles vivantes et mortes, y compris dans le cadre d'opérations de dépopulation ; les foyers d'influenza aviaire faiblement pathogène (IAFP) survenus dans plusieurs états se sont propagés suite à l'évacuation partielle de troupeaux, aux transports de poules épuisées et/ou de fumiers, et aux transports d'oiseaux morts destinés à l'équarrissage.

Aux États-Unis, la lutte contre l'influenza aviaire est pratiquée de deux manières très différentes. Dans les régions de l'Ouest du pays, les foyers d'IAFP (qui ont surtout affecté les dindes) ont été maîtrisés grâce à la vaccination et à des restrictions commerciales, stratégies mises au point avant le foyer d'IAHP de 1983. En revanche, dans les régions de l'Est, la lutte contre l'influenza aviaire s'inspire du programme d'abattage sanitaire mis en œuvre lors du foyer d'IAHP de 1983, qui avait donné de bons résultats. L'auteur présente dans un tableau un résumé des coûts et des stratégies de contrôle.

Mots-clés

Abattage sanitaire – Épidémiologie – Influenza aviaire – Leçons du passé – Prophylaxie – Restrictions commerciales.



Experiencias en los Estados Unidos de América en materia de prevención y control de brotes de influenza aviar

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Resumen

La epidemiología y el control de la influenza aviar (IA) son cuestiones complejas. En condiciones naturales, el virus se desplaza gracias a la actividad de las aves salvajes, y en las explotaciones agrícolas gracias a la actividad de las personas. En general, en todos los brotes de IA ocurridos en los Estados Unidos de América (EE.UU.) han intervenido virus diseminados por los desplazamientos de aves de corral o el movimiento de excrementos y objetos contaminados por las

propias aves y sus excrementos, pero generalmente la causa concreta de la propagación ha sido distinta en cada caso: el brote de influenza aviar altamente patógena (IAAP) de 1924 atravesó la mitad del territorio de los EE.UU. a bordo de jaulas y vagones contaminados; el brote de IAAP de 1983 fue diseminado por los desplazamientos de personas entre explotaciones y el transporte de aves de corral vivas o muertas, lo que incluye las operaciones de sacrificio sanitario; los brotes de influenza aviar levemente patógena (IALP) ocurridos en distintos estados se extendieron por la descarga parcial de bandadas, el transporte de gallinas sobrantes y/o excrementos o el transporte de aves muertas con fines de transformación.

Hay una dicotomía en torno a los métodos de control de la IA en EE.UU. Los grandes brotes de IALP han afectado sobre todo a pavos del oeste del país, y para luchar contra ellos se han utilizado estrategias de vacunación y comercialización controlada definidas antes del brote de IAAP de 1983. En la parte oriental del país, en cambio, la estrategia de lucha contra la infección lleva la impronta del satisfactorio programa de sacrificio sanitario que se instituyó durante el brote de IAAP de 1983. El autor presenta en un cuadro un resumen de los costos y de las estrategias de control.

Palabras clave

Comercialización controlada – Control – Enseñanzas extraídas – Epidemiología – Influenza aviar – Sacrificio sanitario.



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