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Review

The history of avian influenza

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Abstract

The first description of avian influenza (AI) dates back to 1878 in northern Italy, when Perroncito [Perroncito E. Epizoozia tifoide nei gallinacei. Annali Accad Agri Torino 1878;21:87–126] described a contagious disease of poultry associated with high mortality. The disease, termed "fowl plague", was initially confused with the acute septicemic form of fowl cholera. However, in 1880, soon after its first description, Rivolta and Delprato [as reported by Stubs EL. Fowl pest, In: Biester HE, Devries L, editors. Diseases of poultry. 1st ed. Ames, IO: Iowa State College Press; 1943. p. 493–502] showed it to be different from fowl cholera, based on clinical and pathological properties, and called it *Typhus exudatious gallinarum*. In 1901, Centanni and Savunzzi [Centanni E, Savonuzzi E, La peste aviaria I & II, Communicazione fatta all'accademia delle scienze mediche e naturali de Ferrara, 1901] determined that fowl plague was caused by a filterable virus; however, it was not until 1955 that the classical fowl plague virus was shown to be a type A influenza virus based on the presence of type A influenza virus type-specific ribonucleoprotein [Schäfer W. Vergleichender sero-immunologische Untersuchungen über die Viren der Influenza und klassischen Geflügelpest. Z Naturf 1955;10b:81–91]. The term fowl plague was substituted by the more appropriate term highly pathogenic avian influenza (HPAI) at the First International Symposium on Avian Influenza [Proceedings of the First International Symposium on Avian Influenza. Beltsville, MD. 1981, Avian Dis 47 (Special Issue) 2003.] and will be used throughout this review when referring to any previously described fowl plague virus.

Résumé

La première référence à la grippe aviaire remonte à 1878 dans le nord de l'Italie, lorsque Perroncito [1] décrit une maladie contagieuse et hautement mortelle affectant la volaille. Cette

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maladie, appelée « peste aviaire », a tout d'abord été confondue avec une forme septicémique aigüe de choléra aviaire. Cependant, en 1880, peu après l'énonciation de cette première description, Rivolta et Delprato [2] démontrent que cette maladie possède des propriétés pathologiques et cliniques différentes du choléra aviaire et la nomment *Typhus exudatious gallinarum*. En 1901, Centanni et Savunzzi [3] déterminent que la peste aviaire est causée par un virus filtrable. Cependant, ce n'est qu'en 1955 qu'il est démontré que le virus de la peste aviaire classique est un virus influenza de type A basé sur la présence d'une ribonucléoprotéine spécifique aux virus influenza de type A [4]. Le terme *peste aviaire* a été abandonné au profit de *grippe aviaire hautement pathogène*, plus approprié, lors du premier symposium international sur la grippe aviaire [5] et sera utilisé dans ce compte rendu pour faire référence à l'ancienne appellation.

Mots clés : Virus de la grippe aviaire ; Peste aviaire ; Hautement pathogène ; Faiblement pathogène ; Pandémie de grippe*Keywords:* Fowl plague; Avian influenza; History; Highly pathogenic; Low pathogenic; Waterfowl

Contents

1. Avian influenza viruses: historic background

It is believed that outbreaks of highly pathogenic avian influenza (HPAI) occurred in Italy and other European countries prior to the first description by Perroncito [1] and the differentiation from fowl cholera by Rivolta and Delprato in 1880 as reported by Stubs [2] (Table 1). There are reports of subsequent outbreaks of HPAI in 1894 and 1901 in Italy, which spread with the stock of an itinerary poultry merchant to eastern Austria and Germany and later to Belgium and France [6,7]. The spread of HPAI through Germany was aided by the 1901 Brunswick poultry show, where after the detection of sick birds, the authorities panicked, closed the show and send the affected flock to their places of origin, spreading the outbreak even further. HPAI became endemic throughout Italy and Central Europe until it disappeared around the mid 1930 [8]. However, it is important to note that the lack of appropriate diagnostic tests and the concurrent circulation of Newcastle disease virus [9]

Table 1
Mayor events in the history of avian influenza

Year	Event	Reference
1878	First description of highly pathogenic avian influenza (HPAI) or fowl plague	[1]
1880	Differentiation of HPAI from fowl cholera	[2]
1901	Identification of HPAI as a virus	[3]
1901–1930s	Major outbreaks of HPAI throughout the world	[6,7,10]
1918	Major human pandemic	[72]
1931	First influenza virus isolated (swine)	[73]
1941	Recognition of hemagglutination by influenza viruses	[16]
1942	HPAI and Newcastle disease virus shown to agglutinate red blood cells and to be different serologically	[17]
1955	HPAI virus shown to be a type A influenza virus	[4]
1959	Isolation of a HPAI virus serologically different from the classical fowl plague virus in hemagglutination inhibition test	[25]
1970s	Intensive surveillance of influenza viruses in wild birds and recognition that wild birds harbor all identified subtypes of influenza viruses	[30–34,37]
1971	Classification of influenza viruses based on antigenic properties of the NP (type) and HA and NA (subtype) proteins and the species of origin	[39]
1977–1981	Recognition that the presence of multiple basic amino acids in the HA cleavage site correlates with tissue spread and virulence of AI strains	[74,75]
1978	Recognition that the 1957 (H2N2) and 1968 (H3N2) pandemic influenza viruses aroused by reassortment with AI viruses	[76]
1980	Classification of influenza viruses based on antigenic properties of the NP (type) and HA and NA (subtype) proteins regardless of the species of origin	[39]
1981	First International Symposium on Avian Influenza	[5]
1981	The name highly pathogenic avian influenza is proposed to substitute fowl plague	[5]
1999–2001	H9N2 virus transmission to humans	[64–67]
1997-present	HPAI H5N1 transmission to humans	This issue
2000s	H9N2 becomes endemic in Asia	[63]
2003-present	HPAI H5N1 spreads through Asia, Europe and Africa and becomes endemic in Asia	This issue

among poultry during the early 1900s, makes it impossible to determine which of the two viruses were responsible for the reported outbreaks. By the mid 1900s, HPAI had been reported in most of Europe, Russia, North America, South America, Middle East, Africa and Asia [10,11].

The first outbreak of HPAI in United States occurred during the fall and winter of 1924–1925. The disease first appeared to have caused severe losses in live bird markets in New York City [12]. The disease then spread rapidly to New Jersey, Pennsylvania and Connecticut. In all these instances the outbreaks were initiated after introduction of poultry from the affected areas. The outbreaks were also reported in Indiana, Michigan, West Virginia, Missouri and Illinois during February–April 1925. The outbreak in Indiana was confined to six farms and the outbreaks in Michigan and Missouri were reported in Detroit and St. Louis. The outbreaks in Indiana, Michigan and Missouri were during the February, and were probably due to contaminated rail cars originating from

east coast terminals. In West Virginia there was only one diseased flock occurring at about the same time, however, the origin was not known. The last outbreak of 1925 appeared in Illinois in April. It was restricted to some small flocks in the suburbs of Chicago. By the end of April 1925 this outbreak was controlled, however it reappeared involving a few flocks in New Jersey in 1929. Both these outbreaks were successfully controlled due to restrictions placed on shipping of live poultry, quarantine, depopulation, cleaning, and disinfection. These measures resulted in the eradication of this disease prior to spreading throughout the United States. It is estimated that direct losses associated with the 1924–1925 outbreak were more than $1 million [6,12]. Although the origin of United States outbreaks of HPAI is difficult to predict, it has been speculated that they originated from vials of ''fowl plague'' virus imported from France in September 1923 by an investigator working on filterable agents.

There are only two reports of HPAI outbreaks in the United Kingdom in 1922 and 1929 [13]. However, it is believed that HPAI outbreaks occurred more frequently, but the self-imposed stamping-out policy practice by the farmers aid in limiting the spread of the disease in this country.

During the early days of virology, HPAI virus was used as model agent, specially because of the easy of use of chickens as experimental animals [7]. The association of HPAI with neurological problems, lead to the initial comparison of this virus to rabies. Similarly, the high mortality and severity of the symptoms associated with HPAI gave no indication that the virus was related to human influenza viruses. In 1934, Burnet and Ferry [14] showed that both HPAI and Newcastle disease viruses could be titrated in embryonated chicken eggs, based on their ability to kill the developing embryo. Interestingly, it was not until 1936 that Burnet showed that embryonated chicken eggs could be used for the propagation of influenza viruses [15]. Following the discovery of hemagglutination by influenza viruses [16], Lush [17] showed that the HPAI and Newcastle disease viruses were also able to agglutinate red blood cells, and more importantly, that there was no serological relationship between these two avian viruses. The differences between Newcastle disease virus and HPAI were further supported by the discovery that HPAI were indeed influenza viruses [4] (Table 1).

A new era on the history of AI started in the mid 1900s when less virulent forms of AI viruses were isolated for the first time. The so-called ''N'' virus was isolated from a dead adult chicken in Germany (A/chicken/Germany/49 (H10N7)) [18,19] but was not recognized as an AI virus until 1960 [20]. Similarly, several viruses were isolated from domestic ducks with respiratory diseases in Manitoba, Canada (A/Duck/Canada/52 (H10N7)) [21], Czechoslovakia (A/duck/Czechoslovakia/56 (H4N6)) [22], England (A/duck/England/56 (H11N6)) [23] and Ukranie (A/duck/Ukraine/60 (H11N8)) [24]. Until the mid 1950s, all the HPAI (fowl plague) viruses isolated had been of the H7 subtype. However, in 1959 and 1961 two HPAI viruses of the H5 subtype, producing clinical disease indistinguishable from the traditional fowl plague, were isolated in Scotland (A/chicken/Scotland/59 (H5N1)) [25] and in South Africa (A/tern/South Africa/61 (H5N3)) [26]. This led to the misconception that all H5 and H7 viruses were highly pathogenic. This dogma was later shown to be incorrect when low pathogenic H5 and H7 viruses were isolated from turkeys in Canada (A/turkey/Ontario/66 (H5N9)) [27], Wisconsin (A/turkey/Wisconsin/68 (H5N9)) [28] and Oregon (A/turkey/Oregon/71 (H7N3)) [29]. In addition, during the

1960s several low pathogenic (LP) AI viruses of different subtypes were isolated from turkeys, chickens, ducks, quail, pheasants and partridges [27] with respiratory and reproductive disease providing new light into the great variation existing among influenza viruses.

Indications on the potential role of animal influenza on the origin of human pandemics, led the World Health organization to promote studies on the ecology of these viruses in wild animals as early as 1958 [13]. However, it was not until 10 years later that, serologic surveys of wild birds were used to demonstrate the presence of AI virus infection in wild birds in the USA, Australia and Russia [30–34]. Until 1973, type A influenza viruses had only been reported twice from free-flying birds: common tern (A/tern/South Africa/61 (H5N9)) [26,35] and shearwater (A/shearwater/East Australia/72 (H6N5)) [36]. Since then, AI viruses have been isolated from at least 105 wild bird species of 26 different families as summarized by Olsen et al. [37]. Although many wild bird species can be infected with AI viruses, birds of aquatic environments of the orders Anseriformes (duck, geese and swans) and Charadriiformes (terns, gulls and waders) constitute the major avian AI reservoirs. It is well documented that these AI viruses from wild birds spread, occasionally, to poultry where they can produce LP or HP AI outbreaks. For more detailed information on AI and wild birds see ''The role of waterfowl'' in this issue.

The identification of numerous AI strains and the recognition of wild waterfowl and shore birds as reservoirs of type A influenza viruses, led to a change in their classification. Initially, influenza viruses were classified based on the species of origin and the serological reactivity of the ribonucleoprotein (NP) (type) and hemagglutinin (HA) and neuraminidase (NA) (subtype) proteins by double immunodiffusion reactions (Table 2) [38]. However, the recognition that viruses serologically related to those of humans, swine and equine were also present among wild birds population, led to a unified system of classification (Table 2)

Table 2
Nomenclature for type A influenza virus subtypes

HA		NA	
Before 1980	1980-present	Before 1980	1980-present
H0, H1, Hsw1*	H1	N1	N1
H2	H2	N2	N2
H3, Heq2**, Hav7***	H3	Nav1, Nav3	N3
Hav4	H4	Nav4	N4
Hav5	H5	Nav5	N5
Hav6	H6	Nav1	N6
Hav1, Heq1	H7	Neq1	N7
Hav8	H8	Neq2	N8
Hav9	H9	Nav6	N9
Hav2	H10		
Hav3	H11		
Hav10	H12		
–	H13		
–	H14		
–	H15		
–	H16		

*sw, swine; **eq, equine; ***av, avian.

[39]. Since 1980, four new HA subtypes have been identified, and as surveillance in wild birds increases throughout the world, it can be expected for this numbers to increase.

2. Major highly pathogenic avian influenza outbreaks

While the AI outbreak of 1878 in Italy, which caused extremely high mortality in chickens, was of the highly pathogenic form, the first confirmed outbreak of HPAI was reported in Scotland in 1959 [40]. Since then, more than 28 outbreaks have been recorded worldwide, of which half of them have occurred in the past 10 years (Table 3). The majority of HPAI have shown limited geographical spread, and even self-limited to a single flock of birds (Table 3). However, during the HPAI outbreaks of 1983 in the United States [41], 1994 in Mexico [42], 1994 in Pakistan [43], 1997 in Hong Kong (see this issue), 1999 in Italy [44,45], 2002 in Chile [46], 2003 in The Netherlands [47–50], 2004 in Canada [51,52] and 2003-present in Asia, Europe and Africa (see ''The epidemiology of H5N1 avian influenza'' in this issue) the potential impact of HPAI was demonstrated when the disease became widespread, causing enormous economic losses. However, since 1959, none of the outbreaks has approached the size of the H5N1 epizootic that has extended from Asia to Europe and Africa since 2003 and has become endemic in Asia. In this section, we will review some of the major HPAI outbreaks of the 20th and 21st centuries.

2.1. H5N2 Pennsylvania outbreak

In April 1983, a low pathogenic H5N2 virus (A/chicken/Pennsylvania/1/83) circulated in chickens in Pennsylvania. Later that year, this virus had mutated into a highly pathogenic variant (A/chicken/Pennsylvania/1370/83) causing high mortality in the affected flocks [41]. The low virulent predecessor was unusual in that it had multiple basic amino acids at the HA cleavage site. The virulent strain differed from its predecessor by a nucleotide that caused the loss of a glycosylation site in HA [54], thereby exposing the polybasic HA cleavage site to ubiquitous cellular proteases. This outbreak was eventually controlled by the slaughter of more than 17 million birds with a direct cost of $62 million and indirect costs estimated at more than $250 million [55].

2.2. H5N2 Mexican outbreak

In May 1994, a low pathogenic H5N2 virus (A/chicken/Mexico/26654-1374/94) was isolated from chickens in Mexico. This virus was not eradicated by mass slaughter which allowed the virus to accumulate mutations over several months yielding a highly pathogenic strain (A/chicken/Mexico/8623-607/94) with a series of basic residues at the HA cleavage site [56]. Vaccination was implemented after the emergence of highly pathogenic strains, which prevented the spread of HPAI; however, low pathogenic H5N2 strains continue to circulate, as do genetically related viruses in the neighboring countries of El Salvador and Guatemala. Control strategies in Mexico relied solely on vaccination and not on accompanying measures like monitoring and quarantine. This

Table 3
Important outbreaks of HPAI documented since 1959*

	HPAI virus	Subtype	Species affected	Approximately number of birds culled
1	A/chicken/Scotland/59	H5N1	Chicken	1 small farm
2	A/tern/South Africa/61	H5N3	Common tern	1300
3	A/turkey/England/63	H7N3	Turkey	29,000
4	A/turkey/Ontario/7732/66	H5N9	Turkey	8000
5	A/chicken/Victoria/76	H7N7	Chickens, ducks	58,000
6	A/chicken/Germany/79	H7N7	Chicken and goose	1chicken and 1 goose farm
7	A/turkey/England/199/79	H7N7	Turkey	9000
8	A/chicken/Pennsylvania/1370/83**	H5N2	Chicken turkey	17,000,000 chickens and turkeys
9	A/turkey/Ireland/1378/83	H5N8	Turkey	307,000, chickens, turkeys and mostly ducks
10	A/chicken/Victoria/85	H7N7	Chicken	240,000
11	A/turkey/England/50-92/91	H5N1	Turkey	8000
12	A/chicken/Victoria/1/92	H7N3	Chicken	18,000 broiler breeders, ducks
13	A/chicken/Queenland/667-6/94	H7N3	Chicken	22,000
14	A/chicken/Mexico/8623-607/94**	H5N2	Chicken	Millions?
15	A/chicken/Pakistan/447/94**	H7N3	Chicken	>6,000,000
16	A/chicken/NSW/97	H7N4	Chicken	160,000 chickens, emus
17	A/chicken/Hong Kong/97	H5N1	Chicken, duck	1,500,000 chickens and other domestic birds
18	A/chicken/Italy330/97	H5N2	Chicken	8000 chickens, turkeys, guinea-fowl, ducks, quail, pigeons, geese, pheasant
19	A/turkey/Italy/99**	H7N1	Turkey	14,000,000 chickens, turkeys, guinea-fowl, quail, ducks, pheasants, ostriches
20	A/chicken/Chile/02	H7N3	Chicken	700,000 chickens, turkeys
21	A/grey heron/Hong Kong/861.1/02	H5N1	Wild birds	Outbreak in wild birds; over 800,000 domestic birds were culled
22	A/chicken/Netherlands/03**	H7N7	Chicken	>34,000,000
23	A/chicken/Asia, Europe and Africa/03-07**	H5N1	Chicken, duck	100s of millions
24	A/chicken/Texas/04	H5N2	Chicken	6600
25	A/chicken/Canada/04**	H7N3	Chicken	16,000,000
26	A/ostrich/South Africa/04	H5N2	Ostrich	30,000
27	A/chicken/North Korea/05	H7N7	Chicken	219,000
28	A/turkey/England/07	H5N1	Turkey	160,000

* Adapted from [63]; **Outbreaks with significant spread to numerous farms, resulting in great economic losses. Most other outbreaks involved little or no spread from the initially infected farms.

may have resulted in the evolution of H5N2 variants for which current vaccines are not efficacious. A similar situation is thought to be occurring in Asia with the H5N1 virus. Therefore, caution should be exercised on the use of vaccines on the control AI infections.

2.3. H7N3 Pakistan outbreaks

In 1994, an outbreak of highly pathogenic virus of H7N3 subtype occurred in poultry farms in Pakistan. This outbreak killed 3.2 million birds and was eventually brought under control by vaccination [43,44]. However, in 2001, 2003 and 2004, despite vaccination, viruses of low and high pathogenicity continued to emerge resulting in the death of over 10 million birds.

2.4. H7N1 Italy outbreak

In March 1999, a low pathogenic virus of the H7N1 subtype was isolated from chickens in Italy [45,46,57]. The virus was not eradicated and the infection spread, resulting, in the emergence of a highly pathogenic isolate by December of 1999. More than 14 million birds had to be destroyed to control the outbreak. The reappearance of the low pathogenic H7N1 virus in August 2000 was controlled by depopulation followed by a vaccination and was finally eradicated in May 2002. The vaccine used was based on inactivated H7N3 virus to allow differentiation of infected and vaccinated animals (DIVA) [58] based on neuraminidase gene. Vaccination, in combination with intensive monitoring, enabled the eradication of this H7N1 virus from Italy.

2.5. H7N3 Chile outbreak

Although there are reports of HPAI outbreaks in South America during the early 1900s, it was not until 2002 that the first AI virus was isolated on this continent. In May 2002, a LPAI H7N3 virus (A/chicken/Chile/176822/02) was isolated from a broiler breeder flock in Chile. In June, a HPAI virus of the same subtype (A/chicken/Chile/4957/02) was obtained from the same flock [47], leading to the slaughter of 2 million birds. The HPAI virus arose from the original low pathogenic isolate by recombination events that resulted in the insertion of 10 amino acids from the NP protein [59] into the HA cleavage site. The economic losses associated with this outbreak are estimated around $31 million.

2.6. H7N7 The Netherlands outbreak

In February 2003, HPAI of the H7N7 subtype caused outbreaks in layer farms in The Netherlands. The outbreak ultimately spread to Belgium and Germany but was brought under control by mass slaughter [48–51]. The H7N7 2003 outbreak resulted in the destruction of 30 million birds in The Netherlands (one quarter of the country's poultry stock), with 2.7 million destroyed in Belgium and 400,000 in Germany for a total of over 33 million birds. The total costs of this outbreak was estimated at €750 million. Interestingly, an unexpectedly high number of transmissions of H7N7 virus to people directly involved in handling infected poultry, as well as human-to-human transmission was observed during this outbreak [60].

2.7. H7N3 Canada outbreak

Between 17 February and 18 May 2004, an outbreak of AI due to a H7N3 virus occurred among poultry in the Fraser Valley of British Columbia, Canada [52,53].

Within days, the virus causing this outbreak had changed from low to high pathogenicity on the index farm by recombination events that resulted in the insertion of seven amino acids from the M1 protein [61] into the HA cleavage site. Despite enhanced biosecurity measures, the virus was not contained to the index farm and eventually spread to over 40 commercial poultry facilities before massive depopulation efforts enabled its eradication. A total of 14 million birds from 410 commercial farms and 553 backyard premises were depopulated in an effort to halt circulation of the virus. Economic losses were estimated at more than $300 million. During this outbreak, two poultry workers involved in depopulation were confirmed infected with the H7N3 virus [62].

2.8. H5N1 outbreaks

Low levels of HPAI H5N1 viruses were present in East Asia from 1996 to 2003. Interestingly, from December 2003 to February 2004, eight countries in East and South East Asia reported outbreaks of HPAI H5N1. Since then, the virus has spread across Asia into Europe and to Africa. Detail information on H5N1 outbreaks since 1996 is provided in ''Epidemiology of H5N1 Avian Influenza'' in this issue.

3. Current situation on low pathogenic avian influenza and their impact on public health

Avian influenza surveillance studies initiated in the 1970s have resulted in the identification of 16 hemagglutinin and nine neuraminidase subtypes. These AI viruses are occasionally transmitted to domestic poultry resulting in the emergence of LPAI or HPAI viruses. Although HPAI viruses and LPAI of the H5 and H7 subtypes receive the most attention, the impact of other LPAI viruses is more difficult to determine and should not be ignored. For example, outbreaks due to LPAI H9N2 viruses have been reported during the last 10 years in Germany, Italy, Ireland, South Africa, USA, Korea, China, South East Asia, Middle East, Iran and Pakistan and H9N2 is now considered enzootic throughout Asia [63]. H9N2 viruses have been sporadically introduced into humans causing flu-like illnesses indistinguishable from the symptoms of common influenza caused by human H1N1 and H3N2 viruses [64–67] but there has been little evidence of human-to-human transmission [68]. In addition, as H9N2 viruses are not highly pathogenic for poultry, the extent of infection in both poultry and humans is likely to remain underappreciated. Given the predicted affinity of H9N2 viruses for human influenza receptors [44,69,70], the repeated introduction of H9N2 virus into the human population provides increased opportunities for reassortment with human viruses, increasing the likelihood that this virus will acquire the ability of human-to-human transmission. Therefore, while the impact of H5N1 disease in poultry and in humans in Asia has attracted most attention, it is important to include H9N2 viruses high on the list of candidate human pandemic strains and to carry out more studies on its ecology and pathogenesis.

4. Conclusions

We have come a long way since the first description of ''fowl plaque'' in 1878. It was not until much later that the potential role of AI viruses on public health was recognized. For example, it is now known that the 1918 pandemic virus was of avian origin and that the 1957 and 1968 pandemic viruses aroused by recombination between human and avian viruses. The increased transmission of HPAI H5N1, H7N7 and H7N3 and LPAI H9N2 to the human population has put the scientific community on the lookout for the next influenza pandemic. We know that for an AI virus to infect humans there needs to be close contact with infected birds. Therefore, through effective vaccination and eradication programs we should be able to mitigate the probability of emergence of the next pandemic. In addition, we must continue our efforts on better understanding the history, ecology and biological properties of AI viruses.

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