

Chapter 16

**THE IKARUS SYNDROME IN THE POULTRY INDUSTRY
AND THE CONCERN ABOUT A NEW GREAT
INFLUENZA PANDEMIC: WHAT TO LEARN FROM
IKARUS' CRASH**

Sievert Lorenzen

Zoological Institute, University of Kiel, D-24098 Kiel, Germany

ABSTRACT

Evidence is presented that, with respect to the development and spread of highly pathogenic avian influenza viruses (AI viruses), wild birds and small-scale poultry operations are much more biosecure than large-scale industrial poultry operations. Based on reports that more than 250 humans have succumbed to highly pathogenic strains of the avian influenza H5N1 Asia, there is concern that one strain could adapt to humans to cause a disastrous pandemic surpassing the Great Influenza of 1918. According to evidence available, the Great Influenza was largely man-made, and at least two young persons of the 250 supposed victims of H5N1 Asia actually died due to medical treatment against this virus rather than of avian influenza. The new term *Ikarus Syndrome* is explained, pointing to the problem that a glamorous series of successes may increase carelessly taken risks of crashing. The poultry industry seems to suffer from this syndrome. Measures are suggested of how to avoid a crash in the poultry industry and lower the risk of influenza pandemics that might be caused by a highly pathogenic strain of the AI virus H5N1.

INTRODUCTION

For biologists, 2009 is a Darwin year. Charles Darwin was borne in 1809, and his most fundamental work, *The Origin of Species by Means of Natural Selection*, appeared in 1859. It is based on the crucially important wisdom that unlimited growth is impossible when based

upon the dissipation of resources. The reason for this is that the availability of all resources is limited in our limited world. Therefore, the further the limits of growth are exceeded, the greater will be the risk of catastrophes such as a severe influenza pandemic.

We can find this wisdom even in the Greek mythology. Prior to flying away from Crete to the Greek mainland by using wings made of feathers, wax and thread, Daedalus instructed his son Ikarus not to fly too low to prevent his wings from touching the water surface and not to fly too high to prevent the wax of the wings from getting melted by the sun's rays. Any disregard of these instructions would be disastrous. To put Daedalus' instructions into other words: If you do not want to not perish, you must work sufficiently hard without getting slaphappy. Ikarus did not take his father's words seriously. He flew higher and higher, his success exceeded all of his expectations, and made him so giddy that he did not notice that the wax of his wings had begun to melt. When enough wax had melted, Ikarus crashed. Did he fall victim to the sun's rays? No, he had fallen victim to his complacency. If he had only listened to his father's instructions, the sunrays would have been harmless to him. The syndrome of exceeding limits of growth carelessly, thus increasing the risk of a crash, is therefore referred to as the Ikarus Syndrome in the following.

The global finance industry, which had been infested with the Ikarus Syndrome, has just experienced its crash. Prior to this crash, it had lived through many years of glamorous success without noticing that the "wax of its wings" had begun to melt. So, the finance industry has not fallen victim to some kind of "sunrays" but rather to its uncured Ikarus Syndrome.

THE IKARUS SYNDROME IN THE POULTRY INDUSTRY

The poultry industry also looks back on a remarkable success story. It has grown impressively in the last 30 years. It achieved much success by increasing the numbers of farmed birds; by crowding as many farm birds as possible into limited space; by growing big, really big; through increasing degrees of industrialization; and by integrating many farms, slaughterhouses, and feed mills, etc., into mighty, vertically-integrated companies. By these means, poultry products became cheap mass products.

However, the "wax of its wings" has begun to melt. Increased industrialization has had severe effects on farm birds, such as overcrowding stress and increased susceptibility to diseases, and created severe problems of hygiene within the farmhouses. As a result, a daily poultry mortality of up to 1% of the flock is now regarded as quite normal. It was hoped that the hygiene problem would be solved by adopting the *all-in-all-out* principle which allows a farmhouse to be cleaned thoroughly only in the short period between removal of the older poultry stock and bringing in the younger one. But in between, during the fattening period, poultry and hygiene problems grow jointly. Particularly affected are long-lived farm birds—such as turkeys—that need a relatively long time (> 100 days) to reach their final weight. This obverse side of the poultry industry creates the most suitable conditions for low pathogenic (LP) strains of avian influenza viruses (AI viruses) to evolve into highly pathogenic (HP) strains. That is, poultry factories also serve as disease factories. This was evidenced strikingly in Chile in 2002, when a low pathogenic AI H7N3 virus evolved rapidly (from May to June 2002) into a highly pathogenic strain within a single broiler breeder flock (Suarez et al., 2004).

In nature, pathogens are subject to a fundamental tradeoff between virulence (making the host sick) and transmission (infecting new hosts) (Muzaffar et al., 2006). This tradeoff is invalidated in the poultry industry, where populations of pathogens may evolve into highly virulent strains without reducing their chances of transmission, which is taken over by the many movements of the integrated poultry industry (Sharkey et al., 2007).

Through these practices, the poultry industry has created a further problem which may affect the human population. According to the World Health Organization (WHO, 2009), 407 confirmed human cases of avian influenza A/(H5N1) resulting in 254 deaths were reported to the WHO up to 27 January 2009. Fortunately, no strain of H5N1 adapted to humans has yet been found that could be transmitted from human to human, but there is considerable concern that such a strain could emerge in the next few years and cause a pandemic (worldwide epidemic) in the human population with up to 150 million deaths (e.g., Liu et al., 2006; Chen et al., 2006). Should this pandemic emerge, it would clearly be man-made.

What is to be done? Prior to answering this question, the circumstances that have caused the AI problem and the pandemic concern need to be analysed in more detail.

EVOLUTION AND SPREAD OF HIGHLY PATHOGENIC STRAINS OF THE AI VIRUS H5N1 ASIA

The evolution of highly pathogenic strains of the AI virus H5N1 Asia began in Southeast Asia. It was promoted by combining practices of industrial poultry farming with the tradition of selling live poultry destined for slaughter on markets: If birds infected with AI are brought to a market, they may infect there other birds. If those newly-infected birds are not sold on the market, they are brought back to their farms, carrying a high risk of infecting other birds within their flock. Therefore, the markets served and still serve (Amonsin et al., 2008) as exchange sites for AI viruses. Additionally, following advice from the Food and Agriculture Organization (FAO), chicken waste was released directly into adjacent waters to fertilize them. That way, highly pathogenic AI viruses could be released directly into waters through which wild water birds and outdoor poultry could become infected. Even dead chickens were released as fish food into the water (Williams 2006).

The conditions described above allowed the prototype of the highly pathogenic AI virus H5N1 Asia to evolve. It was discovered first in 1996 on a goose farm in Guangdong in southern China and was designated *GS/GD* (Goose/Guangdong) (Xu et al., 1999). Li et al. (2004) found evidence that this strain must have repeatedly come into contact with other influenza viruses, thereby exchanging genes with them (gene reassortment). A strain hence derived from *GS/GD* was responsible for the avian influenza epidemic in poultry holdings in Hong Kong (close to Guangdong) in 1997 (Guan et al., 2002). Over 1.5 million poultry were culled on that occasion, but the virus survived and continued to evolve in southern China, where it cleaved into 12 variants (Li et al., 2004). One of them, whose genotype the authors named Z, became dominant between January 2002 and 2004.

In 2004 and 2005, Chen et al. (2006) observed the diversity of H5N1 strains in southern China to be greater than anywhere else in the world, although the incidences were low (0.26% for chickens, 1.83% for domestic ducks, 1.90% for domestic geese, 0.34% for wild ducks). Additionally, many other avian influenza subtypes were found to circulate both in poultry markets (H3, H6, H9, H11 and others) and among migrating wild ducks (H1, H3, H4, H5,

H6, H10); clinically, all infected birds looked healthy at the moment of taking the samples. Faced with the extraordinarily high diversity of AI viruses in southern China, the authors concluded that this region could become the epicentre from where a terrifying pandemic of human influenza could spread all over the world.

Apart from circulating in southern China, AI viruses of the genotypes Z and V were also found 600 km further north on the huge Poyang Lake (surface area up to 5070 sq km depending on the water level), where they were isolated in January and March 2005 from six birds out of a sample of 4,316—apparently healthy—overwintering migratory ducks (not further identified) and from poultry of farms nearby the lake (Chen et al., 2006). The authors found evidence that the viruses had spread from the domestic to the wild birds rather than the other way around.

In May 2005, 1,700 km further west around the even larger Qinghai Lake (5,700 sq. km), a salt lake at 3,200 m altitude with no outlet, some 6,000 wild water birds—mostly bar-headed geese—died from a highly pathogenic avian influenza caused by a new H5N1 strain, whose genotype was a mixture of the Z and V strains previously found at the Poyang Lake (Yasué et al., 2006; Chen et al., 2006). It is unknown where and how the Qinghai strain originated.

Only the Qinghai strain of H5N1 Asia (clade 2.2. of the phylogenetic H5N1 Asia tree) (WHO/OIE/FAO 2008) spread westwards to reach Europe in October 2005 and Africa in February 2006 (Kilpatrick et al., 2006), whereas all other H5N1 Asia strains (clades 0, 1, 2.1., 2.3, 2.4, and 3 to 9 of the phylogenetic tree) remained confined to the far east.

Assuming that the Qinghai Lake was far away from the poultry industry, Chen et al. (2006) blamed migratory wild water birds for having carried the Qinghai strain or its two precursors from the Poyang Lake to the Qinghai Lake and further to Europe and Africa, not in a single journey, of course, but in a sort of relay race, causing outbreaks of Avian Influenza in various places along the route through Mongolia, Siberian Russia, Europe, and Africa.

This hypothesis collapsed like a house of cards once it was realized that, from 2003 on, bar-headed geese were reared on various farms close to the Qinghai Lake, some for farming purposes, others destined for release into the wild (Butler, 2006). The deaths of large numbers of wild bar-headed geese in 2005 occurred close to these farms. Furthermore, tourism and economic development have been heavily promoted in the region. A railway links the Qinghai Lake with the large town Lanzhou situated 170 km away, which is a hub for traffic via air, railway and highways; particularly, there are routes linking Lanzhou with the Poyang Lake in the East and with Siberia and Europe in the West. Along these routes, AI viruses were found, whose HA-genes matched that of the Qinghai type to 99.6 to 99.8 % (Petermann, 2008).

The new findings strongly support the hypothesis that the H5N1 Qinghai strain was spread from East to West by movements of poultry or poultry products rather than by migratory birds.

The same holds for spreads within Europe. Most spectacular was a case in January 2007, when a specific H5N1 strain struck first two goose farms in Hungary and two weeks later—1,300 km away—a turkey farm in Suffolk (Southeast England). Initially, European authorities blamed migratory birds for having carried the virus from Hungary to the English factory farm, although migration of wild birds was not observed in that midwinter time. Later, an epidemiological study by a group from the British government revealed that the owner of the farm in Suffolk regularly received semi-processed turkey meat from the H5N1-affected

Hungarian area for further processing in his slaughter house adjacent to his turkey farm affected. Hence, the British group concluded that the H5N1 virus was most likely introduced to England via importation of turkey meat from Hungary (DEFRA, 2007).

Despite this and equivalent other evidences, authors of the famous German Friedrich-Loeffler-Institute insisted and still insist that “wild birds play an important role in the global infection affairs and in the spread of HPAIV H5N1” (Schoene et al., 2009).

WILD BIRDS AND OUTDOOR POULTRY SUFFER FROM INDUSTRIAL POULTRY FARMING, NOT THE CONTRARY

According to multiple evidences, highly pathogenic AI viruses may be released from poultry factories into nature where they can infect wild birds and free-ranging poultry. The other way around, there is no conclusive evidence that wild birds or outdoor poultry infected with highly pathogenic AI viruses could have introduced these viruses directly or indirectly (via contaminated feed or straw) into indoor poultry operations, although infected wild birds were evidenced to have carried such viruses over distances of up to 1,000 km until they died (Petermann, 2008; Schoene et al., 2009).

How biosecure wild birds and backyard farming are is impressively demonstrated in Laos (GAIN 2005): Although this country is situated sandwich-like between Vietnam, Cambodia and Thailand, who have suffered much from bird flu outbreaks, Laos was affected only insignificantly. The few outbreaks occurred primarily on commercial enterprises in metropolitan areas and secondarily in smallholder flocks nearby. The commercial enterprises were integrated with foreign poultry companies, importing day-old chicks mainly from Thailand. The measure Laos took against bird flu was to cull all birds within 3 km of identified outbreak sites and to ban imports of day-old chicks and other poultry products from neighbouring countries struck by bird flu. No measures were taken against rural poultry flocks which account for 87 % of the total poultry production in Laos. These birds are bred, raised and consumed locally. They are not fed with imported feed and are allowed to run freely among village homes, where they have frequent chances to mix with wild birds. They remained unaffected by bird flu. Hence, “backyard farming is a solution, not the problem” (GRAIN 2006 a).

In view of the evidence referred to, it is unfair to point the finger of blame at migratory birds and poor people’s backyard birds saying: “We cannot control migratory birds but we can surely work hard to close down as many backyard farms as possible” (Margaret Say, Southeast Asian director for the USA Poultry and Egg Export Council, see GRAIN 2006 a), or to state: “Charoen Pokphand (CP) will succeed in turning a crisis into an opportunity of development” (Sooksunt Jiumjaiswanglerg, Präsident der CP Vietnam Livestock, see GRAIN 2007). When bird flu broke out in Egyptian industrial poultry farms, Egypt’s prime minister took the same line and said: “The world is moving towards big farms because they can be controlled under veterinarian supervision ... The time has come to get rid of the idea of breeding chickens on the roofs of houses” (GRAIN 2006 b), a plan which was realized with a military-style cleansing operation.

By television, we all became witnesses of such actions: Men in white rubber suits and gas masks grasped chickens in rural villages and put them live into plastic sacs for destruction,

while wild birds flying across the sky were blamed for being carriers of AI viruses. Pictures from industrial poultry practices were usually suppressed in such reports.

As a sad result, the senseless mass slaughters of free-ranging poultry have deprived very many poor people of their main source of animal protein. That way, the problem of hunger in the world was increased.

THE PROBLEM OF BIOSECURITY AND ITS MISMATCH IN GERMANY

In Germany, the Friedrich Loeffler Institute (FLI) advises the Germany government on animal diseases. By doing so, the interests of industrial farmers were apparently protected against those of small holders. Only one example may serve as an illustration (Lorenzen 2008): From June to November 2007, a novel strain of the AI virus H5N1 Asia (subclade 2.2.3 in the phylogenetic tree; see Starick et al., 2007) struck wild birds and industrial poultry operations in the Czech Republic, central Germany and France. Outbreaks occurred over a distance of 900 km from east to west (Newman et al., 2007). Germany's largest duck farm in Wachenroth (Bavaria) was affected (outbreak on 24 August 2007), and 285 black necked grebes (*Podiceps nigricollis*) fell victim to the virus on the Kelbra water reservoir in Thuringia/Saxony-Anhalt (August 2007) (Petermann, 2007).

These incidents prompted the FLI (15 October 2007) to blame exclusively wild birds for having spread the novel virus strain, because "at least an indirect causal involvement of wild birds in the latest outbreaks of HPAIV-infections cannot be excluded. The risk that wild birds can introduce HPAIV H5N1 into domestic bird flocks is therefore considered to be *high*." This argument is utterly meaningless, because other causes for the above-mentioned outbreaks "cannot be excluded" either, for example that the new virus strain was spread by human activities. Although the FLI did not disprove this possibility, it insisted that "the risk of transmission as a result of movements of people and vehicles appears to be *negligible*" and, hence, "deserves no further consideration".

Based on this error of judgement, the German government decided (Avian Bird Flu Order of 18 October 2007): "Owners of poultry must keep their birds in closed barns or in enclosures sealed above and at the sides against the entry of wild birds (safety device)." Exceptions to this order can and must be granted by local authorities, "provided that efforts to combat avian influenza are not hindered, and an outbreak of this epidemic must not be feared". Obviously, owners of factory farms are not affected by this order, although factory farming is heavily involved in the evolution and spread of highly pathogenic H5N1 viruses. By its misleading argumentation, the FLI spared the German poultry industry unwanted control measures.

There is much talk of biosecurity in connection with epizootics, of which avian influenza is only one. Biosecurity is understood as "any practice or system that prevents the spread of infectious agents from infected to susceptible animals, or prevents the introduction of infected animals into a herd, region, or country in which the infection has not yet occurred" (Otte et al., 2007). In other words, biosecurity measures are aimed at preventing the spread of an epizootic. According to the evidence presented herein, wild birds, free-ranging poultry and independent farms reach a much higher level of biosecurity than industrial poultry farms, particularly if the latter are integrated into the network of big companies.

A PANDEMIC CAUSED BY H5N1? A RETROSPECTIVE VIEW ON THE 1918 INFLUENZA PANDEMIC

The 1918 influenza pandemic, also known as the Great Influenza, the Spanish Influenza or the mother of all pandemics (Taubenberger & Morens, 2006), caused 20 to 50 millions deaths and led John Oxford (2004, in the famous scientific journal, *Nature*) to accuse nature of being “the greatest bioterrorist of our world”, who had killed “more people than the Nazis, more than the atomic bomb, and more than the First World War” by making use of “the twentieth century’s weapon of mass destruction”—influenza. We all should be cautious with such foolhardy statements, particularly when they refer to the Great Influenza. We all owe our life to nature. We need to live with nature rather than against it. We need to accept that we cannot eradicate influenza viruses. Instead, we need to accept them as natural components of our environment. What we can do at most is to prevent low pathogenic influenza virus strains from evolving into highly pathogenic strains in our poultry operations and, if they should emerge, to take effective rather than ineffective measures against them.

The Great Influenza swept over the continents in three waves (Taubenberger & Morens 2006). The first and smallest one started in March 1918 “in Kansas and in military camps throughout the US” (Billings, 1997). The second and biggest wave started in September 1918, and the third and medium-sized in February 1919. In contrast to the first wave, the second and third were characterized by their “rapid progressions from uncomplicated influenza infections to fatal pneumonia” (Taubenberger & Morens, 2006). Mainly affected were Europe, the USA and India (Billings, 2005). The geographic origin of the Great Influenza is still a matter of debate (Oxford, 2004): It might have originated in army camps in the US, from where it was transported to Europe in 1918, when the US Army entered into the final stage of World War I. Alternatively, it might have originated in British army camps in France and Britain in 1916 and 1917, when so many soldiers had to bear bad and brutal conditions of life. The causative agent isolated from victims of the second Great Flu wave was identified to be a strain of the avian influenza virus H1N1 adapted to humans (Taubenberger & Morens, 2006)

Extraordinary characteristics of the Great Influenza were the following four paradoxes:

1. Unlike “normal” influenza, the Great Influenza produced rapid deaths (Billings 1997):

“People were struck with illness on the street and died rapid deaths. One anecdote shared of 1918 was of four women playing bridge together late into the night. Overnight, three of the women died from influenza (Hoagg). Others told stories of people on their way to work suddenly developing the flu and dying within hours. (Henig). One physician writes that patients with seemingly ordinary influenza would rapidly “develop the most viscous type of pneumonia that has ever been seen” and when cyanosis appeared in the patients, “it is simply a struggle for air until they suffocate” (Grist, 1917). Another physician recalls that the influenza patients “died struggling to clear their airways of blood-tinged froth that sometimes gushed from their nose and mouth, (Starr, 1976).”

According to Bauer (1957), “The death rate appeared to parallel the pneumonia attack rather than that of influenza itself.”

2. Classically, influenza causes relatively high mortality peaks in very young and very old, while comparatively low death frequencies are found at all ages in between. In contrast, the Great Influenza caused much more deaths than any other influenza, and high death frequencies were reported not only from very young and very old, but from young adults (15 – 40 years of age) as well. Many of these young adults, well trained soldiers included, were completely fit and healthy days before their rapid death (Collins, 1957; Taubenberger & Morens, 2006). Graphically, the curve of influenza deaths by age at death is U-shaped in the case of “normal” influenza, while it was W-shaped in the case of the Great Influenza (Collins, 1957) (Fig. 1).

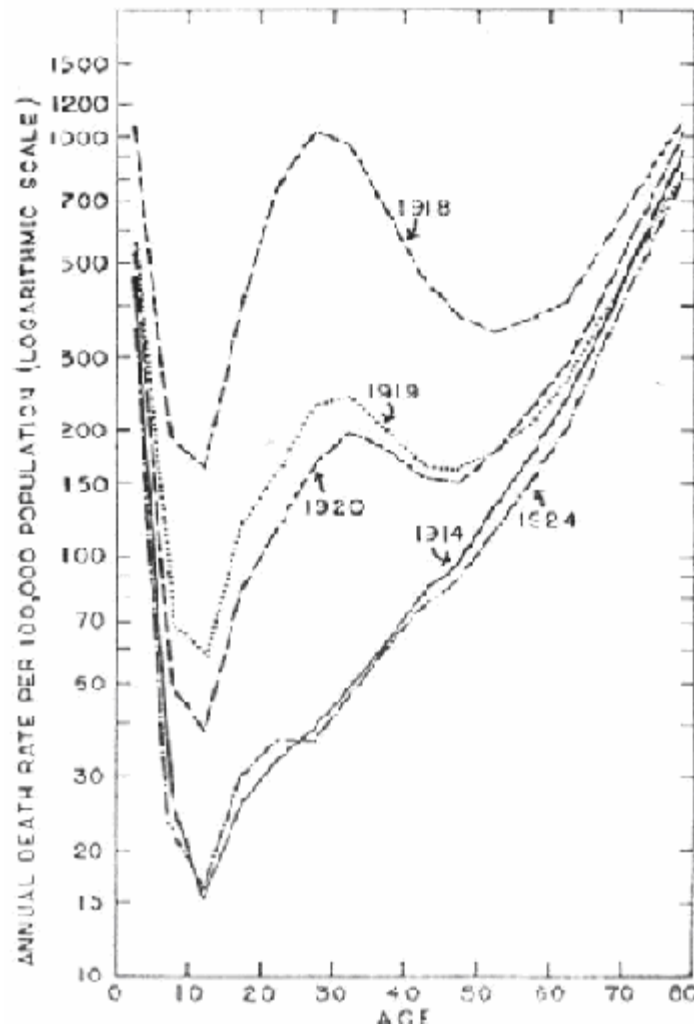


Figure 1. A singular feature of the 1918 influenza pandemic was that not only the very young and the very succumbed to it, but very many young adults as well. As a result, the curve of influenza deaths by age at death is W-shaped in the case of the 1918 influenza pandemic rather than U-shaped as in the case of “normal” influenza epidemics. Taken from Collins (1957).

3. Normally, a pandemic is a highly infectious disease. That is, the infectious agent can be transmitted rather easily from sick to healthy humans. In the case of the Great Influenza, however, five attempts failed to demonstrate the sick-to-well transmission (Cannel et al., 2008). Volunteers in these five experiments were seronegative incarcerated soldiers who “were repeatedly exposed to hospital patients exhibiting influenza-like symptoms in an attempt to make them contract the disease. ... [All] 118 men failed to develop influenza” (Gernhart, 1999). If at all, they developed only minor illness (Cannel et al., 2008).
4. In 1918 and 1919, very many US soldiers and civilians were vaccinated against the Great Flu. Unexpectedly, however, increasing vaccination rates were translated into increasing rather than decreasing illness rates (Cannel et al., 2008). This paradox was confirmed by personal experiences of McBean (1997). Her family had refused all 14 to 20 vaccinations that were strongly recommended at that time. The whole family did not contract the Great Influenza, although the parents went from house to house to look after the sick. She added: “As far as I could find out, the flu hit only the vaccinated. Those who had refused the shots escaped the flu.” As a result, McBean suggested that millions of people had fallen victim to a vaccination catastrophe.

McBean’s conclusion permits to explain the four paradoxes referred to much more consistently than other hypotheses suggested that deal, for instance, with intrinsically high virulence of the H1N1 strain (Taubenberger & Morens, 2006), vitamin D deficiency (Cannell et al., 2008) or the bad battle conditions during World War I (Ahmed et al., 2007). Rather than to explain the four paradoxes, these hypotheses produce additional ones. Hence, Oxford’s accusation of nature to be “the greatest bioterrorist of our world” lacks substantial support, as the Great Influenza appears to be a largely man-made catastrophe. If at all, man can be accused to be “the greatest bioterrorist of our world”.

This suggestion does not invalidate the concern that highly pathogenic strains of the AI virus H5N1 might cause a terrible human pandemic. However, the analysis of the Great Influenza may advise us to approach reports on fatal human cases of H5N1 avian influenza with scientific scepticism. How urgent that is will be underlined by the following two cases.

1. “On 9 May, 1997, a previously healthy 3-year-old boy, who was resident in Hong Kong, developed a sore throat, dry cough and fever. He was diagnosed with pharyngitis and prescribed antibiotics and aspirin. The child continued to be symptomatic and febrile and was hospitalized on 15 May. ... His laboratory tests were most remarkable for leukopenia (2000 white blood cells per cubic millimeter). ... The next day, he was transferred to another hospital, where he developed progressive respiratory distress associated with hypoxemia, consistent with progressive distress syndrome. He also became increasingly unresponsive. ... Despite mechanical ventilation and broad antibiotic coverage, the child died on 21 May with several complications, including respiratory failure, renal failure, and disseminated intravascular coagulopathy” (Subbarao et al., 1998). Shortly before he died, on 19 May 1997, a tracheal aspirate sample was taken, from which an avian H5N1 influenza virus was isolated.
2. In Thailand, a 6-year-old boy suffered from “a progressive viral pneumonia that led to acute respiratory distress syndrome and death 17 days after the onset of illness. He

was initially treated with multiple broad-spectrum antimicrobial agents. Virological diagnosis of H5N1 infection was made on day 7 of illness. After oseltamivir became available in Thailand, he was treated on day 15 of his illness with this agent until he died. He was also treated with methylprednisolone on day 15 until death and with granulocyte colony-stimulating factor for leukopenia from day 5 to day 10 of illness” (Uprasertkul et al., 2005).

The two cases give rise to the hypothesis that the little patients might have succumbed to the medical treatment against the H5N1 virus rather than to the virus itself, as even laymen know that antibiotics are used to combat bacteria rather than viruses and that cocktails of strong medicines may produce unwanted harmful to fatal side effects.

WHAT WE CAN LEARN FROM IKARUS’ CRASH TO MASTER THE INFLUENZA PROBLEM

Ikarus might have avoided his crash if he had followed his father’s instructions and had not flown too high, thus not giving the sunrays any chance of melting the wax of his wings.

To cure the poultry industry from its Ikarus Syndrome and, hence, to master the influenza problem, biosecurity appears to be the most appropriate measure to be taken. The costs this measure will certainly be lower than costs for culling millions of birds and for fighting against a pandemic should it emerge by the adaptation of a highly pathogenic strain of the AI virus H5N1 Asia to humans.

Measures to increase the biosecurity of the poultry industry need to include the following ones:

- Both the numbers of birds per farmhouse and per square metre need to be reduced noticeably to reduce the birds’ susceptibility to infectious diseases.
- Instead of cleaning farmhouses thoroughly only in the short period between *all-out* and *all-in* of poultry flocks, they need to be cleaned thoroughly also during *all-in* periods. Such additional cleanings are particularly important in the case of rather long-lived birds such as turkeys for cutting short the chains of infection that low pathogenic AI virus strains need to evolve into highly pathogenic strains
- The degree of networking within the poultry industry needs to be lowered, and efforts should be encouraged to grow poultry in independent operations, many smallholdings of poultry included. This measure serves to reduce the risk of spreading AI viruses over short and long distances, thus avoiding large AI outbreaks and endemic AI infections (Sharkey et al., 2007).
- The national and international poultry trade, which is essentially out of control, needs to be controlled rigidly to prevent AI-viruses to be spread by trade movements suggested to be legal.
- When humans are reported to have died from an H5N1 virus strain, information on the medical treatment is needed for permitting an examination of whether these humans might have succumbed to the virus or to the medical treatment against it.

Once the poultry industry will be cured of its Ikarus Syndrome, there will be no need to worry any more about a catastrophic influenza pandemic that might be caused by a highly pathogenic strain of the AI virus H5N1 Asia.

REFERENCES

- Ahmed, R., Oldstone, M.B.A., Palese, P. (2007): Protective immunity and susceptibility to infectious diseases: lessons from the 1918 influenza pandemic. *Nature Immunology* 8, 1188-1193.
- Amonsin, A., Choatrakol, C., Lapkuntod, J. et al. (2008): Influenza virus (H5N1) in live markets and food markets, Thailand. *Emerging Infectious Diseases* 14, 1739-1742.
- Bauer, C.C. (2005): The Pandemic of Influenza in 1918-1919. www.history.navy.mil/library/online/influenza%20pan.htm
- Billings, M. (2005): The Influenza Pandemic of 1918. <http://virus.stanford.edu/uda>
- Butler, D. (2006): Blogger reveals China's migratory goose farms near site of flu outbreak. *Nature* 441, 263.
- Cannell, J.J., Zaslhoff, M., Garland, C.F., Scragg, R., Giovannucci, E. (2008): On the epidemiology of influenza. *Virology Journal* 5:29, <http://www.virologyj.com/content5/1/29>
- Collins, S.D. (1957): Influenza in the United States, 1887 – 1956. Extract from "Review and study of illness and medical care with special reference to long-time trends." *Public Health Monographs* No. 48. http://www.history.navy.mil/library/online/influenza_collins.htm
- Chen, H., Smith, G.J.D., Li, K.S. et al. (2006): Establishment of multiple sublineages of H5N1 influenza virus in Asia: Implications for pandemic control. *PNAS* 103, 2845-2850, 2006.
- DEFRA /2008): Outbreak of highly pathogenic H5N1 avian influenza in Suffolk in January 2007. A report of the epidemiological findings by the national emergency epidemiological group. news.bbc.co.uk/1/shared/bsp/hi/pdfs/20_04_07_defra_bird.pdf
- GAIN (2005): Laos poultry and products avian influenza. <http://www.fas.usda.gov/gainfiles/200503/146119131>
- Gernhart, G. (1999): A forgotten enemy: PHS's fight against the 1918 influenza pandemic. *Public Health Reports* 114: 559-561. Online: www.history.navy.mil/library/online/influenza_forgot.htm
- GRAIN (2006 a): Fowl play. The poultry industry's central role in the bird flu crisis. www.grain.org/go/birdflu
- GRAIN (2006 b): The top-down global response to bird flu. <http://grain.org/articles/index.cfm?id=12>
- GRAIN (2007): Bird flu: a bonanza for 'Big Chicken'. <http://www.grain.org/articles/index.cfm?id=22>
- Guan, Y., Peiris, S.M., Lipatov, A.S. et al. (2002): Emergence of multiple genotypes of H5N1 avian influenza viruses in Hong Kong SAR. *PNAS* 99, 8950-8955.
- Kilpatrick, A.M., Chmura, A.A., Gibbons, D.W. et al. (2006): Predicting the global spread of H5N1 avian influenza. *PNAS* 103, 19368-19373.

- Li, K.S., Guan, Y., Wang, J. et al. (2004): Genesis of a highly pathogenic and potentially pandemic H5N1 influenza virus in eastern Asia. *Nature* 430, 209-213.
- Liu, J., Xiao, H., Lei, F. et al. (2005): Highly pathogenic H5N1 Influenza virus infection in migratory birds. *Science* 309, 1206.
- Lorenzen, S. (2008): Evolution und Ausbreitung des Vogelgrippe-Virus H5N1 Asia sowie Aspekte der Biosicherheit. *Tierärztliche Umschau* 63, 333-339.
- McBean, E. I. (1997): Swine Flu expose, chapter 2: The Spanish Influenza Epidemic of 1918 was caused by vaccinations. www.whale.to/vaccine/sf1.html
- Muzaffar, S.B., Ydenberg, R.C., Jones, I.L. (2006): Avian influenza: An ecological and evolutionary perspective for waterbird scientists. *Waterbirds* 29, 243-257.
- Newman, S., Pinto, J., DeSimone, L. et al. (2007): HPAI in Europe 2007: Concurrent outbreaks in Poultry and wild birds. www.fao.org/docs/eims/upload/231765/EW_Europe_aug07_ai
- Oxford, J. (2004): Nature's biological weapon. The 1918 flu pandemic killed 50 million people – and it could happen again. *Nature* 429: 345-346.
- Petermann, P. (2008): Das Rätsel der "Vogelgrippe": Geflügelpest oder Wildvogelseuche? *Anzeiger des Vereins Thüringer Ornithologen* 6, 117-141.
- Petermann, P. (2007): 285 tote Schwarzhalstaucher, ein Nachruf und ein dringender Aufruf. www.netzwerk-phoenix.net
- Schoene, C., Harder, T., Globis, A., Conraths, F.J., Mettenleiter, T.C. (2009): Die Wildvogel-Frage: Welche Rolle spielen Wildvögel im Infektionsgeschehen der hochpathogenen aviären Influenza? *Tierärztliche Umschau* 64, 77-83.
- Sharkey, K.J., Bowers, R.G., Morgan, K.L. et al. (2007): Epidemiological consequences of an incursion of highly pathogenic H5N1 avian influenza into British poultry flock. *Proc. R. Soc. B*: doi:10.1098/rspb.2007.1100.
- Starick, E., Beer, M., Hoffmann, B. et al. (2007): Phylogenetic analyses of highly pathogenic avian influenza virus isolates from Germany in 2006 and 2007 suggest at least three separate introductions of H5N1 virus. *VETMIC* 3852, doi: 10.1016/j.vetmic.2007.10.012.
- Suarez, D.L., Senne, D.A., Banks, J. et al. (2004): Recombination resulting in virulence shift in avian influenza outbreak, Chile. *Emerging Infectious Diseases* 10, 693-699.
- Subbarao, K., Klimov, A., Katz, J. et al. (1998): Characterization of an Avian Influenza A (H5N1) Virus isolated from a child with a fatal respiratory illness. *Science* 279, 393-396.
- Taubenberger, K.K., Morens, D.M. (2006): 1918 Influenza: the mother of all pandemics. *Emerging Infectious Diseases* 12, 15-22.
- Uiprasertkul, M., Puthavathana, P., Sangsiriwut, K. et al. (2005): Influenza A H5N1 replication sites in humans. *Emerging Infectious Diseases* 11, 1036-1041.
- WHO (2009): Cumulative number of confirmed human cases of avian influenza A/(H5N1) reported to WHO. http://www.who.int/csr/disease/avian_influenza/country/cases_tab
- WHO/OIE/FAO/ H5N1 (2008): Toward a unified nomenclature system for highly pathogenic avian influenza virus (H5N1). <http://www.cdc.gov/eid/content/14/7/e1-T2.htm>
- Williams, M. (2006): Farm fish fed dead chickens a risk for H5N1 influenza in Indonesia? www.drmartinwilliams.com/conservation/catfish-farm.html
- Xu, X., Subbarao, K., Cox, N.J., Guo, Y. (1999): Genetic characterization of the pathogenic influenza A/Goose/Guangdong/1/96 (H5N1) Virus: Similarity of its hemagglutinin gene to those of H5N1 viruses from the 1997 outbreaks in Hong Kong. *Virology* 261, 15-19.

Yasue, M., Feare, C.J., Bennun, W., R, Fiedler, W. (2006): The epidemiology of H5N1 Avian Influenza in wild birds: Why we need better ecological data. *BioScience* 56-11, 1-7.