Avian Killer Flu Pandemic: Fact, Fear, or Fiction

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Key words: avian flu, HN51, pandemic

Summary and Conclusions

The avian flu virus was first found in China in 1996 and killed six people in Hong Kong in 1997. The virus differs from the one that caused the 1918-1919 influenza pandemic, which killed millions of people, and other influenza epidemics of the 20th century. Avian flu has now affected humans in many countries in Asia, Europe and Africa and has killed over 100 people. For the interested reader, Hilleman [7] provides an extensive review of the pathogenesis, epidemiology and control of human viral influenza. The control of a possible avian flu pandemic will require further research in addition to national and international preparedness at various levels. Also, as Drs. Wong and Yuen [8] stress: “the epidemiology, neurology, clinical features, laboratory diagnosis, management and hospital control measures need to be examined from a clinical perspective.” The earlier influenza pandemics need to be compared to the human avian influenza [6]. Advising patients, targeting patients, and coping with the demand for services if an avian flu pandemic occurs must all be considered [6]. Little is written in the literature about the management and/or prevention of human avian flu.

The usual antiviral agents may not be effective in avian flu [9]. This H5N1 virus may become resistant to currently available antiviral agents. The exact risk or threat of an avian influenza pandemic is unknown [10,11]. The pharmaceutical industry is currently working on developing an effective vaccine or anti-H5N1 antiviral agent [12].

The title of this essay – “Avian killer flu pandemic: fact, fear or fiction” – is intentionally provocative to stimulate thought and dialogue. It is an accepted fact that human avian flu exists and has been reported in over 200 people, more than half of whom have died. It is not yet known whether a pandemic is likely or probable, but it is certainly possible – notwithstanding the response of some scientists who downplay an immediate or future threat. Perhaps the most exciting news is the recent demonstration by an Israeli team of the efficaciousness of elderberry extract against avian flu [5]. In the meantime, the world is in a race against time to develop vaccines against avian influenza [13]. This fact is important in view of a report of lethal influenza A (H5N1) infection in a pregnant woman in Ankui Province, China.

The Asian bird flu virus (HN51) was first detected in Asia in 2003. Since then it has caused the deaths of millions of chickens, turkeys and other birds and fowl. To date, this virus has caused about 100 human deaths in people who had direct contact with birds or their internal organs. Millions more chicken and turkeys are being slaughtered by the governments of various countries to try to limit the spread of this fatal disease. The virus has now been found in chickens and other birds in Asia, Europe and Africa. Fortunately, this virulent strain of virus has not mutated sufficiently to allow it to spread from human to human. Public health authorities around the world, including the World Health Organization and the United States Centers for Disease Control, are debating the likelihood of a pandemic of this killer flu virus although no one is certain that such a pandemic will occur. Is it possible? Yes. Is it likely? Debatable. This essay addresses the facts and discussions to date regarding the Asian killer flu virus, reviews the most recent literature on the subject, and discusses the implications of the findings, observations, debates and speculations.

Brief history

The avian flu virus surfaced in China in 1996 and killed six people in Hong Kong in 1997. In 2003, it began its deadly spread throughout Asia and human cases have since been confirmed in Europe, Africa and the Middle East [1]. The occurrence of human avian flu virus (H5N1) in Southeast Asia has paralleled large outbreaks of avian influenza A (H5N1) that killed millions of chickens, turkeys and other birds. The avian epidemics of 2004 and 2005 only rarely led to disease in humans [2]. People who are involved in mass culling of poultry do not become infected.

The expanding geographic distribution of avian flu (H5N1) infections, with recent outbreaks in Kazakhstan, Mongolia and Russia, indicates that human populations are indeed at risk [2]. At the time of writing, May 2006, over 200 human cases have been confirmed, half of which were fatal [1]. So far, avian flu has occurred only in individuals or communities with close links to poultry [3-5]. None has occurred where human-to-human transmission was implicated beyond doubt [6].
A recently described vaccine may be effective in preventing influenza A (H5N1) disease in humans [15,16]. Most scientists feel that an avian flu epidemic is inevitable. The question is not “if” but “when.” This essay reviews the subject and debates surrounding it.

References

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Capsule

Genes and chronic fatigue

The U.S. Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia, announced that chronic fatigue syndrome (CFS) has a biological and genetic basis. CDC Director Julie Gerberding called the study “groundbreaking” and also hailed its novel methodology. But, like most aspects of CFS, the study and its findings are controversial. CFS is defined as severe fatigue lasting more than 6 months, accompanied by symptoms such as muscle pain and memory problems. It is thought to affect at least 1 million Americans, mostly women. The lack of specific diagnostic criteria since CFS was first defined 20 years ago has led to debate over whether the cause could be an infectious agent, psychiatric, or something else – and made research funding for the disorder highly political. The new project, led by William Reeves, CDC’s top CFS researcher, took an unusual approach. Instead of recruiting patients already diagnosed with the syndrome, CDC surveyed one-quarter of the population of Wichita, Kansas, by phone, to find people suffering from severe fatigue. Several thousand then underwent screening at a clinic for CFS. The population-based aspect is a “big plus” because it avoids the possible bias in tapping a pool of patients seeking treatment for their problems. Out of this survey, 172 people, most of them white middle-aged women, fit the criteria for CFS (n=58) or CFS-like illness (n=114). A total of 227 people, including 59 controls, then underwent an extensive 2 day battery of clinical measurements, including sleep studies, cognitive tests, biochemical analyses, and gene-expression studies on blood cells. In another unusual step, the CDC then handed this massive data set to four teams of outside epidemiologists, mathematicians, physicists, and other experts. Some groups looked for associations between CFS and 43 common mutations in 11 genes involved in the hypothalamic-pituitary-adrenal axis, which controls the body’s response to stress. The results, which include the finding that the patterns of expression of about two dozen genes involved in immune function, cell signaling, and other roles are different in CFS patients, provide “solid evidence” for a biological basis of CFS. They dispel the notion that “this is a bunch of hysterical upper-class professional white women.” Other scientists are much more cautious. The most controversial assertion, however, is that the Wichita study has tied CFS to particular mutations in three genes, including the glucocorticoid receptor and one affecting serotonin levels. Genetic epidemiologists are skeptical for two reasons. First, the team looked for associations with just 43 gene variants; another set of genes might have correlated just as closely. Second, the researchers studied no more than 100 or so individuals with fatigue. The results, although meeting the threshold for statistical significance, are “very likely not robust.” CDC researchers are now planning to repeat the study with 100 CFS patients. They will also validate the gene-expression results and will hold another computational exercise at Duke University in Durham, North Carolina, with a larger data set.

Science 2006;312:669
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