

*Review Article***CONCENTRATED ANIMAL FEEDING OPERATIONS (CAFOs) AS POTENTIAL INCUBATORS FOR INFLUENZA OUTBREAKS****J. E. Hollenbeck\***

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**ABSTRACT**

The most significant change in the evolution of the influenza virus is the rapid growth of the Concentrated Animal Feeding Operations (CAFOs) on a global scale. These industrial agricultural operations have the potential of housing thousands of animals in a relatively small area. Evidence has discovered that the respiratory tracts of humans and pigs have “avian-like” receptors that may have contributed to the 1918 influenza pandemic and recent 2009-2010 H1N1 outbreak. Evidence is growing for interspecies transmission of the flu among humans, swine, and birds assisted by genetic assortment of the virus while hosted by swine. With the development of CAFOs throughout the world, the need for training of animal caretakers to observe, identify, treat, vaccinate and cull if necessary is important to safeguard public health. The best defense against another pandemic is constant monitoring of the livestock and handlers of CAFOs and the live animal markets of Southeast Asia. These are the most likely epicenters of the next pandemic.

**Key words:** Influenza, avian influenza, swine influenza, airborne pathogens, agricultural health and disease prevention, rural health, pandemics, environmental health

**The Rise of Industrial Agriculture and its Impact on Health**

The most significant change in the evolution of the influenza virus is the rapid growth of Concentrated Animal Feeding Operations (CAFOs). These industrial agricultural operations have the potential of housing hundreds of thousands of animals in relatively small areas

near human population centers. According to the United States Department of Agriculture there is little fluctuations of the swine population from 1915 to 2000, yet the number of farm producers have significantly declined since 1970, and the number of commercial pork producer operations have increased from 14,500 to over 22,000 by 2010 as reported by the shown by **Table 1**.

**Table 1.** Number of Commercial and Farm-base Producers in the United States from 1970-2012

<http://www.ers.usda.gov/data-products/livestock-meat-domestic-data.aspx#26112>, Retrieved Nov. 17, 2012.

	1970	1975	1980	1985	1990	1995	2000	2005	2010
Farms	199	194	184	79	54	38	24	20	19
Commercial	14,500	11,585	16,433	14,728	15,300	17,811	18,928	20,511	22,437
Total	14,699	11,779	16,617	14,807	15,334	17,849	18,952	20,531	22,456

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The concern about CAFOs is the harboring and providing a rich environment for the evolution of new strains of influenza. CAFOs hold large populations of animals, they can facilitate the rapid spread of a pathogenic agent to population

centers as was in the case in 2009-2010 Mexican H1N1 outbreak. Training in the surveillance, and vaccination against disease, especially airborne pathogens should be a priority (1). These facilities provide an unnaturally high concentration of animals with limited air space, and waste removal which allows for the rapid selection of, amplification, and with the rapid transportation of animals from one site to another, and results in never before spread of zoonotic pathogens on such a large scale (1, 2, 3). These are unique circumstances to animal husbandry.

### **Past Experience with Influenza**

Human-animal cohabitation started 5,000 to 10,000 years ago when humans began to capture and breed livestock (cattle, sheep, swine, chickens, ducks and other beasts of burden) (2, 4). This enabled the transmission of enzootic pathogens to cross species and find alternative reservoirs. All major domesticated animals (birds, bovines, camels, sheep and swine were found and adapted in Eurasia, which is the usually the epicenter for majority of communicable epidemics and pandemics (2, 5). Not until the last three-four hundred years has the symptoms of influenza has been collected and analyzed with a degree of accuracy. The documentation is anecdotal until the past century with an Iowan veterinarian and inspector for the U.S. Bureau of Animal Industry in Fort Dodge, Iowa, J.S. Koen, observed in pigs a disease that resembled the raging human influenza of 1918-1919:

"Last fall and winter we were confronted with a new condition, if not a new disease. I believe that has as much to support this diagnosis in pigs, as the physicians have to support a similar diagnosis in man. The similarity of the epidemic among people and the epidemic in pigs was so close, the reports so frequent that an outbreak in the family would be followed immediately by an outbreak among the hogs, and vice versa, as to present a most striking coincidence if not suggesting a close relationship between the two conditions. It looked like the "flu" and until proven it was the "flu," I shall stand by that diagnosis" (6, 7).

Koen's observations were unpopular, especially among farmers raising pigs. Ten years later researchers with the U. S. Bureau of Animal

Industry reported the successful transmission of influenza from pig to pig by taking mucus from the secretions from the upper respiratory tract of infected pigs to healthy pigs. Richard Shope, working with the Rockefeller Institute of Comparative Anatomy repeated the study and was able to reproduce the disease in healthy pigs with material taken from sick pigs and passed through a Pasteur-Chamberlain filter. Shope provided the first evidence of virus transmitted by swine (6, 7). In 1923 Richard Shope showed that people who were alive during the 1918-1919 epidemic had antibodies against the "pig" virus, but those born after 1920 lacked such antibodies (6, 7, 8). Shope's conclusion, which would be the dominant hypothesis, was that the source of the pandemic was a virus that crossed from one species to another to eventually infect humans.

Supporting evidence of Shope's hypothesis of trans-species infection occurred in a related study in 1928, of canine distemper, with ferrets being used as the study animals, at the United Kingdom's Medical Research Council's laboratory: unexpectedly, the ferrets became ill with symptoms of human influenza. When one of the researchers became ill with the flu, washings were obtained from the researcher's throat and injected the filtrate into healthy ferrets, they became ill with the same symptoms (7, 8, 9). This provided the first evidence that a virus caused human influenza, based on the conclusion fulfilling Koch's postulates. This became the most widely accepted model on influenza.

### **New research on influenza and its conversion**

Human influenza viruses do not replicate efficiently in ducks and other waterfowl and neither does duck influenza viruses in humans (3, 8, 10). Influenza viruses found in their natural hosts do not seem to pose a threat, however, once disturbed or have migrated to another host there are two means in which they can enter the human species: assortment or adaptation in an intermediated host (3). The increase of CAFOs incorporating rice-duck farming (4, 11), pig-waterfowl-fish aquaculture (3, 12); and live poultry markets (4, 13, 14) are all implicated in the emergence of an influenza virus with pandemic potential. With the expansion of the intensive poultry CAFOs to 10-million-bird-mega-farms could account for the dramatic genetic shift resulting from the change in the

ecology and epidemiology of avian influenza. In China alone, there are now over 10 billion potential reservoirs the influenza virus to exist and change. This translates into the greater risk of genetic drift and a greater degree of reassortment by just simple probability to a novel strain. This model alone can offer with evidence that Galliform species (i.e., terrestrial birds such as chickens, turkeys and quail) may serve as a suitable intermediate hosts to influenza to convert to more virulent strains.

The intermediate host must have a receptor specificity that will allow for the virus to attach to the host cell membrane. The influenza virus must attach to sialic (N-acetylneuramic) acid in an alpha-2, 3 or alpha-2, 6 attachments. The intestinal epithelium of a duck has the alpha- 2, 3 linkages, and the human respiratory tract contains the alpha-2, 6 linkages. This necessitates an intermediate host. In the past it was assumed that swine played this role, but in 2003 an outbreak in the Netherlands suggested that it was a direct avian-human transmission, without 7

recombination and adaptation, resulting in human-human transmission (1). The human H5N1 cases that appeared in 1997 suggest that the avian influenza can directly be a virulent zoonotic pathogen. The 1997 Hong Kong outbreak of H5N1 influenza brings another intermediate host scenario to be considered. The Hong Kong victims were infected by chickens, not ducks as the usual route (1, 15, 16). Research as indicated that Gallinaceous poultry can act as intermediate host by:

- Reassortment,
- Direct Adaptation,
- Exaptive Adaptation,
- Rerouting of transmission.

Chickens may be heterozygous hosts for humans if the virus is able to adapt to the respiratory tract of galliforms and swine rather than the enteric tract of waterfowl. With the number of swine and poultry CAFOs, the cross transmission of the influenza virus becomes more likely to occur.

**Table 2. Timeline of Recent Influenza Pandemics Since 1918**

<http://www.flu.gov/individualfamily/about/pandemic/history.html#timelines>, Retrieved October 10, 2012

Year of Outbreak	Geographic Origin	Host Origin
1918, The <i>Spanish</i> Flu	Kansas, USA	Swine
1957-1958	China	avian
1968-1969, The Hong Kong Flu	China	Swine/avian
1976: Swine Flu Threat	Ft. Dix New Jersey, USA	Swine
1977: Russian Flu Threat	Northern China/Siberia	Chickens to Human
1997: Bird Flu Threat	Southern China	Avian
2009-2010 H1N1	California/Mexico	Swine

### Regulation of CAFOs and Protection of Human Health

The challenge of the future is recognizing the potential which the health effects of CAFOs may have on human health centers. The 1918 influenza pandemic has been traced back to a single soldier near present day Ft. Riley, Kansas, cleaning the pig pens one spring day. Since that day time we have recorded a number of pandemics (**Table 2**) linked to swine and birds. With the expansion of CAFOs as shown on table 1, the threat continues. The water run-off and odors have been exhaustively studied, yet the

potential viral load in airborne particulates has been ignored. The massive populations of animals should force researchers to move beyond odor based studies to that of one encompassing research of composition and potential health risks to humans (4). Recent research and shared results indicate that neighbors of CAFOs do experience health problems at a significantly higher rate than the control populations (17, 18). Dust from feedlots and animal housing units contain biologically active organisms such as bacteria, mold, and fungi from feces, and feed; this dust poses a

greater health hazard than does general “nuisance” dust (17). In a recent study of swine confinements in Texas, 20% of workers suffered from Organic Dust Toxic Syndrome (ODTS). ODTS is an acute influenza-like illness that follows four-six hours of intense exposure to agricultural dusts (18). The emissions from CAFOs alone create problems as aerosols and act as vectors for airborne viruses. With so many swine and poultry CAFOs in close proximity, the acceleration of the “mixing” and assortment of influenza viruses is unfathomable (19, 20).

The evidence is growing for interspecies spread of human, swine and avian viruses. With the number of CAFOs developing throughout the world, the need for training of animal caretakers to observe, identify, treat, vaccinate and cull if necessary is vital. These workers may be the next patient zero in the next pandemic. Our best defense against the next pandemic is to monitor the CAFOs and their workers.

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