

Bird flu linked to free range farming

JOHN ROSS THE AUSTRALIAN JUNE 24, 2014 6:00AM

FREE-RANGE farming has increased the risk of a bird flu pandemic, British scientists have suggested.

Oxford University researchers say gradual changes in farming practices may have allowed domestic ducks to become the “Trojan Horse” that sparked the deadly H5N1 flu strain, causing almost 400 confirmed human deaths so far.

The researchers say birds’ natural immunity has helped prevent deadly bird flu strains from spreading to humans. But immunity patterns are being destabilised by a global trend towards farming ducks in open barns or pastures.

“The main threat ... may arise from a potential disturbance to the established immunodynamics of influenza rather than from the potential evolution of new virulent strains,” the team reports today in the journal PNAS (<http://www.pnas.org/cgi/doi/10.1073/pnas.1401849111>).

“Changes in farming practices bringing domestic ducks into more prolonged contact with wildfowl (may) have contributed to the increase in highly pathogenic avian influenza outbreaks in the last 20 years.”

Dangerous bird flus are thought to originate in wild bird flocks. But the new study found domestic ducks regularly generate more dangerous strains because they have shorter lifespans and higher population turnover.

These dangerous strains usually do not take hold because milder strains in wild birds prompt similar immune responses, giving wildfowl advance immunity and preventing the killer subtypes from spreading widely and undergoing more mutations.

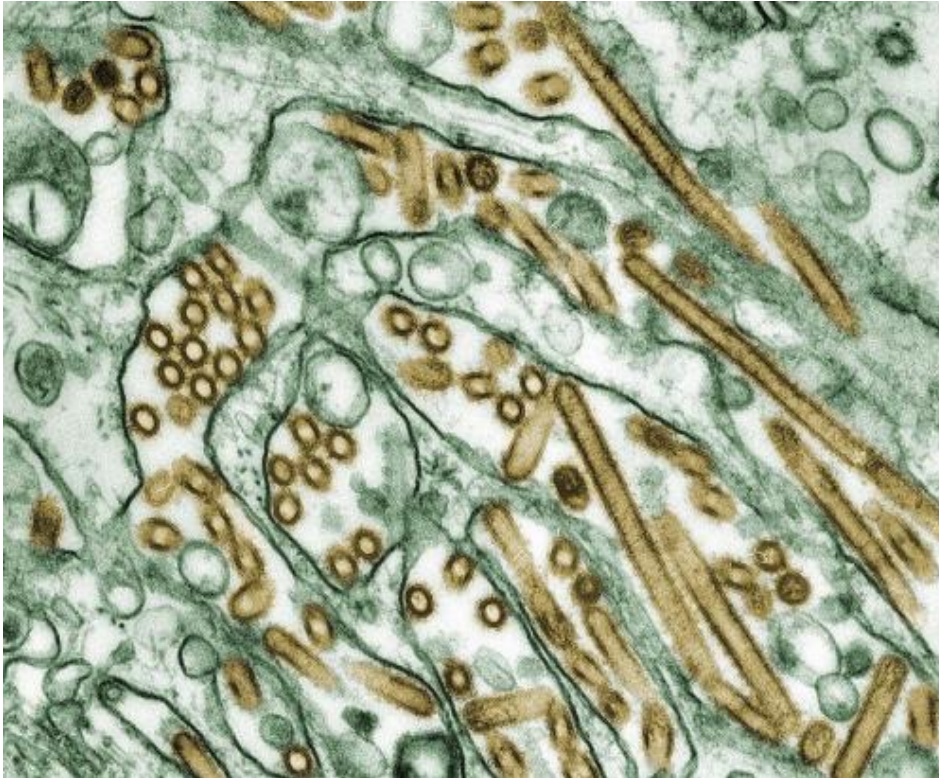
But this balance can be jeopardised when domestic ducks are raised in spaces frequented by their wild cousins. “Contact between species of different lifespans can ... provoke the emergence of a previously suppressed strain,” the paper warns.

H5N1, which emerged in its current form in the early 2000s, is considered the world’s largest pandemic threat. Scientists believe a few mutations could make it directly transmissible between humans, creating the risk of a pandemic like the catastrophic Spanish flu.

The new study may help explain why H5N1 kills wild and domestic birds alike — unlike most dangerous avian flus, which cause only mild symptoms in wild birds.

It may also explain why Thai agricultural officials tackling a 2004 H5N1 outbreak found the virus was widespread in “open house” and free-range ducks, but non-existent in “closed” barns isolated from wild flocks.

Researchers find putting birds of different life-spans together can promote emergence of dangerous influenza strains



Colorized transmission electron micrograph of Avian influenza A H5N1 viruses. Credit: Public Domain

(Phys.org) —A trio of researchers in the U.K. has found that housing birds of different life-spans together can contribute to the emergence of dangerous flu strains. In their paper published in *Proceedings of the National Academy of Sciences*, Paul Wikramaratna, Oliver Pybus and Sunetra Gupta of Oxford University describe how they developed a model that allows for studying the evolution of flu viruses in domestic and wild bird species with various life-spans and what they found when running it.

Most research involving the spread of avian flu, the researchers note, is focused on the ability of such viruses to mutate and spread to other possible hosts. In their study, the trio took the opposite approach, looking at the immunity capabilities of birds of different species to see how they respond to such viruses. To gain a better understanding of flu transmission from the perspective of the birds, the team built a model (combining three properties—pathogenicity, antigenicity, and transmissibility) that was able to use data from prior studies on flu prevalence with birds in Europe, focusing most specifically on the competition that exists between strains that are considered more or less pathogenic.

The result was a model that could be used to show the degree to which different species of birds were able to ward off different flu pathogens. The researchers found that strains considered to be highly pathogenic were more likely to emerge in bird species that didn't live very long compared to long-lived varieties. This they attributed to higher turnover in the population. The model was also able to show that contact between

short-lived species and long-lived species could promote the emergence of highly pathogenic flu variants. In real-world terms, the researchers suggest, this means that housing short-lived domestic birds, such as ducks near to areas where long-lived wild birds lived could promote the emergence of dangerous flue variants, which would allow for the spread of highly pathogenic avian influenza variants by the wild birds. They suggest that changes in farming practices (where birds are now kept in open enclosures rather than closed) over the past 20 years have likely contributed to the increase in virulent outbreaks that have been observed in recent years.

More information: Contact between bird species of different lifespans can promote the emergence of highly pathogenic avian influenza strains, Paul S. Wikramaratna, *PNAS*, [DOI: 10.1073/pnas.1401849111](https://doi.org/10.1073/pnas.1401849111)

Abstract

Outbreaks of highly pathogenic strains of avian influenza viruses (AIVs) cause considerable economic losses to the poultry industry and also pose a threat to human life. The possibility that one of these strains will evolve to become transmissible between humans, sparking a major influenza pandemic, is a matter of great concern. Most studies so far have focused on assessing these odds from the perspective of the intrinsic mutability of AIV rather than the ecological constraints to invasion faced by the virus population. Here we present an alternative multihost model for the evolution of AIV in which the mode and tempo of mutation play a limited role, with the emergence of strains being determined instead principally by the prevailing profile of population-level immunity. We show that (i) many of the observed differences in influenza virus dynamics among species can be captured by our model by simply varying host lifespan and (ii) increased contact between species of different lifespans can promote the emergence of potentially more virulent strains that were hitherto suppressed in one of the species.

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