Infectious diseases

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Scientists condemn 'crazy, dangerous' creation of deadly airborne flu virus

Researchers say recreation of Spanish flu strain highlights risk of pandemic, but critics say work puts global population at risk

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Scientists at the University of Wisconsin-Madison used a technique called reverse genetics to build the virus from fragments of wild bird flu strains. They then mutated the virus to make it airborne to spread more easily from one animal to another.

"The work they are doing is absolutely crazy. The whole thing is exceedingly dangerous," said Lord May, the former president of the Royal Society and one time chief science adviser to the UK government. "Yes, there is a danger, but it's not arising form the viruses out there in the animals, it's arising from the labs of grossly ambitious people."

Influenza viruses circulate freely in wild bird populations. Most remain in chickens, ducks and other birds, but occasionally strains mutate into a form that can infect humans. The H5N1 bird flu strain has killed at least 386 people since 2003, according to WHO figures. The Spanish 1918 flu is thought to have come from birds too.

Writing in the journal Cell Host and Microbe Yoshihiro Kawaoka describes how his team analysed various bird flu viruses and found genes from several strains that were very similar to those that made up the 1918 human flu virus. They combined the bird flu genes into a single new virus, making a new pathogen that was only about 3% different from the 1918 human virus.

The freshly made virus - the first of several the team created - was more harmful to mice and ferrets than normal bird flu viruses, but not as dangerous as the 1918 strain. It did not spread between ferrets and none of the animals died. But the scientists went on to mutate the virus, to see what changes could make it spread. Seven mutations later, they had a more dangerous version that spread easily from animal to animal in tiny water droplets, the same way flu spreads in humans.

Kawaoka, who led the research in a high-security lab at the University of Wisconsin-Madison, said the work highlighted how flu viruses found in wild bird populations had the potential to adapt to humans and cause a pandemic.

Follow-up experiments showed that the 2009 swine flu vaccine and the anti-viral drug tamiflu should be effective against the virus. "This is important information for those making decisions about surveillance and pandemic preparedness," Kawaoka told the Guardian.

This is a risky activity, even in the safest labs. Scientists should not take such risks without strong evidence that the work could save lives, which this paper does not provide," he added.

In an article published last month, Lipsitch argued that experiments like Kawaoka's could unleash a catastrophic pandemic if a virus escaped or was intentionally released from a high-security laboratory.

But Kawaoka defended the work, saying that critics failed to appreciate the impact of his and others' work on dangerous viruses. "There were discussions on the usefulness of stockpiling H5N1 [bird flu] vaccines until our H5N1 papers were published. Similarly, this paper strongly supports stockpiling anti-influenza drugs. If this is not a 'lifesaving benefit', what is?" he said.

Many of the groups that create dangerous viruses to understand their workings are funded by the US National Institutes of Health (NIH). Lord May said he suspected the NIH supported the work because officials there were "incompetent" and believed the justifications that scientists told them. "This is work that shouldn't be done. It's as simple as that," he said.

The experiments show that a 1918-like flu virus could emerge in the wild as bird viruses swap genes and mutate. "Influenza viruses readily swap genes to generate new viruses, so something like this could happen, especially since many of these viruses have circulated in recent years," Kawaoka said. The viruses "have the potential to become adapted to mammals and possibly cause a human pandemic," he added.

The study identifies particular mutations that made the virus spread so easily. But that is not much use for surveillance, said Lipsitch, because there are scores of other mutations that could have the same effect. "The chance that a virus very similar to the one they study will appear in nature is extremely remote," he said. Kawaoka argues that his team is fully aware of this, and that the underlying mechanisms that make the virus so dangerous are more important for preventing future pandemics.

Simon Wain-Hobson, a virologist at the Pasteur Institute in Paris, said he feared that governments and funding bodies would only take the risks seriously once an accident had happened. "It's madness, folly. It shows profound lack of respect for the collective decision-making process we've always shown in fighting infections. If society, the intelligent layperson, understood what was going on, they would say 'What the F are you doing?'"

Carole Heilman, director of microbiology and infectious diseases at the National Institute of Allergy and Infectious Diseases (Niaid) in the US, said: "This study was conducted as part of a research project on understanding the molecular mechanisms of virulence of the 1918 influenza virus. NIH peer review determined that the research was scientifically meritorious. It was also determined that the information gained had the potential to help public health agencies in their assessment of circulating and newly emerging strains. In addition, NIH determined that all the research was being done under appropriate biosafety conditions and with appropriate risk mitigation measures."