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Swine flu variant may have sabotaged lungs' clearing mechanism

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A variant of last year's pandemic influenza that was linked to fatal cases carried a mutation that enabled it to infect different cells that line the airway, according to a study from scientists at the Medical Research Council National Institute for Medical Research (NIMR), Imperial College London and the University of Marburg. The findings suggest that the mutant virus could have impaired the lungs' ability to clear out germs and highlight the potential for deadlier strains of flu to emerge and spread.

The 2009 pandemic of H1N1 influenza caused thousands of deaths worldwide, but the majority of cases were relatively mild. A variant of the virus carried a mutation termed D222G in a protein called haemagglutinin on the surface of the virus, and people infected with this variant were more likely to have severe and fatal illness. According to a World Health Organisation report, the D222G mutation was found in less than two in every hundred cases of the 2009 pandemic flu, but was responsible for around seven in every hundred deaths.

This new research shows that flu virus with the D222G mutation has an increased capacity to infect ciliated cells in the airways. Ciliated cells have hair-like projections that help move mucus with trapped germ particles upward toward the mouth, which is then normally swallowed or coughed up. When the cilated cells become infected, the cilia stop moving and this vital clearance function is impaired. Inhaled viruses and bacteria can then reach the lung more easily, where they can potentially cause pneumonia. The researchers believe this sabotage of the lungs' clearing mechanism could be one factor that made the D222G mutation more virulent.

Professor Ten Feizi, who is funded by the Medical Research Council at Imperial College London and led the study, said:

"This simple mutation, which swapped one building block of a virus protein for another, apparently resulted in a more virulent version of the H1N1 virus. We think this is at least partly due to the virus being able to bind to different receptors, which allowed it to infect ciliated cells and stop them from clearing out germs."

"If the mutant virus were to acquire the ability to spread more widely, the consequences could be very serious. The study goes to show how important it is that the WHO Global Influenza Surveillance Network continues to monitor closely the emergence of new variants of the flu virus. Even though the 2009 pandemic was relatively mild, it's vital that we handle outbreaks cautiously and stay vigilant."

The scientists studied different flu viruses by using carbohydrates that mimic the cell receptors on the surface of airway lining and attaching these to a glass surface. The virus was then incubated on top of the

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glass surface, and using a fluorescent dye, the researchers could see which receptors the virus has bonded with.

The MRC's National Institute for Medical Research (NIMR) houses the World Influenza Centre, one of five World Health Organization (WHO) Collaborating Centres for Reference and Research on Influenza – bringing together the brightest minds together to understand the underlying mechanics of the flu virus and develop vaccine strategy to cover existing and emerging threats.

The study was funded by the Wellcome Trust, the Medical Research Council, Biotechnology and Biological Sciences Research Council, the UK Research Councils' Basic Technology Initiative, Engineering and Physical Sciences Research Council and grants from the Von Behring-Roentgen Foundation and LOEWE Program UGMLC of the State of Hesse.

The paper, 'Altered receptor specificity and cell tropism of D222G haemagglutinin mutants from fatal cases of pandemic A(H1N1) 2009 influenza', is currently available in the *Journal of Virology* online and will be published in the November 2010 issue (Volume 84, Issue 22).

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