Interleukin-22 protects against post-influenza bacterial superinfection

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Researchers from the Pasteur Institute, Lille, France have shown in a mouse model that interleukin-22 protects against bacterial superinfections that can arise following influenza. Their research is published in the June 2013 issue of the *Journal of Virology*.

Influenza A viral infection can lead to primary pneumonia and, later on, to serious complications including secondary bacterial pneumonia and sepsis. Post-influenza bacterial superinfections that occur during seasonal epidemics and pandemics are of great concern to human health and impose a considerable socio-economic burden.

"It is therefore critical that we reach a better understanding of the causes of and potential treatments for post-influenza bacterial superinfection," says corresponding author François Trottein.

"Mouse studies have revealed that impairment of the host innate immune defense, as well as lung damage caused by the virus are cardinal features of bacterial superinfection," says Trottein. The authors tested the hypothesis that interleukin-22, an important cytokine implicated in mucosal immunity, inflammation and tissue repair, might play an important role during influenza.

The authors show that several cell types belonging to the innate immune system produce interleukin-22 soon after infection. They also demonstrate that the lack of interleukin-22 aggravates the pathogenesis that develops in the lungs and in particular exacerbates epithelial damage

caused by the virus. Furthermore, endogenous interleukin-22 displays a protective role during secondary bacterial (pneumococcus) infection in the mouse system.

Although the mechanisms sustaining the protective effect of interleukin-22 are not yet fully elucidated, the authors speculate that its beneficial effect is due to its role in the maintenance of epithelial integrity.

"If it works as well in humans, the production of interleukin-22 could confer a substantial benefit on patients having flu," says Trottein.

More information: S. Ivanov, J. Renneson, F. Trottein, et al. Interleukin-22 reduces lung inflammation during influenza A virus infection and protects against secondary bacterial infection. J. Virol. June 2013; 87:12 6911-6924. Published ahead of print17 April 2013. doi:10.1128/JVI.02943-12

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