More jabs needed

Study suggests that vaccinating many more people could slow the seasonal influenza virus's ability to evade vaccines.

Anne Trafton, MIT News Office

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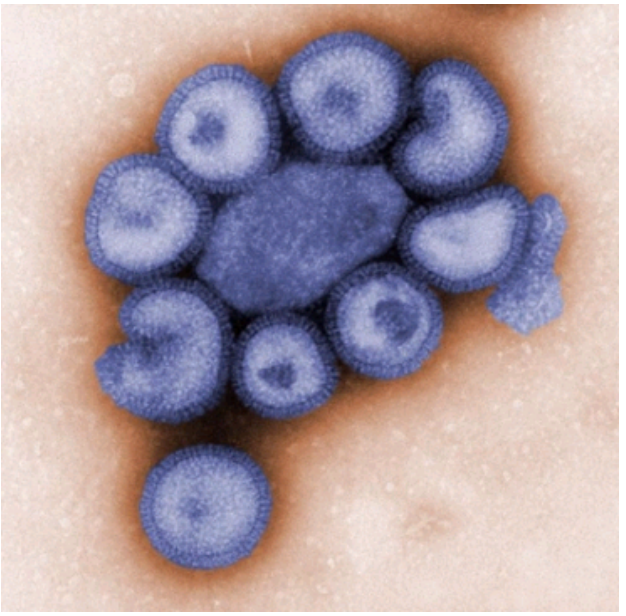
The seasonal influenza virus is extremely adaptable: each year, it evolves ways to evade vaccines, forcing vaccine makers to come up with new formulations.

That evolution, known as antigenic drift, occurs when viruses change the sequence of their major surface protein, known as hemagglutinin (HA). HA is one of the primary targets of the natural immune response and key to eliciting an effective vaccine response.



This colorized negative-stained transmission electron micrograph (TEM) depicts the ultrastructural details of a number of influenza virus particles, or "virions."
Image: CDC/Dr. F. A. Murphy

A paper published in today's issue of Science suggests how the spread of influenza between vaccinated and non-vaccinated (naïve) individuals may affect antigenic drift. The results suggest that maximizing the number of vaccinated people could slow antigenic drift, according to the research team.

"Our model predicts that decreasing the immunologically naïve population — by increasing the number of children vaccinated against influenza, for example — could slow the rate of antigenic drift and extend the duration of effectiveness of seasonal influenza vaccines," says Jonathan Yewdell, a virologist at the National Institute of Allergy and Infectious Disease (NIAID) and author of the Science paper.

The team, led by the NIAID, includes several researchers from MIT, including Ram Sasisekharan, director of the Harvard-MIT Division of Health Sciences and Technology.

Using a strain of flu virus isolated several decades ago, the researchers found that in vaccinated mice, viruses evolve to evade antibodies. They do this by mutating to improve their ability to stick to glycan receptors located on cell surfaces, shielding the viruses from antibody attack, according to Yewdell. However, there is a tradeoff: Those viruses become so sticky that they often end up binding to cells in the throat and nose, preventing them from infecting the lungs. These mutated flu viruses are relatively harmless.

When the researchers introduced these mutated viruses into unvaccinated mice, the viruses reverted to a low-affinity form that easily spreads and infects the lung cells. These viruses also mutated in the sections of the HA proteins targeted by antibodies.

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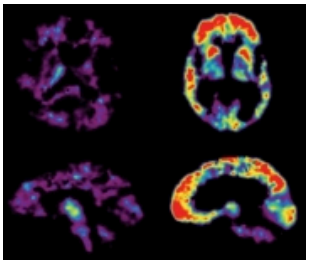

PET Scans Showing PiB Uptake in the brain of a cognitively healthy person (left) and in the brain of a person with AD (right).
Photo - Images courtesy of the Alzheimer's Disease Education and Referral Center

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The findings suggest that flu viruses may alternate between strains with high and low affinity for glycan receptors as they spread between people who are vaccinated and those that are not vaccinated. Those repeated cycles could give the viruses more chances to mutate their HA proteins and accelerate antigenic drift.

“This knowledge could help public health officials identify potential changes that could occur over the course of an epidemic,” said Jeremy M. Berg, director of the National Institutes of Health’s National Institute of General Medical Sciences, which partially funded the work.

The study demonstrates the importance of looking at the structure of the entire HA protein instead of focusing on mutations in the section that antibodies generally attack, says Andrew Pekosz, an immunologist at the Johns Hopkins Bloomberg School of Public Health, who was not involved in the research. “This forces us to take a step back and take a global perspective,” he says.

The work could also help vaccine researchers formulate new vaccines by potentially targeting flu viruses' glycan-binding sites.

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