Cyber exploring the 'ecosystems' of influenzas

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Predicting the infection patterns of influenzas requires tracking both the ecology and the evolution of the fast-morphing viruses that cause them, said a Duke University researcher who enlists computers to model such changes.

A single mutation can put a flu virus on a new-enough path to re-infect people who had developed immunity to its previous form, said Katia Koelle, a Duke assistant professor of biology.

For example, a commonplace Influenza A virus known as H3N2 emerged in 1968. But since then fully one-third of the component amino acids in its hemagglutinin protein -- the "H" in H3N2 -- have changed.

"That's a huge amount of evolution," Koelle said. "If there's a new escape mutant that can actually so change the protein's configuration that our antibodies can't recognize the virus anymore, that means it's going to have a huge advantage and infect more of us.

"How much of an advantage the new virus strain has will depend on how many people have gotten infected in the past. So the epidemiological dynamics will shape the evolutionary dynamics. And vice versa, the evolutionary dynamics will shape the epidemiological dynamics because mutations of the virus will allow people to become re-infected."

Koelle's group at Duke has developed a two-tiered model to simulate that interplay in such viruses, allowing scientists to "quantitatively

reproduce the patterns we observe," she said.

Koelle is scheduled to describe her work today during a symposium at the 2009 Ecological Society of America annual meeting in Albuquerque.

"We're interested in having a flexible and simple model that would not only be able to reproduce the dynamics of H3N2 but also help us understand how flu evolves differently in different hosts," Koelle added. For example, H3N2 (not to be confused with the H1N1 "swine flu" virus) also has been circulating in pigs, with the virus showing distinctly different evolutionary patterns in these hosts.

One of her group's models is focusing on that difference, which she suspects
is linked to man's and animals widely disparate lifespans -- about 80 years for humans versus under 2 for farm-raised hogs.

"The virus doesn't have to evolve rapidly to avoid being wiped out by the pigs' immunity to it," she said. "That's because there are always many more susceptible new hosts coming into the pig population."

Another challenge is Influenza B, a comparatively mild virus that infects mostly children but is complicated by the fact that two genetically distinct strain lineages circulate in human populations. During any given flu season, only one B sequence predominates, presenting a challenge for vaccine makers who must choose between them.

"They have to make an educated guess about which influenza B lineage is going to be the main one that season," Koelle said. "Sometimes there is a big B outbreak when it turns out to be the one not included in the vaccine."

More information:

http://eco.confex.com/eco/2009/techprogram/S4132.HTM

Source: Duke University (news : web)