

Why is the avian flu virus so lethal?

Two mutations in the H1N1 avian flu virus killed at least 50 million people during a 1918 pandemic, according to a team of MIT researchers led by Indian American Ram Sasisekharan.

The new study could aid scientists in monitoring mutations in the H5N1 avian flu strains currently circulating in Asia, says Sasisekharan in a paper in the forthcoming issue of the Proceedings of the National Academy of Sciences (PNAS)

These mutations could enable the virus to jump from birds to humans, as many epidemiologists fear.

The researchers used the 1918 virus as a model to probe the biochemical basis for hemagglutinin (HA) binding to glycans, facilitating viral transmission.

The team explained how the 1918 strain developed these mutations in HA, a surface molecule, allowing it to bind to receptors in the upper respiratory tract.

Two mutations dramatically change the HA binding affinity to such receptors, said Sasisekharan, a graduate of [Bangalore](#) University who is currently the Underwood Prescott Professor of Biological Engineering and Health Sciences and Technology at Massachusetts Institute of Technology (MIT).

In January, Sasisekharan and colleagues had reported that flu viruses can only bind to human respiratory cells if they match the shape of sugar (or glycan) receptors found on those cells.

The glycan receptors in the human respiratory tract are known as alpha 2-6 receptors, shaped like an open umbrella and a cone.

The MIT team discovered that to infect humans, avian flu viruses must gain the ability to bind tightly to the umbrella-shaped glycan receptors.

'The affinity between influenza virus HA and glycan receptors appears to be a critical determinant for viral transmission,' said Sasisekharan.

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