

Study explains why coronavirus did not spread as fast in East Asian countries

New Delhi, Feb 09 (India Science Wire): Novel coronavirus took many lives globally but the number of deaths recorded was more in Europe and in the United States than in Asia countries particularly east Asia. A team of scientists from the National Institute of Biomedical Genomics (NIBMG) in Kalyani, West Bengal led by Nidhan Biswas and Partha Majumder, has pointed out the biological mechanism that led to the novel coronavirus (SARS-CoV-2) variant with the D614G mutation, to spread significantly faster in Europe and North America but not in East Asia.

The team has explained how higher levels of a human protein- neutrophil elastase- helps the virus to enter the human cell, multiply and also spread faster from infected individuals. According to the paper, the variant with the D614G mutation introduced an additional cleavage site on the ACE-2 receptor for the SARS-CoV-2 virus to gain entry into cells.

The mere presence of an additional cleavage site alone does not automatically lead to better efficiency in entering the cells. For the virus to enter the cells more efficiently, the additional cleavage site has to be opened. "The cleavage site is opened by a human protein called the neutrophil elastase. This protein is available in plenty in the lungs," says Dr. Partha Majumdar. When the level of neutrophil elastase is high in an individual, the additional entry point created by the mutation opens up in a larger number of cells thus allowing more cells to be infected. Thus, the variant with the D614G mutation was able to spread better from one infected individual to another.

But an excessive amount of the protein neutrophil elastase can damage the lung tissue. Hence, the amount of neutrophil elastase produced is naturally kept under check by a protein called AAT (alpha1-antitrypsin), which means the AAT protein inhibits neutrophil elastase production.

However, some naturally-occurring mutations in the AAT-producing gene result in a deficiency of the AAT protein. The AAT protein deficiency thus results in a higher level of neutrophil elastase and hence enhanced ability of the virus to infect human cells and spread among people.

The researchers said that in order to reach 50% relative frequency the 614G subtype took a significantly longer time in East Asia (5.5 months) compared to Europe (2.15 months) as well as North America (2.83 months). Many were speculating why coronavirus spreads differentially across geographies. "The most popular speculation was the higher temperature in Asia was not congenial to the spread of the coronavirus. We believe the cause had to be biological, rather than physical or social," said Majumder.

According to the paper, AAT deficiency is the least in East Asian countries – 8 per 1,000 individuals in Malaysia, 5.4 per 1,000 in South Korea, 2.5 in Singapore. On the other hand, 67.3 in per 1,000 individuals in Spain are AAT deficient, 34.6 in the UK and 51.9 in France and in the US it is prevalent in 29 individuals among 1,000. The study has been published in the journal *Infection, Genetics and Evolution*. (India Science Wire)

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