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## Hypothesis on mystery of dengue virus infection confirmed

From ANI

Washington, Feb 12: La Jolla Institute scientists have proved a hypothesis that said antibodies contribute to severe dengue virus-induced disease.

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The findings of the La Jolla Institute for Allergy and Immunology have major implications for efforts to develop a first-ever vaccine against the dangerous infectious dengue disorder.

La Jolla Institute scientist Sujan Shresta said: "Our lab has proven the decades old hypothesis that subneutralizing levels of dengue virus antibodies exacerbate the disease.

"This is a situation where antibodies can be bad for you, which is counter to everything we know about the normal function of antibodies. It also presents a special challenge for researchers working to develop a dengue virus vaccine, since most vaccines work by prompting the body to produce antibodies."

Dengue infection is spread by mosquitoes and is caused by any of four closely related virus serotypes of the genus Flavivirus. Infection can cause diseases ranging from dengue fever, a flu-like illness, to the severest form -- dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS), which can cause the blood vessels to leak, leading to life-threatening shock.

The dengue virus antibody phenomenon, termed antibody-dependent enhancement of infection (ADE), was first hypothesized in the 1970s by Scott Halstead, a renowned scientist and one of the world's top experts on dengue virus infection. Dr. Halstead said he got his first inkling of the phenomenon while doing extensive clinical studies of dengue virus patients in Thailand in the 1960s.

He said: "We were able to detect that the severe patients all had a secondary antibody response, meaning that they had all been infected before.

"That was the first evidence we had that a person had to have a previous dengue infection to get the severe disease."

Further epidemiological observations, including cases in which severe dengue virus occurred in infants born to previously infected mothers, along with lab cell studies, prompted Dr. Halstead to put forth the ADE hypothesis.

Dr. Shresta's work, conducted in mouse models, provides the first in vivo proof of ADE's occurrence.

Commenting on the latest research, Dr. Halstead said: "A model like this is really a breakthrough in tools.

"We've been looking for 40 years for a model to be able to test this (ADE) phenomenon. It will allow us to study the virus and the antibody enhancement in ways never before possible."

Using the mouse model, Shresta's group has already made a key and surprising observation that a type of liver cells, called liver sinusoidal endothelial cells (LSCEs), but not the previously expected cells types (such as macrophages and dendritic cells) support ADE of dengue infection.

Scientists had long complained that the lack of a good animal model hampered efforts to develop a first-ever dengue vaccine. Dr. Shresta said her group's ADE findings emphasize the importance of special caution in designing a dengue virus vaccine.

She said: "Researchers will have to be extremely careful to ensure that, under no conditions, will a dengue vaccine generate these subneutralizing antibody conditions.

"Otherwise, it could cause people to develop the severest and potentially lethal form of the disease -- dengue hemorrhagic fever/dengue shock

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syndrome."

Dr. Halstead agreed and said: "The vaccine should cause you to make antibodies to each of the four dengue viruses.

"That's what makes it difficult; you have to make four vaccines in one. The kind of model Dr. Shresta has done will be important as researchers work to develop a vaccine."

Dr. Shresta said the fact that dengue viruses exist as four different serotypes that circulate simultaneously underlies the development of the subneutralizing antibodies. Infection with one of these serotypes provides lifelong immunity to the infecting serotype only. In subsequent dengue infections, where a different serotype of the virus is involved, the antibodies do not recognize enough of the virus to neutralize it.

She said: "This starts a cascade of unusual molecular events - the ADE process-which leads to the antibodies contributing to, rather than fighting, the dengue infection."

Shresta's findings have appeared online in Cell Host and Microbe in her paper entitled, "Enhanced Infection of Liver Sinusoidal Endothelial Cells in a Mouse Model of Antibody-Induced Severe Dengue Disease."

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