

## **Researchers identify protein that fights West Nile virus**

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(PhysOrg.com) -- Yale and McGill University scientists have identified a protein that is critical in fighting mosquito-borne West Nile Virus in mice. This finding could have therapeutic implications for controlling the potentially deadly virus in humans. The study appears in the Advance Online Publication of *Nature Immunology*.

Researchers studied the role of caspase-12, a protein that activates secretion of substances that are part of the body's immune response. Caspase-12's function in fighting bacterial infection has been studied before, but its role in viral immunity has not.

The Yale-McGill team found that caspase-12 regulated the signaling of RIG-I, a protein of the immune system that detects viral infection by recognizing its <u>genetic components</u>. This recognition pattern is necessary to trigger the immune system's response to <u>West Nile Virus</u>.

"Mice without caspase-12 protein died more rapidly from West Nile virus infection and had higher levels of virus than normal mice," explained co-author Erol Fikrig, M.D., professor of epidemiology, public health and <u>microbial pathogenesis</u> at Yale School of Medicine and a Howard Hughes Medical Institute investigator.

West Nile Virus has spread rapidly through North America over the past decade, and can be life-threatening to those with susceptible immune systems.



The team's findings open possible avenues of future research on whether the human immune system can be regulated in the same way, noted coauthor Maya Saleh, Ph.D., assistant professor of medicine at McGill University and medical scientist in the Division of Critical Care at the Research Institute of the McGill University Health Centre. "Our results now set the stage for the development of pharmacological compounds to boost the activity of caspase-12 in promoting virus elimination," Saleh said. "These findings have significant potential to be translated into therapies."

Other authors are Penghua Wang, Alvaro Arjona, Yue Zhang, Jianfeng Dai and Long Yang of Yale School of Medicine, Hameeda Sultana of Yale and a Howard Hughes Medical Institute Investigator, Philippe M. LeBlanc and Karine Doiron of McGill University Department of Microbiology and Immunology.

Provided by Yale University

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