Study identifies mutations that promote HIV-1 infection in the brain

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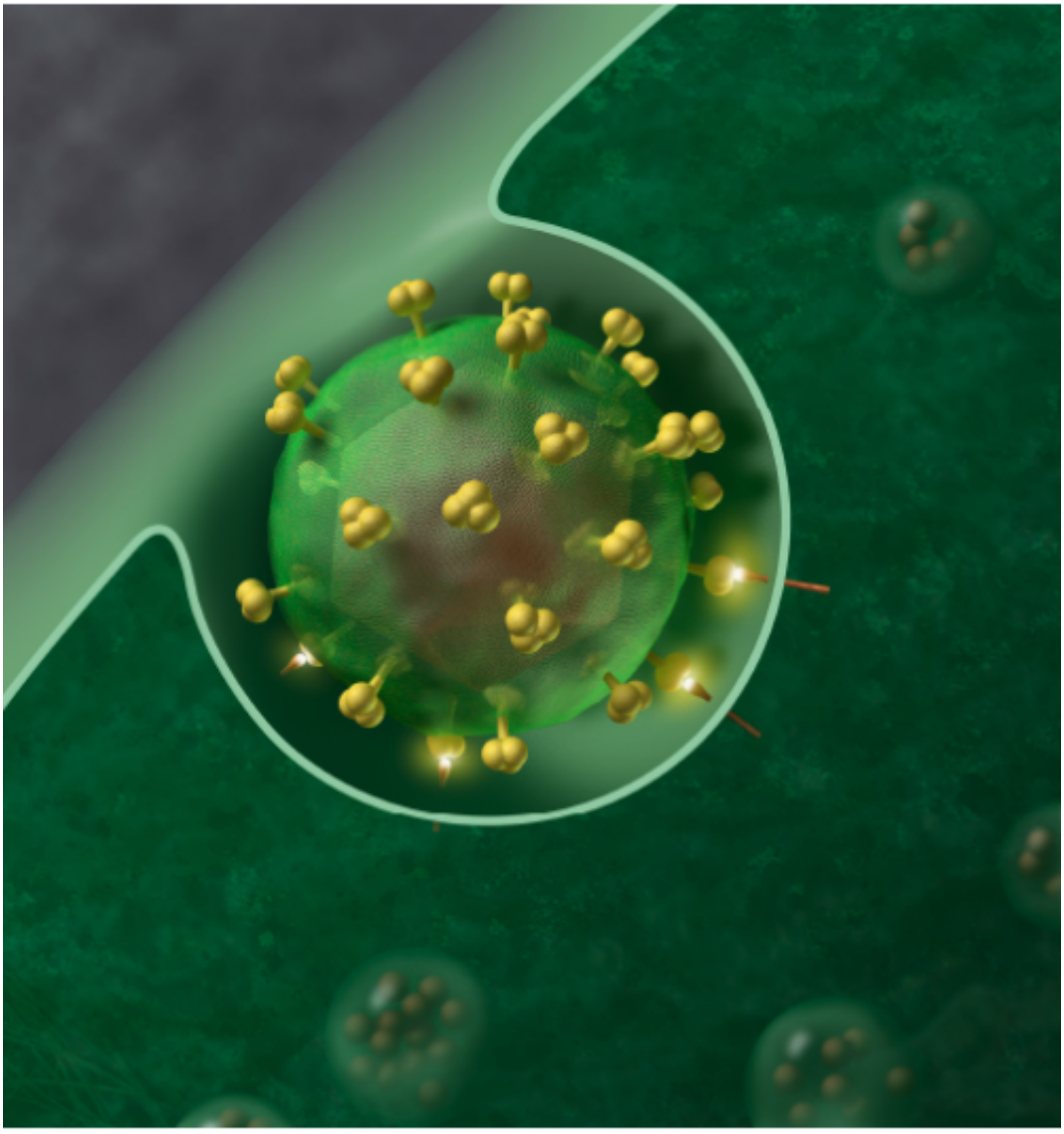

HIV-1 Virus. Credit: J Roberto Trujillo/Wikipedia

Although combinatorial antiretroviral therapies (cARTs) have dramatically reduced the occurrence of HIV-1-related dementia and encephalitis, recent studies have shown a rise in the number of HIV-positive individuals who will experience minor cognitive and neurological symptoms. Identifying the mechanisms that are important for viral infection in the central nervous system (CNS) could lead to more effective therapies for preventing HIV-1-related neurocognitive disorders.

In this month's issue of the *JCI*, a research team led by Vanessa Hirsch at the National Institute of Allergy and Infectious Diseases discovered that BST-2, a protein expressed on immune cells, is an important target for viral replication in the brain. They identified 4 amino acid changes in the viral envelop of a neurovirulent SIV strain that are not present in a parental strain that does not readily infect the CNS. Introduction of these mutations into the parental strain resulted in efficient viral replication in the CNS of a non-human primate model. The authors determined that the neurovirulent envelope inhibits BST-2, which is known to restrict viral release from infected cells.

These results suggest that BST-2 limits viral replication in the CNS and that preventing HIV inhibition of this factor has potential as a strategy for reducing HIV-1-related neurocognitive disorders.

More information: Kenta Matsuda et al, Enhanced antagonism of BST-2 by a neurovirulent SIV envelope, *Journal of Clinical Investigation* (2016). DOI: 10.1172/JCI83725

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