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Researchers identify how herpes virus infects host cells

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Virginia Commonwealth University researchers have uncovered new information about how the herpes simplex virus takes control of the host cell, setting the stage for the development of antiviral drugs that serve to fight herpes infections.

In the United States, the herpes simplex virus, or HSV, is common and can cause symptoms on the mouth or genitals in the form of cold sores or fever blisters. The virus is spread through close contact with an infected person. In rare cases, transmission of the virus can occur between mother and newborn at birth. Under these circumstances, HSV can cause devastating infections of newborns, including fatal encephalitis or inflammation of the brain.

Anthony V. Nicola, Ph.D., associate professor in the Department of Microbiology and Immunology in the VCU School of Medicine, and Mark G. Delboy, a predoctoral candidate in Nicola's laboratory who led the study, report that HSV takes complete control of normal cell function, or parasitizes, to infect host cells.

Specifically, they found that HSV requires the proteasome for entry into target cells. The proteasome is a large enzyme complex in all cells that is essential for normal cellular functions. The findings are published in the April issue of the *Journal of Virology*. Additionally, the research paper is featured by the editors of the journal in the Spotlight column as an article of significant interest.

"HSV initiates infection by taking advantage of cellular machinery that is required for normal host function. If we understand how HSV initiates infection, we can design improved ways to prevent infection and disease," Nicola said.

"Further, this research has identified new targets that can potentially be used to develop novel antiviral drugs against herpes infections. HSV appears to be the first virus to take advantage of the proteasome in this way," he said.

In future studies, Nicola and his team plan to examine and identify the virus and cell proteins that are involved in this process. Identification of the mechanism of action will further define targets for antiviral intervention, said Nicola.

Source: Virginia Commonwealth University