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Researchers show an oncolytic virus switches off cancer cell surival signal

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Researchers from Boston University School of Medicine (BUSM) have identified a mechanism by which specific viruses acting as oncolytic agents can enter and kill cancer cells. This finding, which is currently featured in an online edition of the *Journal of Virology*, could help lead to the development of more targeted treatments against many types of cancer.

The study was conducted by Ewan F. Dunn, a postdoctoral fellow, under the direction of John H. Connor, an assistant professor of microbiology at BUSM.

The virus, known as vesicular stomatitis virus (VSV), is being developed in the Connor lab and in other international research laboratories to kill cancer cells. VSV is not a significant human pathogen.

VSV is sensitive to the innate immune response, which causes lymphocytes to release interferon and protect the body from developing an infection. Cancer cells lose the ability to respond in that way, said Dunn. "When cancer cells transform, they become non-responsive, leaving them vulnerable to viruses attacking the cell and its function."

Previous research has shown that a major signaling pathway in cancer cells, called the AKT signaling pathway, is frequently turned on. AKT signaling is a cell survival signal, helping to keep the cancer cells alive. The team demonstrated was that VSV can switch off that signaling pathway, which suggests that a single viral protein could play a major

role in cancer cell death.

"This study showed the important role of VSV in killing cancer cells through turning off a major survival signal," added Connor. "The identification of this mechanism is fundamental to understanding how VSV and other oncolytic viruses function."

Provided by Boston University Medical Center