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Virus kills melanoma in animal model, spares normal cells

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Researchers from Yale University School of Medicine have demonstrated that vesicular stomatitis virus (VSV) is highly competent at finding, infecting, and killing human melanoma cells, both in vitro and in animal models, while having little propensity to infect non-cancerous cells.

"If it works as well in humans, this could confer a substantial benefit on patients afflicted with this deadly disease," says Anthony van den Pol, a researcher on the study. The research was published online ahead of print in the *Journal of Virology*.

Most normal cells resist virus infection by activating antiviral processes that protect nearby cells. "The working hypothesis was that since many cancer cells show a deficient ability to withstand virus infection, maybe a fast-acting virus such as VSV would be able to infect and kill cancer cells before the virus was eliminated by the immune system," says van den Pol. And indeed, the virus was able to selectively infect multiple deadly human melanomas that had been implanted in a mouse model, yet showed little infectivity towards normal mouse cells, he says.

Many different mechanisms are involved in innate immunity, the type of immunity that combats viral infection. van den Pol plans to investigate which specific mechanisms are malfunctioning in cancer cells, knowledge that would be hugely beneficial both in understanding how cancer affects immunity, and in enhancing a virus' ability to target cancer cells, he says.

Melanoma is the most deadly skin cancer. Most melanomas are incurable once they have metastasized into the body. The incidence of melanoma has tripled over the last three decades, and it accounts for approximately 75 percent of skin cancer-related deaths.

More information: G. Wollmann, J.N. Davis, M.W. Bosenberg, and A.N. van den Pol, 2013. Vesicular stomatitis virus variants selectively infect and kill human melanomas but not normal melanocytes. J. Virol. Published ahead of print 3 April 2013 , doi:10.1128/JVI.03311-12

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