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Ras controls herpes virus penetration in cells

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The molecular basis of host-cell permissiveness to herpes simplex viruses (HSV) is under intense investigation because of HSV prevalence in human disease and its potential use as an anti-cancer therapeutic. In the August issue of Nature Cell Biology, Faris Farassati and colleagues from the University of Calgary, Alberta, Canada show for the first time that oncogenes in Ras signalling pathway are essential in host-cell permissiveness to herpes simplex virus 1.

Farassati *et al.* exposed to the HSV-1 virus (strain F) parental Swiss mouse embryo fibroblasts (NIH-3T3 cells) and NIH-3T3 cells transformed with the oncogenes v-*erb*B, *sos* or *ras* - all activators of the Ras signaling pathway. Permissiveness to HSV-1 infection (assessed by induction of cytopathic effects, viral protein synthesis and virus output) was significantly increased in NIH-3T3 cells transformed with Ras activators. In addition inhibitors of the Ras signaling pathway, such as farnesyl transferase inhibitor 1 and PD98059, effectively suppressed HSV-1 infection of ras-transformed cells (*Nat Cell Biol* 2001, **3**:745-750).

These results may provide models for treating herpes virus infections, for example by downregulating the Ras pathway in infected cells. The authors also speculate that the association of herpes infections with cancers may be due to enhanced HSV permissiveness of cancer cells with an activated Ras signaling pathway.

References

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2. University of Calgary, [http://www.ucalgary.ca/]