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EDITORS' CHOICE

Virology

For Whom the Toll-Like Receptor Signals

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Viruses are dependent on their hosts, and it therefore makes sense for them to have ways of monitoring the status of the host organism. Gregory *et al.* report that Kaposi's sarcoma-associated herpesvirus (KSHV, which causes Kaposi's sarcoma in humans) monitors a danger signal from the host's innate immune system. The virus exists in latent form in B lymphocytes but in response to certain cues will enter the lytic, replicative phase of its life cycle. In searching for physiological cues that might regulate reactivation of the virus, Gregory *et al.* focused on Toll-like receptors (TLRs), which allow the host innate immune system to detect infectious agents such as bacteria and viruses. TLR7 and TLR8, for example, recognize RNA molecules that may be derived from infectious agents or dying host cells. The authors found that stimulation of a human lymphocyte cell line latently infected with KSHV with single-stranded RNA and consequent activation of TLR8 led to reactivation of KSHV. Similarly, infection of cultured lymphocytes with another virus, vesicular stomatitis virus (which activates TLR7 and TLR8), also caused reactivation of latent KSHV. The authors propose that such sensitivity to signaling by the host's innate immune system allows the virus to initiate replication and escape from host cells that are likely to die.

S. M. Gregory, J. A. West, P. J. Dillon, C. Hilscher, D. P. Dittmer, B. Damania, Toll-like receptor signaling controls reactivation of KSHV from latency. *Proc. Natl. Acad. Sci. U.S.A.* **106**, 11725–11730 (2009). [[Abstract](#)] [[Full Text](#)]

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