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Viral protein K5 modulates T-cell costimulation

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Herpesviruses use elaborate strategies to establish themselves in their host, including evading, destroying or redirecting immune cells. In the June 15 Journal of Clinical Investigation, Coscoy and Ganem describe how viral proteins from the human herpesvirus associated with Kaposi's sarcoma impair the ability of B cells to induce the protective response of cytotoxic T lymphocytes.

Coscoy and Ganem examined the ability of two viral genes, *K3* and *K5*, to influence the disposition of host surface proteins implicated in immune recognition and activation. They found that expression of K5, but not K3, in B cells dramatically reduces the expression of ICAM-1 and B7-2 on the cell surface, causing these proteins to be internalized rapidly and routed to the lysosome. This downregulation of two proteins known to be important in signalling between cells of the immune system is functionally significant, because K5-transfected B cells show substantial impairment in their ability to induce T-cell activation (*J Clin Invest* 2001*,* **107**:1599-1606).

K5 is thus the first example of a viral modulator of immunological synapse formation and T cell co-stimulation, and its effect is probably most important early in the disease when most viral antigens are confined to infected B cells.

References

1. Coscoy L, Ganem D: A viral protein that selectively downregulates ICAM-1 and B7-2, key components of the immunological synapse, modulates T cell costimulation. *J Clin Invest* 2001*,* 107:1599-1606., [http://www.jci.org/cgi/content/abstract/107/12/1599]