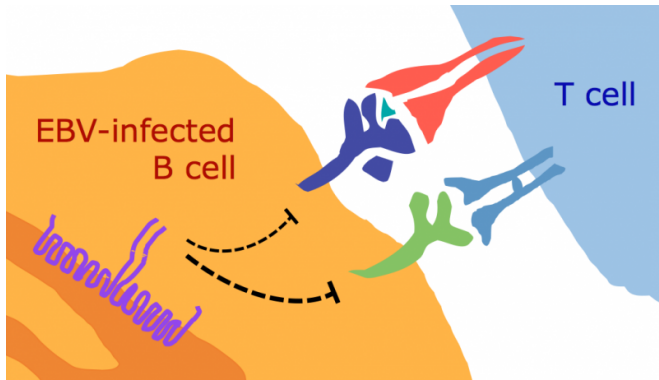


A viral protein that helps EBV-infected B cells to escape human killer T cells

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In EBV-infected cells, the viral protein LMP2A (violet) cuts back the number of molecules that activate antiviral T cells. Credit: Rancan, CC-BY

About 90% of adults worldwide are infected with Epstein-Barr virus, or EBV. The virus infects B cells (the white blood cells that make antibodies) and can contribute to B-cell-derived cancers, but in most people it remains dormant—a state scientists refer to as "latent infection"—for the rest of their lives. A study published on June 11th in *PLOS Pathogens* sheds new light on why the infected person's immune system cannot eliminate EBV, or the associated cancer risk.

Interested in the [immune response](#) against EBV, Andreas Moosmann, from the Helmholtz-Zentrum in Munich, Germany, and colleagues focused in this study on the role of a viral protein called LMP2A, which is present in latently infected B [cells](#) and also in many EBV-associated cancers, which have somehow escaped detection and elimination by the immune system. The scientists studied an engineered EBV [virus](#) that cannot make LMP2A and compared this mutant virus with the normal one.

They infected human B cells with normal and LMP2A-deficient EBV. Because EBV transforms

these cells, meaning that they can be changed to grow indefinitely, the researchers were able to examine so-called lymphoblastic cell lines that contained either virus. They found that LMP2A counteracts the recognition of EBV-infected B cells by EBV-specific immune lymphocytes called CD8+ killer T cells. In contrast, EBV-transformed cells without LMP2A are more efficiently identified, and the ability of these T cells to recognize and kill the EBV-infected B cells is enhanced.

Examining the mechanism underlying the LMP2A-mediated evasion, they found several ways in which it interferes with the recognition of EBV-infected cells. First, LMP2A reduced levels of several EBV proteins whose fragments are recognized by CD8+ T cells on the surface of the cell targeted for killing. Second, LMP2A disturbs expression of cellular molecules on infected B cells that interact with NKG2D, a host molecule on the surface of CD8+ T cells that aids their activation, thereby weakening the immune response against EBV-infected cells.

"Taken together", the researchers conclude, "we describe here a functional immunomodulatory effect for the EBV protein LMP2A, and show that LMP2A mediates partial escape of infected B cells from recognition by CD8+ T cells." They also suggest that that similar immune evasion mechanisms to the ones revealed may operate in different types of LMP2A-expressing cancers caused by EBV.

More information: Rancan C, Schirrmann L, Hüls C, Zeidler R, Moosmann A (2015) Latent Membrane Protein LMP2A Impairs Recognition of EBV-Infected Cells by CD8+ T Cells. *PLoS Pathog* 11(6): e1004906. [DOI: 10.1371/journal.ppat.1004906](#)

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