

How and why herpes viruses reactivate to cause disease

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The mere mention of the word "herpes" usually conjures negative images and stereotypes, but most people have been infected with some form of the virus. For most, a sore appears, heals and is forgotten, although the virus remains latent just waiting for the right circumstances to come back. Now, the mystery behind what triggers the virus to become active again is closer to being solved thanks to new research published in the *Journal of Leukocyte Biology's* November 2012 issue. In the report, scientists show how the immune system may lose its control over the virus when facing new microbial threats, such as when it must fend off other viral invaders or bacteria.

"Because almost all people are infected by one or more herpes family viruses during their lifetime, the potential impact of these findings are significant," said Charles H. Cook, M.D., FACS, FCCM, director of surgical critical care at The Ohio State University College of Medicine in Columbus, Ohio, and a researcher involved in the work. "We hope that by understanding how these latent [viral infections](#) are controlled that we can prevent reactivation events and improve people's lives."

To make this discovery, researchers studied mice with latent herpes family cytomegalovirus (CMV) during severe bacterial infections. They found that T-cells responsible for CMV control were reduced significantly during a new infection with bacteria. This, in effect, reduced the "brakes" which kept the virus under control, allowing the virus to reactivate and cause disease. When the [immune system](#) eventually sensed the reactivation, the memory T-cell levels returned to normal, effectively restoring the body's control over the virus.

"Finding ways to control herpes flare ups is important, not only for the health of the person with the virus, but also for preventing its transmission," said John Wherry, Ph.D., Deputy Editor of the [Journal of Leukocyte Biology](#). "This report highlights the important interplay when we are 'co-

infected' with more than one microbe and provides important insights into why the immune system sometimes fails as well as how it can regain control of latent [herpes](#) virus infections."

More information: Jonathan Campbell, Joanne Trgovcich, Michelle Kincaid, Peter D. Zimmerman, Paul Klenerman, Stuart Sims, and Charles H. Cook. Transient CD8-memory contraction: a potential contributor to latent cytomegalovirus reactivation. *J Leukoc Biol* 92:933-937; [doi:10.1189/jlb.1211635](https://doi.org/10.1189/jlb.1211635)

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