Study finds herpesvirus activates RIG-I receptor to evade body's immune system

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Using herpesvirus, molecular immunologists from the University of Southern California (USC) Norris Comprehensive Cancer Center have discovered a cellular process that activates a critical immune defense against pathogens, which could have implications for developing drugs to bolster one's immunity to infection. Some herpesvirus infections lead to cancer.

Led by Pinghui Feng, Ph.D., associate professor of molecular microbiology and immunology at the Keck School of Medicine of USC, the team found that herpesvirus proteins activate retinoic acid-induced gene I (RIG-I) by removal of an amino group from the glutamine and asparagine amino acids through a process called deamidation. RIG-I is a cellular receptor that recognizes RNA derived from invading pathogens.

Prior to this study, it was unclear whether RIG-I—whose activation is central to the body's innate immune defense response—could be activated by a component other than viral RNA. This is the first example wherein host immune defense is activated by an enzymatic activity, implying that deamidation can be a highly regulated process, reshaping the conventional notion that deamidation is a non-specific process associated with protein decay. The team also identified the first bona-fide enzyme that causes protein deamidation in eukaryotes.

The study appears in the April 2 issue of the peer-reviewed scientific journal *Molecular Cell*.

More information: He, S., Zhao, J., Song, S., He, X., Minassian, A., Zhou, Y. ... & Feng, P. (2015). Viral pseudo-enzymes activate RIG-I via deamidation to evade cytokine production. *Molecular Cell*, 57(6). Published online March 5, 2015. DOI: dx.doi.org/10.1016/j.molcel.2015.01.036