Researchers discover gene that suppresses herpesviruses

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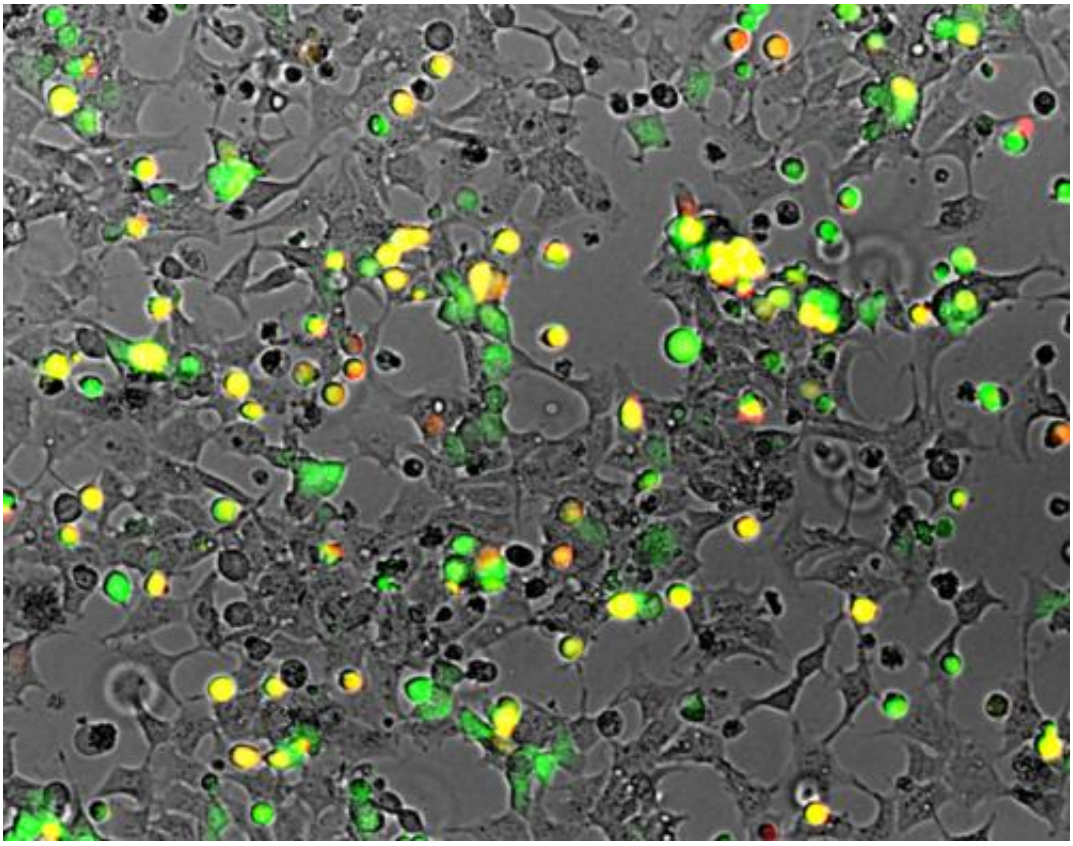

Cells infected with the KSHV virus fluoresce yellow. The KSHV virus remains dormant in more than 95 percent of infected patients. Credit: UNC/Damania Lab

Kaposi's sarcoma-associated herpesvirus (KSHV) and Epstein-Barr virus (EBV) hide within the worldwide human population. While dormant in

the vast majority of those infected, these active herpesviruses can develop into several forms of cancer. In an effort to understand and eventually develop treatments for these viruses, researchers at the University of North Carolina have identified a family of human genes known as Tousled-like kinases (TLKs) that play a key role in the suppression and activation of these viruses.

In a paper published by *Cell Host and Microbe* on Feb. 13, a research team led by Blossom Damania, PhD, of the Department of Microbiology and Immunology and member of the UNC Lineberger Comprehensive Cancer Center, found that suppressing the TLK enzyme causes the activation of the lytic cycle of both EBV and KSHV. During this active phase, these viruses begin to spread and replicate, and become vulnerable to anti-viral treatments.

"When TLK is present, these viruses stay latent, but when it is absent, these viruses can replicate" said Dr. Damania.

Patrick Dillon, a postdoctoral fellow in Dr. Damania's lab, led the study. Other co-authors included UNC Lineberger members Drs. Dirk Dittmer, Nancy Raab-Traub and Gary Johnson.

KSHV and EBV are blood-borne viruses that remain dormant in more than 95 percent of those infected, making treatment of these viruses difficult. Both viruses are associated with a number of different lymphomas, sarcomas, and carcinomas, and many patients with suppressed immune systems are at risk for these virus-associated cancers.

"The dormant state of these viruses is what makes it so hard to treat these infections and the cancers associated with these infections," said Dr. Damania.

Researchers have known that stimuli such as stress can activate the virus from dormancy, but they do not understand the molecular basis of the viral activation cycle. With the discovery of the link between these viruses and TLKs, Dr. Damania said that researchers can begin to look for the molecular actions triggered by events like stress, and how they lead to the suppression of the TLK enzymes.

"What exactly is stress at a molecular level? We don't really understand it fully," said Dr. Damania.

With the discovery that TLKs suppresses these viruses, Dr. Damania said that the proteins can now be investigated as a possible drug target for these virus-associated cancers. In its normal function in the cell, TLKs play a role in the maintenance of the genome, repairing DNA and the assembly of the chromatin, but there is a lot more to learn about the function of the TLKs, said Dr. Damania. One avenue of her lab's future research will investigate how TLKs function in absence of the virus.

"If we prevent this protein from functioning, and we combine this with a drug that inhibits viral replication, then we could have a target to cure the cell of the virus. If the virus isn't there, the viral-associated cancers aren't present," said Dr. Damania.

Provided by University of North Carolina Health Care

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