Researchers reveal Epstein-Barr virus protein contributes to cancer

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Researchers at the University of Toronto have shown that the EBNA1 protein of Epstein-Barr virus (EBV) disrupts structures in the nucleus of nasopharyngeal carcinoma (NPC) cells, thereby interfering with cellular processes that normally prevent cancer development. The study, published October 3rd in the open-access journal *PLoS Pathogens,* describes a novel mechanism by which viral proteins contribute to carcinogenesis.

EBV is a common herpesvirus whose latent infection is strongly associated with several types of cancer including NPC, a tumor that is endemic in several parts of the world. With NPC only a few EBV proteins are expressed, including EBNA1. EBNA1 is required for the persistence of the EBV genomes, however, whether or not EBNA1 directly contributes to the development of tumors has not been clear, until now.

In this study Frappier and her team examined PML nuclear bodies and proteins in EBV-positive and EBV-negative NPC cells. Manipulation of EBNA1 levels in each cell type clearly showed that EBNA1 expression induces the loss of PML proteins and PML nuclear bodies through an association of EBNA1 with the PML bodies. PML nuclear bodies are known to have tumor-suppressive effects due to their roles in regulating DNA repair and programmed cell death, and accordingly, EBNA1 was shown to interfere with these processes.

The researchers conclude that there is "an important role for EBNA1 in

the development of NPC, in which EBNA1-mediated disruption of PML nuclear bodies promotes the survival of cells with DNA damage." Since EBNA1 is expressed in all EBV-associated tumors, including B-cell lymphomas and gastric carcinoma, these findings raise the possibility that EBNA1 could play a similar role in the development of these cancers. The cellular effects of EBNA1 in other EBV-induced cancers will require further investigation.

Citation: Sivachandran N, Sarkari F, Frappier L (2008) Epstein-Barr Nuclear Antigen 1 Contributes to Nasopharyngeal Carcinoma through Disruption of PML Nuclear Bodies. PLoS Pathog 4(10): e1000170. doi:10.1371/journal.ppat.1000170
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