

How Epstein-Barr Virus Leads to Tumor Development

EBV causes chromosome breakage that can trigger a cascade of genomic instability leading to tumor growth.

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More than 90% of the world's population has been infected with Epstein-Barr Virus (EBV), and for most people, the infection is mild and passes in childhood. But for some, the virus persists in the body and increases the risk of certain cancers, including lymphoma, leukemia, and head and neck cancer. How exactly EBV leads to cancer, however, has until now remained poorly understood.

But a new study from Damon Runyon Fellow Julia Su Zhou Li, PhD, and her colleagues at Ludwig Institute for Cancer Research sheds light on the mechanism by which EBV causes chromosome breakage in the human genome, triggering a cascade of genomic instability that results in tumor growth.

The research team zoomed in on a viral protein, known as EBNA1, that is present in cells infected with EBV. Previous studies have shown that EBNA1 binds to a specific site in the EBV genome: an 18-base-pair sequence repeated twenty times. Now, Dr. Li and her colleagues have shown that EBNA1 also binds to a similar, "EBV-like" sequence on human chromosome 11. This binding site, the researchers noticed, has the structure of other sites in the genome known for their susceptibility to breakage—"fragile sites," as they are called. An accumulation of EBNA1 proteins at this site causes chromosome 11 to break, which can promote tumor growth through a number of paths; certain key genes for regulating cell growth and suppressing tumors are located on chromosome 11, and the downstream effects of genomic instability can lead to other key genes becoming activated or inactivated.

Indeed, an analysis of nearly 2,500 cancers across 38 tumor types revealed that cancer cells with detectable EBV were much more likely to have chromosome 11 abnormalities, including 100% of EBV-associated head and neck cancers.

For a nearly ubiquitous virus that is often harmless but occasionally deadly, identifying how and where EBV wreaks havoc in the cell is a major step towards preventing EBV-associated cancers.

"Going forward, this knowledge paves the way for screening risk factors for the development of EBV-associated diseases," says Dr. Li. "Moreover, blocking EBNA1 from binding to chromosome 11 can be exploited to prevent their development."

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