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EPSTEIN-BARR COLLABORATIVE GROUP

New study analysis: link found between Epstein-Barr Virus and chromosome instability in cancer development

Article written for DetectEBV by Silvia Gramolelli

Silvia Gramolelli is an Italian researcher. After obtaining her master degree at Sapienza University of Rome, she moved to Germany to do a PhD in infection biology. There, she started working on oncogenic herpesviruses, in particular on Kaposi's sarcoma associated herpesvirus which is closely related to EBV. Then she moved to Helsinki where she continued working on important aspects of herpesviral oncogenesis, and she has now set up her own research group. She is currently focused on gastric cancer.

Understanding the molecular mechanisms underlying the development of certain cancers is a major challenge in contemporary medical research. A new study published in the journal Nature brings new insights into the links between the presence of Epstein-Barr virus (EBV) and the risk of developing different types of cancer. This study highlights the complex interactions between the proteins produced by EBV and human DNA sequences which can lead to structural instability of chromosome 11 and genetic alterations that promote tumor growth.

The Epstein-Barr virus (EBV) is often referred to as a common infection, yet this label is reductive given that it is present in 90% of humans, making it ubiquitous. While most people experience no symptoms, the presence of EBV has been linked to the development of various types of cancer, including lymphomas, nasopharyngeal carcinoma, and gastric cancer. Despite this link, how EBV-infected cells lead to cancer remains largely unknown.

In a recent study, Li and collaborators discovered that EBNA1, a protein produced by EBV, causes instability in the structural integrity of human chromosome 11, which could predispose individuals to cancer. This occurs when EBNA1 binds to specific human DNA sequences on chromosome 11 and induces the formation of unstable structures that tend to break, creating fragile sites. This instability increases the likelihood of DNA ruptures and gene copies alterations, ultimately affecting the regulation of cell growth.

But how this relates to EBV-induced cancer?

To answer this question Li and collaborators also analyzed cancer databases and found that EBV-associated nasopharyngeal carcinoma cases had structural alterations of chromosome 11 in 80% of cases. Further genome sequence analysis of over 2000 cancers showed that detectable EBV was frequently associated with DNA breaks on chromosome 11.

Li's and his collaborators' discovery opens up new avenues of research to better understand the more complex molecular mechanisms underlying the development of certain cancers. It also underscores the importance of interdisciplinary approaches to answer these complex questions. Knowledge of protein interactions by EBV with human DNA also paves the way for the development of new therapeutic strategies aimed at preventing the progression of cancers associated with EBV infection. These results demonstrate the importance of continuing basic research in the field of molecular biology, which will broaden the horizons of the fight against cancer and save lives.

References

Published: 12 April 2023 - Chromosomal fragile site breakage by EBV-encoded EBNA1 at clustered repeats. Julia Su Zhou Li, Ammal Abbasi, Dong Hyun Kim, Scott M. Lippman, Ludmil B. Alexandrov & Don W. Cleveland. https://doi.org/10.1038/s41586-023-05923-x

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