

## The Effect of the Epstein-Barr Virus on the Development of Multiple Sclerosis

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**Abstract :** Background and Objective: Multiple sclerosis (MS) is the most common inflammatory autoimmune disease of the central nervous system (CNS) that affects the myelination process in the CNS. Complex interactions of various "environmental or infectious" factors may act as triggers in autoimmunity and disease progression. The association between viral infections, especially Epstein-Barr virus (EBV) and MS, is one potential cause that is not well understood. In this study, we aim to summarize the available data on EBV infection in MS disease progression. Materials and Methods: For this study, the keywords "Multiple sclerosis," "Epstein-Barr virus," and "central nervous system" in the databases PubMed, Google Scholar, Sid, and MagIran between 2016 and 2022 were searched, and 14 articles were chosen, studied, and analyzed. Results: Demyelinated lesions isolated from MS patients contain EBNAs from EBV proteins. The EBNA1 domain contains a pentapeptide fragment identical to B-crystallin, a heat shock peptide, that is increased in peripheral B cells in response to B-crystallin infection, resulting in myelin-directed autoimmunity mediated by proinflammatory T cells. EBNA2, which is involved in the regulation of viral transcription, may enhance transcription from MS risk loci. A 7-fold increase in the risk of MS has been observed in EBV infection with HLA-DR15 synergy. Conclusion: EBV infection along with a variety of specific genetic risk alleles, cause inflammatory cascades in the CNS by infected B cells. There is a high expression of EBV during the course of MS, which indicates the relationship between EBV and MS, that this virus can play a role in the development of MS by creating an inflammatory state. Therefore, measures to modulate the expression of EBV may be effective in reducing inflammatory processes in demyelinated areas of MS patients.

**Keywords :** multiple sclerosis, Epstein-Barr virus, central nervous system, EBNAs

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