Abstract

Viruses have long been studied as developmental cofactors for their influence on the development of autoimmune diseases. Among the mechanisms under consideration, viral mechanisms of molecular mimicry, epitope spreading, and bystander cell activation may contribute to the development of autoimmunity.

An increase in the incidence of many autoimmune diseases, including type 1 diabetes mellitus, has been observed during the SARS-CoV-2 pandemic. The similarity of human and viral proteins, i.e. molecular mimicry, together with the inflammatory response that SARS-CoV-2 infection induces, are possible factors in the development of type 1 diabetes. However, the hyperglycemia and other markers associated with type 1 diabetes observed in SARS-CoV-2 patients are largely due to viral mechanisms contributing to the development of secondary diabetes and type 2 diabetes.

Epstein-Barr virus infection increases the risk of developing multiple sclerosis but the exact mechanism by which this occurs is not yet known. Molecular mimicry has been observed in the model of Epstein Barr-virus, whereas epitope spreading and bystander activation have been observed in the model of multiple sclerosis. These mechanisms represent hosts immune response which in turn has an opposite effect. Molecular mimicry, together with the Epstein-Barr virus's own mechanisms, by which it manipulates the host immune system, probably contribute to the pathogenesis of multiple sclerosis.

Key words: viruses, Epstein-Barr virus, diabetes mellitus, multiple sclerosis, autoimmune diseases, Covid-19