“How a common, everyday virus like Epstein-Barr virus likely induces autoimmune diseases such as multiple sclerosis”

Epstein-Barr virus (EBV) has been identified as a putative environmental trigger of multiple sclerosis (MS), yet EBV’s role in MS remains elusive. We utilized murine gamma herpesvirus 68 (γHV-68), the murine homolog to EBV, to examine how infection by a virus like EBV could enhance CNS autoimmunity. Mice latently infected with γHV-68 developed more severe EAE including heightened paralysis and mortality. Similar to MS, γHV-68 EAE mice developed lesions composed of CD4 and CD8 T cells, macrophages and loss of myelin in the brain and spinal cord. Further, T cells from the CNS of γHV-68 EAE mice were primarily Th1, producing heightened levels of IFN-γ and T-bet accompanied by IL-17 suppression, whereas a Th17 response was observed in uninfected EAE mice. Clearly, γHV-68 latency polarizes the adaptive immune response, directs a heightened CNS pathology following EAE induction reminiscent of human MS and portrays a novel mechanism by which EBV likely influences MS and other autoimmune diseases.