Title:	Different Mechanisms of Inflammation Induced in Virus and Autoimmune-Mediated Models of Multiple Sclerosis in C57BL6 Mice
Authors:	Kanaujia, Anurag (/jspui/browse?type=author&value=Kanaujia%2C+Anurag)
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Abstract:	Multiple sclerosis (MS) is an inflammatory demyelinating disease of the human central nervous system (CNS). Neurotropic demyelinating strain of MHV (MHV-A59 or its isogenic recombinant strain RSA59) induces MS-like disease in mice mediated by microglia, along with a small population of T cells. The mechanism of demyelination is at least in part due to microglia-mediated myelin stripping, with some direct axonal injury. Immunization with myelin oligodendrocyte glycoprotein (MOG) induces experimental autoimmune encephalomyelitis (EAE), a mainly CD4+ T-cell-mediated disease, although CD8+ T cells may play a significant role in demyelination. It is possible that both autoimmune and nonimmune mechanisms such as direct viral toxicity may induce MS. Our study directly compares CNS pathology in autoimmune and viral-induced MS models. Mice with viral-induced and EAE demyelinating diseases demonstrated similar patterns and distributions of demyelination that accumulated over the course of the disease. However, significant differences in acute inflammation were noted. Inflammation was restricted mainly to white matter at all times in EAE, whereas inflammation initially largely involved gray matter in acute MHV-induced disease and then is subsequently localized only in white matter in the chronic disease phase. The presence of dual mechanisms of demyelination may be responsible for the failure of immunosuppression to promote long-term remission in many MS patients.
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