

Wednesday, May 2, 2012  
12:00pm  
in LSC3

## Dr. Marc Horwitz

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## “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 ( $\gamma$ HV-68), the murine homolog to EBV, to examine how infection by a virus like EBV could enhance CNS autoimmunity. Mice latently infected with  $\gamma$ HV-68 developed more severe EAE including heightened paralysis and mortality. Similar to MS,  $\gamma$ HV-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  $\gamma$ HV-68 EAE mice were primarily Th1, producing heightened levels of IFN- $\gamma$  and T-bet accompanied by IL-17 suppression, whereas a Th17 response was observed in uninfected EAE mice. Clearly,  $\gamma$ HV-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.

This Seminar is sponsored by:

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Host: Dr. Ed Prydzial, Clinical Professor Pathology and Laboratory Medicine & Centre for Blood Research

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Refreshments will be served 10 minutes before the seminar  
Seminar information: 604 822 7407

