KANSAS STATE UNIVERSITY

Division of Biology Presents:

The Role of CSF in a Murine Model of Neuroborreliosis

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Lyme Neuroborreliosis (LNB) caused by infection with the bacterium Borrelia burgdorferi (Bb), occurs in 10–15% of Lyme disease cases. Central nervous system (CNS) manifestations can include lymphocytic meningitis, radiculoneuritis, cranial neuritis, and rarely encephalitis and vasculitis. We have developed a model of CNS borreliosis in mice, and our long-term goal is to utilize this model to identify pathogen, host, and environmental factors that trigger CNS involvement in order to develop new diagnostic markers as well as prophylactic and therapeutic treatments for LNB.

The meninges serves as an interface between CNS and periphery that acts to maintain homeostasis and immunity. The outermost layer of the meninges, the dura mater, possesses fenestrated blood vessels, lymphatic drainage, and a high density of resident immune cells capable of supporting a robust immune response. We demonstrated persistent Bb colonization of the dura mater with leukocyte infiltration and mild meningitis, indicating an inflammatory state in the meninges of Bb-infected mice.

In contrast to the dura mater, the leptomeninges and brain parenchyma possess barriers to entry in the form of the arachnoid barrier, blood-brain-barrier, and blood-cerebrospinal fluid (CSF) barrier. Nonetheless, our data show an increase in inflammatory markers including IFN-stimulated genes in both cortex and hippocampus of infected mouse brains; despite a lack of detectable spirochetes in the brain parenchyma. The stimulus for this sterile inflammation in the brain remains unclear. The CSF is an important signaling medium between the meninges and the brain, and patients with clinical LNB often present with inflammatory cytokines/chemokines, lymphocytic pleocytosis, Bb-specific antibodies, and occasionally live Bb in the CSF.

If you would like to visit with Dr. Catherine Brissette, please contact Dr. Vanessa Ante at ante@ksu.edu.

Coffee & snacks served preceding the seminar in Ackert Hall, Room 225