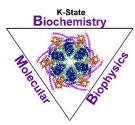
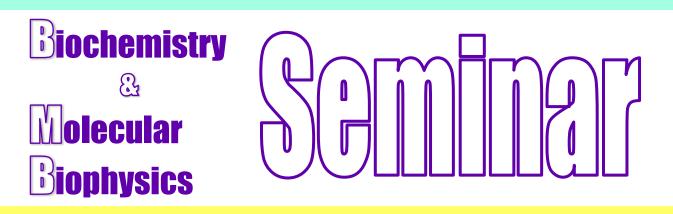
Ackert Hall, Room 120 Wednesday, November 6 4:00 P.M.



Coffee and Cookies Chalmers Hall, Room 168 3:45 P.M.



The *Borrelia burgdorferi* cAMP pathway: *cyaB* regulates virulence

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Borrelia burgdorferi is the causative agent of Lyme disease, the most common vector-borne illness in the United States with symptoms ranging from flu-like sickness to debilitating arthritis. B. burgdorferi exists in a complex enzootic cycle transitioning between the disparate environments of a tick vector and vertebrate host. The establishment of long-term infection in the host develops from B. burgdorferi regulation of virulence factors in response to unknown environmental cues. The focus of my research is on understanding the molecular mechanisms used by *B. burgdorferi* to regulate pathogenesis with a particular emphasis on characterizing *B. burgdorferi* genetic and physiological adaptation to the host environment. My research characterizing the *B. burgdorferi* adenylate cyclase *cyaB* has established it plays a role in regulating mammalian virulence factors and infectivity. Found in both eukaryotes and prokaryotes, adenylate cyclases are the enzymes responsible for converting ATP into the important second messenger cAMP. In bacteria, signaling pathways involving cAMP control a wide range of cell responses, including pathogenesis. I will discuss how B. burgdorferi cyaB regulates mammalian virulence factors important in adhesion and dissemination, loss of *cyaB* results in attenuation in mice, and expression of cyaB is influenced by mammalian host factors. Taken together, my findings have begun defining the cAMP pathway, a novel signal transduction cascade in *B. burgdorferi* and a potential mechanism for directly relaying host environmental signals into the bacteria contributing to pathogenesis. The ultimate goal of this research is to translate the findings characterizing the *B. burgdorferi* cAMP signaling pathway to identify potential novel therapeutic targets for treating Lyme disease.