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Borrelia burgdorferi and the Subversion of the Adaptive Immune Response

Dr. Baumgarth is a Professor of Immunology at the Dept. of Pathology, Microbiology & Immunology and the Center for Comparative Medicine, University of California, Davis. Dr. Baumgarth's research encompasses studies on the regulation of immune responses to infections and B cell biology. Dr. Baumgarth received her veterinary degree and her PhD from the School of Veterinary Medicine, Hannover, Germany. She then conducted post-doctoral studies in Australia and at Stanford University. In 2000 she set-up her own lab at the University of California, Davis, at the Center for Comparative Medicine, then under the directorship of Dr. Stephen Barthold, with whom she began to collaborate.

Dr. Baumgarth has been continuously funded by the NIH for her studies on the regulation of B cell and B cell subset responses. She is also interested in the regulation and function of natural IgM, a product of innate-like B-1 cells. Dr. Baumgarth's laboratory investigates these topics using mouse models to two very different pathogens and immune responses: Acute influenza virus infection, an infection that fully resolves and induces highly protective and long-lived B cell-mediated immunity, and B cell responses to *Borrelia burgdorferi*, a bacterial spirochete and the causative agent of Lyme disease. Her laboratory is investigating how infection-induced inflammatory signals shape B cell response quality to

induce long-lasting local and systemic immunity to influenza. Her studies on *Borrelia burdorferi* infection of mice have shown a profound dysregulation in the T-dependent B cell responses after infection, explaining the establishment of bacterial persistence for the life of the mouse. Her current studies are focused on determining whether appropriate activation of CD4 T cells could overcome the immunological defects observed following infection with this pathogen and lead to bacterial clearance either alone or in conjunction with antibiotic treatments.

Conference Lecture Summary

Borrelia burgdorferi is capable of establishing persistent infections in a wide variety of species, particularly rodents. Infection is asymptomatic or mild in most reservoir host species, indicating successful co-evolution of the pathogen with its natural hosts. Infected humans and other incidental hosts, however, can develop Lyme disease, a serious inflammatory syndrome characterized by tissue inflammation of joints, heart, muscles, skin and CNS. While *B. burgdorferi* infection induces both innate and adaptive immune responses, they are ultimately ineffective in clearing the infection from reservoir hosts, leading to bacterial persistence. The goal of our work is to document evidence of immune suppression and to identify the exact immune targets of Borrelia such immune suppression. Here I will present studies conducted in our laboratory using mouse models of infection that identify the adaptive immune response, and particularly, “T cell dependent antibody responses” as a particular target. This type of immune response usually provides high affinity antibodies against pathogens and provides the host with long-lasting “memory” and immune protection in response to an infection Ineffective immunity would result in a lack of bacterial/pathogen clearance and allow for repeat infections to occur. A better understanding of the mechanisms causing

persistence in rodents may help to increase our understanding of the pathogenesis of Lyme disease and ultimately aid in the development of therapies that support effective clearance of the bacterial infection by the host’s immune system.