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The Roles of Macrophage Subsets in Response to Borrelia burgdorferi

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Borrelia burgdorferi is the causative agent of Lyme disease. Lyme disease is a bacterial infection commonly acquired by being bitten by an infected tick. Early symptoms of Lyme disease include fever, headache, fatigue, and a bulls-eye shaped rash. If untreated with antibiotics, patients with Lyme Disease can develop symptoms of carditis, arthritis, and neurological issues. C3H/HeJ mice develop an infective carditis and arthritis following infection with *Borrelia burgdorferi*. Following three weeks of infection, however, the infectious arthritis and carditis that spontaneously resolve approximately 3 weeks post-infection. It is suggested that macrophages play a significant role in this spontaneous resolution of the infection's symptoms. During inflammatory responses, pro-inflammatory M1 macrophages are present early in the response and then switch to anti-inflammatory M2 cells later and induce inflammation resolution. We hypothesize that M2 cells produce cytokine and lipid mediators that induce arthritis resolution. RAW cells are a murine macrophage cell line and will be used for experiments. Cells will be treated with lipopolysaccharide (LPS) and gamma interferon (IFN-γ) to stimulate the M1 phenotype, or with IL-4 to stimulate the M2 phenotype. The cells will then be co-cultured with *B. burgdorferi* for various time points. Cytokine production, lipid mediator production, and bacterial killing will be measured using ELISA, EIA, and a phagocytosis assay. The contribution of M1 and M2 cells in the development and resolution of Lyme arthritis is unclear. This study will provide important information regarding the function of M1 and M2 cells during infection with *B. burgdorferi* and may provide clues for development of future therapeutics.