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### **MCP-1 and its role in lyme arthritis**

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Lyme disease is caused by infection with the bacteria *Borrelia burgdorferi*, causing arthritis and carditis in infected animal models. The presence of spirochetes in the joint and other soft tissues induce recruitment of leukocytes to the area of infection and leads to the production of proinflammatory cytokines which are thought to play a role in the development of pathology. MCP-1 (macrophage chemoattractant protein-1) is one of the cytokines responsible for the recruitment of macrophages to infected tissue during normal disease pathogenesis. It was previously found that MCP-1 does not play a significant role in the pathogenesis of Lyme carditis, therefore the importance and role of MCP-1 in the development of Lyme arthritis was investigated. It was hypothesized that arthritis severity and pro-inflammatory cytokine production would be decreased in MCP-1 knock out (KO) mice, while the number of the number of *B. burgdorferi* organisms found in joint tissue of MCP-1 KO mice would be increased due the lack of recruited macrophages. MCP-1 KO and wild type (WT) C3H and B6 mice were infected with *B. burgdorferi* in the hind paws and they were sacrificed at 21 days post infection, the peak of Lyme disease pathogenesis. Ankle diameter was measured weekly throughout the infection and histological ankle sections were scored for the severity of inflammation on a scale from 0 to 3, according to the number of infiltration foci. Ankle diameter of C3H WT mice was on average greater than C3H MCP-1 KO mice, while the pro-inflammatory and anti-inflammatory cytokine levels were on average lower than KO mice. In B6 mice, WT mice had decreased ankle diameter and higher cytokine levels compared to B6 MCP-1 KO mice. These data suggest that MCP-1 may play more of a role in Lyme arthritis than Lyme carditis pathology.