Format: Abstract

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Interleukin-10 (IL-10) inhibits Borrelia burgdorferi-induced IL-17 production and attenuates IL-17-mediated Lyme arthritis.

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Abstract

Previous studies have shown that cells and cytokines associated with interleukin-17 (IL-17)-driven inflammation are involved in the arthritic response to Borrelia burgdorferi infection. Here, we report that IL-17 is a contributing factor in the development of Lyme arthritis and show that its production and histopathological effects are regulated by interleukin-10 (IL-10). Spleen cells obtained from B. burgdorferi-infected, "arthritis-resistant" wild-type C57BL/6 mice produced low levels of IL-17 following stimulation with the spirochete. In contrast, spleen cells obtained from infected, IL-10-deficient C57BL/6 mice produced a significant amount of IL-17 following stimulation with B. burgdorferi. These mice developed significant arthritis, including erosion of the bones in the ankle joints. We further show that treatment with antibody to IL-17 partially inhibited the significant hind paw swelling and histopathological changes observed in B. burgdorferi-infected, IL-10-deficient mice. Taken together, these findings provide additional evidence of a role for IL-17 in Lyme arthritis and reveal an additional regulatory target of IL-10 following borrelial infection.

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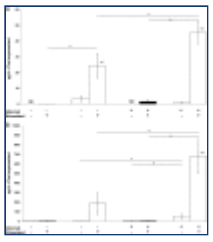
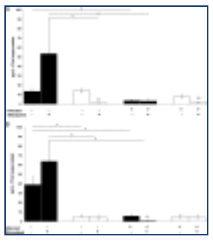
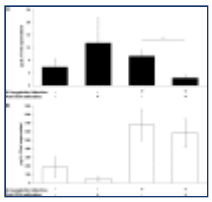
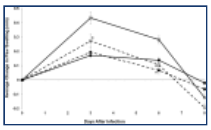
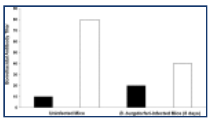

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