Abstract

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Vitamin A deficiency exacerbates murine Lyme arthritis.

Cantorna MT, Hayes CE.

Department of Biochemistry, University of Wisconsin-Madison 53706, USA.

OBJECTIVE: Vitamin A deficiency predisposes the host for a strong inflammatory response, suggesting that it may foster susceptibility to diseases, such as Lyme arthritis, in which activated macrophage and inflammatory cytokine production are pathogenic.

METHODS AND RESULTS: Infected mice had a rapid serum retinal decline that correlated with the onset of arthritis. The mice with the least retinol developed acute arthritis earlier and more severely than those with the highest retinol. Earlier and stronger interleukin (IL)-12, interferon-gamma (IFN)-gamma, and tumor necrosis factor responses were found in Borrelia burgdorferi-infected, vitamin A-deficient mice compared with controls. The spirochetes induced IFN-gamma secretion from unprimed cells, and retinoid addition in vitro inhibited IFN-gamma synthesis.

CONCLUSIONS: Vitamin A deficiency may exacerbate acute Lyme arthritis by enhancing an acute arthritogenic inflammatory response initiated by spirochete-driven IFN-gamma secretion. Conversely, vitamin A may lessen acute Lyme arthritis pathology by blocking IFN-gamma and IL-12 synthesis.

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