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- ☑ 1: [Autoimmun Rev.](#) 2004 Jun;3(4):295-300.

Sarcoidosis succumbs to antibiotics--implications for autoimmune disease.

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From time to time there have been reports of autoimmune disease succumbing to tetracycline antibiotics, but many have assumed this was due to coincidence, or to some ill-defined 'anti-inflammatory property' of the tetracyclines. But now the inflammation of sarcoidosis has succumbed to antibiotics in two independent studies. This review examines the cell wall deficient (antibiotic resistant) bacteria which have been found in tissue from patients with sarcoidosis. It examines how such bacteria can infect the phagocytes of the immune system, and how they may therefore be responsible for not only sarcoid inflammation, but also for other autoimmune disease. Proof positive of a bacterial pathogenesis for Sarcoidosis includes not only the demonstrated ability of these studies to put the disease into remission, but also the severity of Jarisch-Herxheimer shock resulting from endotoxin release as the microbes are killed. Studies delineating the hormone responsible for phagocyte differentiation in the Th1 immune response, 1,25-dihydroxyvitamin D, are discussed, and its utility as a marker of Th1 immune inflammation is reviewed. Finally, data showing that the behavior of this hormone is also aberrant in rheumatoid arthritis, systemic lupus erythematosus, and Parkinson's, raise the possibility that these diseases may also have a CWD bacterial pathogenesis.

PMID: 15246025 [PubMed - indexed for MEDLINE]