naturalistic setting, comparing the momentary relationship between symptoms and cortisol levels. We chose a shorter assessment period in an attempt to minimize study intrusion into participants’ lives. Similarly, as we noted in our report, we assessed stress, pain, and fatigue symptoms using simple 10-point Likert scales to minimize disruption of participants’ daily activities. We believe that this choice likely enhances the generalizability of our findings to “real world” patients. However, as the authors note, there are also trade-offs (e.g., lack of standardized stressor) with the use of this methodology.

Samuel A. McLean, MD
David A. Williams, PhD
Richard E. Harris, PhD
Richard H. Gracely, PhD
Michael E. Geisser, PhD
Ananda Sen, PhD
Daniel J. Clauw, MD
University of Michigan Medical Center
Ann Arbor, MI

Leslie J. Crofford, MD
University of Kentucky College of Medicine
Lexington, KY

William J. Kop, PhD
Uniformed Services University of the Health Sciences
Bethesda, MD


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Further support for the statins as antiinflammatory and immunomodulatory agents: comment on the review by Abeles and Pillinger and the editorial by Arnaud and Mach

To the Editor:
I very much enjoyed the review of statins as antiinflammatory and immunomodulatory agents by Drs. Abeles and Pillinger (1), as well as the accompanying editorial by Drs. Arnaud and Mach (2). These investigators have presented compelling evidence to support further investigation of this group of agents in treating rheumatologic disorders.

Although the review was clearly very comprehensive, I would like to add existent data regarding the observed effect of statin administration to mevalonate kinase–deficient patients with the hyperimmunoglobulin D syndrome, as provided by Simon and colleagues (3). Those investigators reported that 5 of 6 patients treated with simvastatin had a decrease in the number of days during which they were febrile and a decrease in the urinary mevalonic acid concentration.

Unfortunately, Simon et al did not measure cytokines in their experiment. It has, however, been demonstrated that peripheral blood mononuclear cells from patients with the hyperimmunoglobulin D syndrome release increased levels of interleukin-1β (IL-1β), IL-6, and tumor necrosis factor α compared with patients with familial Mediterranean fever and normal controls (4). This lends further encouragement and support for continued investigation of the utility of hydroxymethylglutaryl-coenzyme A inhibitors in treating inflammatory rheumatologic diseases.

Kenneth N. Schikler, MD
University of Louisville School of Medicine
Louisville, KY

Tonella infection has been associated with sudden cardiac death in Swedish orienteers with extensive arthropod exposure (11).

Transmission of B. henselae from ticks to humans has been postulated in conjunction with B. burgdorferi. A recent study using a highly specific but relatively insensitive polymerase chain reaction assay showed that B. henselae coinfection occurred in 22 of 86 patients (26%) with Lyme disease (2). As with other tick-borne coinfections such as babesiosis, ehrlichiosis, and anaplasmosis, coinfection with B. henselae and B. burgdorferi may lead to more severe symptoms of Lyme disease, including debilitating arthropathy and neuropsychiatric symptoms (2,3,12). Elimination of B. henselae infection may be difficult in both animals and humans, and treatment of coinfection in chronic Lyme disease may require prolonged antibiotic therapy (12–14).

From a pathophysiologic standpoint, Bartonella species are unique among bacteria in their ability to induce angioproliferative lesions that result in cutaneous and enteric vasculopathy (15). Of interest, B. henselae utilizes amino acid catabolism rather than glycolysis to derive energy from its host, generating ammonia in the process (16). Ammonia production may contribute to the encephalopathy reported in patients with chronic B. henselae infection, and it may also play a role in arthropathy, as noted with other arthritogenic bacteria (17). It is unclear whether vasculopathy or ammoniagenesis is involved in the joint inflammation described by Giladi et al.

Because transmission of B. henselae is not limited to cats, we propose that the term “Bartonella-associated arthropathy” should be used to identify this form of infection-related joint disease. The role of B. henselae as an emerging tick-borne coinfection in chronic Lyme arthropathy merits further study.

Raphael B. Stricker, MD
Joseph H. Brewer, MD
Joseph J. Burrascano, MD
Richard Horowitz, MD
Lorraine Johnson, JD, MBA
Steven E. Phillips, MD
Virginia R. Savely, FNP-C
Virgina T. Sherr, MD
International Lyme and Associated Diseases Society
Bethesda, MD