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.

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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.

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Possible role of tick-borne infection in "cat-scratch disease": comment on the article by Giladi et al

To the Editor:

In the report of their excellent study of "cat-scratch disease"–associated arthropathy, Giladi et al provide a comprehensive review of chronic arthropathy caused by infection with *Bartonella henselae* (1). The authors state that this "often severe" arthropathy "should be classified in the infection-related arthropathy group, potentially similar to Lyme disease arthritides." The similarity to Lyme disease may be more than coincidental.

Infection with *B henselae* has been recognized as an emerging tick-borne disease (2–9). The organism has been detected in questing *Ixodes* ticks in North America, Europe, and Asia (4–9), and in some areas the prevalence of *B henselae* in ticks is reportedly higher than the prevalence of *Borrelia burgdorferi,* the spirochetal agent of Lyme disease (4). *Peromyscus leucopus,* the white-footed mouse, serves as a reservoir for both *B burgdorferi* and *B henselae* (10). The fact that 72% of "cat-scratch disease" cases in the US occur in a seasonal pattern between June and December supports the notion of arthropod transmission of *B henselae,* and human infection via bites from flies, fleas, and mites may also occur (2,9). *Bar-*

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.

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