

Letter to the Editor

Chronic infection in 'post-Lyme borreliosis syndrome'

From STEVEN E PHILLIPS, JOSEPH J BURRASCANO, NICK S HARRIS, LORRAINE JOHNSON, PATRICIA V SMITH and RAPHAEL B STRICKER*

Cairns and Godwin provide strong evidence that patients with Lyme borreliosis may have persistent fatigue, musculoskeletal pain, and neurocognitive difficulties despite 'adequate' antibiotic therapy.¹ The authors state that 'ongoing infection has not been excluded' in these patients with 'post-Lyme borreliosis syndrome'. Based on the evidence, we postulate that ongoing infection is the most likely explanation for chronic Lyme disease symptoms.²⁻⁶

Recent molecular, biochemical, and immunological studies of *Borrelia burgdorferi*, the causative agent of Lyme borreliosis, have demonstrated the complexity and elusiveness of this tick-borne spirochete.^{3,6,7} The Lyme spirochete possesses functional properties that are found in other agents of chronic infection, such as *Mycobacteria*, *Brucella*, and *Treponema* species.⁷ Thus it is highly likely that *B. burgdorferi* would evade both the human immune response and perfunctory antibiotic therapy to produce chronic infection in certain patients, especially those who initially go untreated owing to lack of recognition of the tick-borne disease or those who are coinfecting with other tick-borne agents such as *Babesia*, *Anaplasma*, *Ehrlichia*, and *Bartonella* species.^{3,6} In fact, the medical literature contains numerous examples of persistent human infection with *B. burgdorferi*.^{3,6}

What is the evidence for 'post-Lyme borreliosis syndrome', defined as the persistence of symptoms in the absence of chronic infection with *B. burgdorferi*? Cairns and Godwin cite a study that found negative PCR testing in blood samples from 1800 patients with chronic Lyme disease. This study has been criticized for the lack of sensitivity of its non-nested PCR testing because it is highly unlikely that not a single patient in this Lyme disease cohort would have a positive PCR test.^{3,5,6} Moreover, it is widely recognized that when minimal numbers of organisms are present in the blood, a negative blood PCR test does not exclude the presence of infection because rigorous tissue sampling may yield positive results.^{8,9} For example, a necropsy study in dogs using PCR analysis of 25 tissue samples per dog demonstrated persistent infection after treatment.⁹ Thus the argument that negative blood PCR testing excludes persistent infection is erroneous.

Cairns and Godwin also cite the hypothesis that infection with *B. burgdorferi* may trigger some autoreactive inflammatory processes leading to persistent symptomatology. Despite the attractiveness of this hypothesis, there is no convincing evidence to support it, and attempts to identify a candidate autoantigen have consistently failed.^{3,6,10} The studies that have shown persistent inflammation in animal models of chronic Lyme

disease have not excluded ongoing infection, and persistent infection with *B. burgdorferi* has been demonstrated in mice, dogs, and chimps with experimental Lyme disease.^{3,6} Thus we are left with the strong assumption that chronic Lyme disease is caused by chronic infection with the Lyme spirochete.

As long as the medical community perceives chronic Lyme disease as an untreatable process that will somehow disappear with faith and prayer, patients with the debilitating symptoms of this disease will continue to suffer. Conversely, if the persistent symptoms described so elegantly by Cairns and Godwin are recognized as markers of chronic infection, then treatment of patients with chronic Lyme disease will become a logical approach, and the suffering of patients with chronic Lyme disease symptoms will be alleviated.

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International Lyme and Associated Diseases Society, PO Box 341461, Bethesda, MD 20827-1461, USA.

* Corresponding author. California Pacific Medical Center, 450 Sutter Street, Suite 1504, San Francisco, CA 94108, USA. E-mail: rstricker@usmamed.com