Bobbi Pritt and colleagues reveal that *Borrelia mayonii* is a new member of the *Borrelia burgdorferi* sensu lato complex, and show that this spirochete is pathogenic to human beings. However, the authors err on several key points. They state that *Borrelia burgdorferi* sensu stricto was previously the only cause of Lyme borreliosis in the USA. With the discovery of *B mayonii*, they take credit for describing the first non-*B burgdorferi* sensu stricto in patients in the USA; this assumption is not true. Girard and colleagues reported a *B bissetti* strain in a Florida patient. In all of these cases, patients had clinical symptoms commonly associated with Lyme borreliosis.

In their Introduction, Pritt and colleagues state that *B garinii, B afzelii,* and *B burgdorferi* sensu stricto are the only *B burgdorferi* sensu lato genospecies that cause Lyme borreliosis in Europe. In fact, at least five genospecies of the *B burgdorferi* sensu lato complex are pathogenic to human beings, including *B afzelii, B bavariensis, B burgdorferi* sensu stricto, *B garinii,* and *B spielmanii.* Additionally, *B bissetti, B kurtzenbachii, B lusitaniae,* and *B valaisiana* have been reported in patients in Europe.

The authors state that *B burgdorferi* sensu lato has never been reported in peripheral blood, but actually several researchers in the USA and Europe have isolated spirochetes from blood.

Biogeographically, the unique strain of *B burgdorferi* sensu lato (W97F51) from southeastern Wisconsin was completely overlooked. This novel strain was reported before *B mayonii,* and should have been addressed in both the text and the phylogenetic analyses (figure 4 in the Article).

Furthermore, the authors contend that Wisconsin is the epicentre of *B mayonii,* however, migratory songbirds play a vital part in the epidemiology of Lyme disease vector ticks, including the blacklegged tick (*Ixodes scapularis).* Since passerine migrants transport *I. scapularis* immatures thousands of kilometres during bidirectional, spring and fall migration, these avian hosts will, undoubtedly, disperse *B mayonii* spirochetes much further than currently recognised.

The authors claim that *B mayonii* causes symptoms that distinguish it from other *B burgdorferi* sensu lato genospecies. With only six patients, however, it seems audacious to state that *B mayonii* clinical symptoms are distinct from patients infected with *B burgdorferi* sensu stricto. The authors laud the US Food and Drug Administration-approved Lyme disease test kits, but some patients were seronegative. Based on the recommended two-tiered algorithm (table 2 in the Article), patient 6 was entirely negative and the other patients had inconsistent serological results. Finally, the authors recommend standard, short-term treatment for *B mayonii,* but fail to mention the limitations of this treatment for persistent Lyme disease.

I declare no competing interests.

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5 Golovchenko M, Vancová M, Clark K, Oliver JH Jr, Grubhoffer L, Rudenko N. A divergent spirochete strain isolated from a resident of the southeastern United States was identified by multilocus sequence typing as *Borrelia bissettii.* Parasit Vectors 2016; 9: 68.


**Authors’ reply**

We thank John Scott for his comments regarding our recent Article describing the identification of a novel genospecies of *Borrelia burgdorferi* sensu lato (*Borrelia mayonii*) as a new cause of Lyme borreliosis among patients in the upper midwestern USA. We agree with Scott that multiple *B burgdorferi* sensu lato genospecies can infect human beings. Our wording “nearly all human infections are caused by three *B burgdorferi* sensu lato genospecies” reflects this, and does not exclude other *B burgdorferi* sensu lato genospecies. A more thorough review of *B burgdorferi* sensu lato genospecies was neither within the focus of our Article nor possible due to the word limit. We concur that studies on other *B burgdorferi* sensu lato genospecies as a cause of Lyme borreliosis in North America and Europe are important.
Steroid use and clinical sequelae in two survivors of EVD

John Mattia and colleagues described the clinical sequelae during early Ebola virus disease (EVD) convalescence in a cohort of 277 survivors in Sierra Leone: arthralgias (76%), new ocular symptoms (60%), and uveitis (18%) were the more common presentations. Ebola viral load at the time of EVD diagnosis was the independent predictor of ocular symptoms, and specifically uveitis.

In Rome, Italy, we successfully treated two Italian patients with EVD who had a high viral load at the time of diagnosis (5 × 10⁴ copies per μL and 5 × 10⁷ copies per μL, respectively). Both patients received intravenous steroids at high doses; in the first case, to manage a severe adverse event to the infusion of an EVD convalescent plasma, and, in the second case, to treat a thrombocytopenic febrile syndrome with diffuse lymphadenopathy and pericardial effusion. Both patients fully recovered at discharge and no clinical sequelae at months 13 and 7 of follow-up were reported, respectively.

Use of corticosteroids in acute severe infectious diseases has been well documented. Tuberculosis and cryptococcal meningitis, immune reconstitution inflammatory syndromes, and severe community-acquired pneumonia are all diseases with evidence that favours the use of steroids. Additionally, experience with another virus with haemorrhagic expression, the Puumalavirus, documented an excellent outcome in a few patients with multiorgan failure treated with an aggressive approach that included the use of steroids.

As suggested by Hunt and Knott, knowledge of a likely exposure to steroids during the EVD acute phase in the Sierra Leone cohort could be of great interest. Apart from anecdotal cases, only a systematic collection of epidemiological and clinical data from large EVD patient cohorts could provide unequivocal evidence on the impact of steroid use on the occurrence of clinical sequelae after Ebola virus infection.

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