
Informed consent was obtained from all participants in the study. Institutional review boards at the University of Groningen Medical Center approved the study.

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Detection of Attenuated, Noninfectious Spirochetes in Borrelia burgdorferi-Infected Mice, after Antibiotic Treatment

To the Editor—The interesting and carefully executed study by Bockenstedt et al. [1] provides additional corroboration for the persistence of borrelial organisms and their DNA, in mammalian hosts, after application of parenteral antibiotics, such as ceftriaxone. However, the authors’ proposal that the animals will fully resolve the residua of their infections and will self-cure their infections, after the initial application of antibiotics, may not be justified by the study findings. That the authors were unable to xenotransfer the infection to other animals or to culture the infection from tissues does not necessarily mean that the host animal had dispatched or would successfully dispatch the infection, nor does it mean that the animal would not suffer injury as a result of borrelial persistence in protected niches, such as in intracellular sites and/or in the central nervous system (CNS). Considering that encephalopathy is a major manifestation of morbidity in patients with human Lyme disease [2], it would be instructive to assess cognitive function in mice—such as by assessment of their performance in a multiple-T maze—at various intervals after the initial antibiotic treatment, in comparable groups of mice either treated further or untreated with antibiotics. It would be important to use a strain of mouse known to be susceptible to CNS infection by borreliae and to choose a neurotropic strain of the Lyme organism. Fundamental questions of pathogenesis and appropriate management of borrelial infection could be addressed by such a study.

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References

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Reply

To the Editor—Although the mouse model of Lyme borreliosis has been a valuable tool for the study of many aspects of human Lyme disease, it is not a model of neuroborreliosis. Borrelia burgdorferi does not persistently colonize the central nervous system in mice, and nervous system pathology is not a consequence of B. burgdorferi infection in this animal model [1]. Moreover, to our knowledge, “neurotropic” strains of B. burgdorferi have not been identified. Thus, the experiments proposed by Dr. Liegner [2] to evaluate cognitive function in mice after antibiotic treatment for Lyme borreliosis would have no meaning.

In our recently published study [3], we emphasize that, in mice treated with antibiotics, the presence of spirochete DNA should not be equated with persistent infection. Viable spirochetes were not found immediately before or at the time that mice were killed, despite the use of multiple methods to detect them, including xenodiagnosis after immunosuppression of mice. In mice treated with antibiotics, spirochete DNA in tissues was not associated with histopathologic evidence of disease, as it was with control infected mice. Taken together, our data support the interpretation that infectious spirochetes are effectively eliminated by antibiotic therapy in the mouse model of Lyme borreliosis.

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