

STUDY SUPPORTS CHRONIC LYME

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STUDY SUPPORTS CHRONIC LYME

Protective Niche for *Borrelia burgdorferi* to Evade Humoral Immunity

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The Lyme disease spirochete, *Borrelia burgdorferi*, is an extracellular microbe that causes persistent infection despite the development of strong immune responses against the bacterium. *B. burgdorferi* expresses several ligand-binding lipoproteins, including the decorin-binding proteins (Dbps) A and B, which may mediate attachment to decorin, a major component of the host extracellular matrix during murine infection. We show that *B. burgdorferi* was better protected in the joints and skin, two tissues with a higher decorin expression, than in the urinary bladder and heart, two tissues with a lower decorin expression, during chronic infection of wild-type mice.

Targeted disruption of decorin alone completely abolished the protective niche in chronically infected decorin-deficient mice but did not affect the spirochete burden during early infection. The nature of protection appeared to be specific because the spirochetes with higher outer surface protein C expression were not protected while the protective niche seemed to favor the spirochetes with a higher dbpA expression during chronic infection.

These data suggest that spirochetal DbpA may interact with host decorin during infection and such interactions could be a mechanism

that *B. burgdorferi* uses to evade humoral immunity and establish chronic infection.

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Yale University; *Borrelia burgdorferi* changes antigens based on host immune response 08 December 2004 Biotech Week Copyright 2004, Biotech Week via NewsRx.com (NewsRx.com & NewsRx.net)

— *Borrelia burgdorferi* changes its surface antigenic expression in response to host immune responses. The Lyme disease spirochete, *Borrelia burgdorferi*, causes persistent mammalian infection despite the development of vigorous immune responses against the pathogen. To examine spirochetal phenotypes that dominate in the hostile immune environment, the mRNA transcripts of four prototypic surface lipoproteins, decorin-binding protein A (DbpA), outer surface protein C (OspC), BBF01, and VlsE, were analyzed by quantitative reverse transcription-PCR under various immune conditions. We demonstrate that *B. burgdorferi* changes its surface antigenic expression in response to immune attack," investigators in the United States report. TD "dbpA expression was unchanged while the spirochetes decreased ospC expression by 446 times and increased BBF01 and vlsE expression up to 20 and 32 times, respectively, under the influence of immune pressure generated in immunocompetent mice during infection," stated Fang Ting Liang and collaborators at Yale University, Centocor, Inc., and the Centers for Disease Control and Prevention. "This change in antigenic expression could be induced by passively immunizing infected severe combined immunodeficiency mice with specific *Borrelia* antisera or OspC antibody and appears to allow *B. burgdorferi* to resist immune attack." Liang and associates published their study in *Infection and Immunity* (*Borrelia burgdorferi* changes its surface antigenic expression in response to host immune responses).

*Infec Immunity*, 2004;72(10):5759–5767). For additional information, contact Erol Fikrig, Section of Rheumatology, Department of Internal Medicine, Yale University School of Medicine, S525A, 300 Cedar Street, New Haven, CT 06520–8031, USA.

E-mail: erol.fikrig@y.... The publisher of the journal *Infection and Immunity* can be contacted at: American Society for Microbiology, 1752 N Street NW, Washington, DC 20036–2904, USA. The information in this article comes under the major subject areas of Lyme Disease, Tick-Borne Disease, Zoonosis, Lyme Disease Vaccine, Vaccine Development, Proteomics, Immunotherapy, and Immunology. This article was prepared by Biotech Week editors from staff and other reports. Copyright 2004, Biotech Week via NewsRx.com & NewsRx.net.

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Lyme Disease Receptor Identified in Tick Guts

[November 2004] Researchers at Yale School of Medicine have identified a Lyme disease receptor called TROSPA that is used by disease agents to invade ticks.

Lyme disease, the most common tick-borne disease in the United States, is caused by spirochete bacteria *Borrelia burgdorferi*, which also cause arthritis in humans. The purpose of the study, published November 12 in the journal *Cell*, was to identify how Lyme disease pathogens survive inside ticks.

"We identified a receptor inside the tick gut that the spirochete bacteria use to colonize or invade ticks," said principal investigator Erol Fikrig, M.D., professor of internal medicine/rheumatology and in the Section of Microbial Pathogenesis, and Department of Epidemiology and Public Health at Yale School of Medicine.

"When we eliminated or blocked the receptor in the ticks, they were no longer able to carry the Lyme disease agent *Borellia burgforferi*."

"This opens up potential new avenues to disrupt the *Borellia*'s life cycle and offers strategies for improving diagnosis and treatment of Lyme disease," Fikrig added.

To characterize the Lyme disease receptor, the team cloned the gene for the receptor from ticks. After they expressed the purified receptor gene, they showed that the Lyme disease agent *Borellia burgforferi* binds to the receptor. "When we blocked the receptors with antibodies or when we used RNA interference to knock the receptor out of the ticks, they no longer carried *Borellia burgforferi*," said Fikrig.

"We are excited to learn more about the life cycle of this important pathogen," Fikrig added. "This information can also be used to study other vector-borne diseases such as West Nile virus and Malaria," Fikrig added.

Other authors on the study included Utpal Pal, Xin Li, Tian Wang, Ruth R. Montgomery, Nandhini Ramamoorthi, Aravinda M. deSilva, Fukai Bao, Xiaofeng Yang, Marc Pypaert, Deepti Pradhan, Fred S. Kantor, Sam Telford and John F. Anderson.

Citation: *Cell*, No. 19 Volume 4, November 12, 2004

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