Study provides greater understanding of lyme disease-causing bacteria

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Lyme disease in the U.S. is caused by the tick-borne bacteria *Borrelia burgdorferi* and usually begins with a skin lesion, after which the bacteria spread throughout the body to the nervous system, heart or joints. About 60 percent of untreated individuals develop arthritis, which affects the knees in particular. Lyme disease usually responds well to antibiotic therapy, but in rare cases arthritis can persist for months or years after treatment, a rare condition known as antibiotic-refractory Lyme arthritis. Joint fluid usually tests negative for B burgdorferi after treatment, indicating that joint inflammation may persist even after the bacteria has been eradicated.

Two genetic marker systems are used to correlate the variation of this bacterial strain with clinical outcomes: OspC typing divides *B burgdorferi* strains into 21 types, while the ribosomal RNA intergenic spacer type (RST) system divides them into just three groups, with certain RST groups corresponding uniquely to specific OspC types.

A new study led by Allen Steere of Massachusetts General Hospital and Harvard Medical School analyzed joint fluid samples from 124 patients with Lyme arthritis who were seen over a 30-year period. It identified *B. burgdorferi* strains in the joints of patients with Lyme arthritis and found that the genotype frequencies in joints reflected those in skin lesions. However, RST1 strains were the most frequent in patients with antibiotic-refractory arthritis. The study was published in the July issue of *Arthritis & Rheumatism* (http://www3.interscience.wiley.com/journal/76509746/home).

The researchers were able to identify 10 of the 16 B burgdorferi OspC types found in the northeastern U.S. and all three RST types in the joint fluid of patients with Lyme arthritis. Although it was only possible to determine *B burgdorferi* phenotypes in 40 percent of the samples, the researchers feel confident that the distribution reflects what has been observed in the skin because they were able to identify numerous OspC and RST types, and the distribution was similar to what has been reported in previous studies of skin lesions.

One might presume that the association of RST1 strains with antibiotic-refractory arthritis may reflect a greater ability of these strains to survive in the joint despite antibiotic therapy. However, this seems not to be the case. Rather, RST1 strains seem to induce a more marked immune response, which may set the stage for joint inflammation that persists after antibiotic therapy in genetically susceptible individuals.

"We hypothesize that RST1 strains are more virulent, leading to larger numbers of organisms in blood, and more inflammation in joints," the authors state. They conclude that the results of this study "add to the emerging literature concerning the differential pathogenicity of strains of *B burgdorferi*."

More information: "Analysis of Borrelia burgdorferi Genotypes in Patients with Lyme Arthritis," Kathryn L. Jones, Gail A. McHugh, Lisa J. Glickstein, Allen C. Steere, *Arthritis & Rheumatism*, July 2009.

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