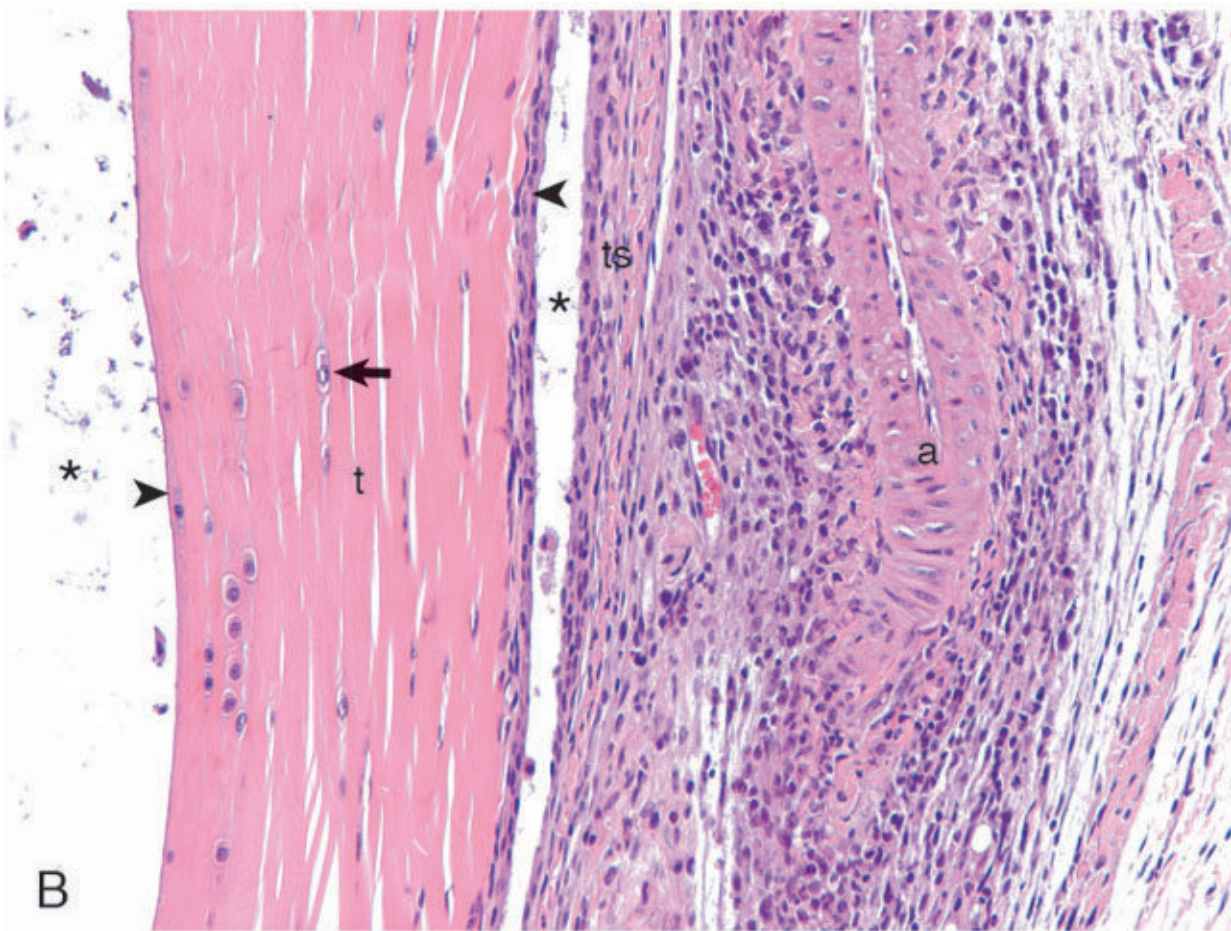


Cascading inflammation associated with Lyme arthritis linked to overactive immune response

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Analysis of joint tissue reveals reactive and thickened synovium covering the tendon sheath. Credit: Sarah Whiteside

Every year, more than 300,000 Americans contract Lyme disease, an infection caused by *Borrelia burgdorferi*, a bacterium transferred during a tick bite. In a small percentage of patients, infection symptoms, including arthritis, persist despite antibiotic treatment.

Scientists at University of Utah Health believe they identified a mechanism that activates T cells, a key component of the immune system, which could explain the elusive link between a tick bite and persistent Lyme arthritis. The results are published online in the February 5th issue of the *Journal of Immunology*.

"We believe that in this model persistent Lyme arthritis is a result of [overactive] immune response," said the study's first author Sarah Whiteside, a graduate student in Janis Weis's lab at U of U Health.

The researchers identified a receptor on T cells that interacts with molecules on the surface of *B. burgdorferi*. Like a key fitting into a lock, the receptors join in a process resulting in bystander activation. This activation mechanism triggers the T cell to produce inflammatory molecules that accumulate around the joints and contribute to inflammation and arthritis.

Some of the newly 'turned-on' T cells can interact with residual bacteria that persists long after the initial tick bite, producing a cascading cycle of inflammation that could lead to infection-induced autoimmunity.

"Through bystander activation, a whole repertoire of T cells may be activated, independent of their specificity for infecting pathogens," said Weis, Ph.D., professor of Pathology at U of U Health.

Whiteside cautions the exact mechanism of T cell activation needs clarification, but the results from this study suggest new therapeutic approaches, such as focusing on anti-inflammatory mechanisms, might

be more effective for patients with persistent Lyme arthritis.

"If you can suppress T cell activation for the short-term, we might help re-establish the control mechanism [for the [immune response](#)] in the body," said Weis.

In addition to new therapies, future studies may focus on the broader implications of bystander activation for other pathogen-induced and autoimmune diseases.

These inquiries will benefit from the mouse model developed for this study by Whiteside. The mice, which lack an anti-inflammatory molecule (IL-10), mimic the symptoms of sustained arthritis in human patients. In the absence of the anti-inflammatory molecule, the mice reveal the disease-causing potential of T cells during infection.

Mice in the study were followed for 18 weeks following infection. Within two weeks, the concentration of T cells and inflammation markers in the joint fluid of the mice increased, despite the extremely low or often undetectable amounts of *B. burgdorferi* in the joint tissue. Microscopic examination of [joint tissue](#) of mice in the study revealed thickened tissue covering the tendon sheath, resulting from infiltration of [cells](#) producing inflammatory molecules. Neutralization of the inflammatory [molecules](#) resulted in reduced ankle swelling and arthritis severity in the [mice](#).

"Growing up in Vermont, I know several people with Lyme disease," said Whiteside. "It is rewarding to develop a model that mimics [arthritis](#) so we can study what is happening in these patients."

Most patients diagnosed with a Lyme infection receive a 2- to 3-week course of antibiotics and recover without long-term complications.

Provided by University of Utah

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