

# OX40-positive follicular helper T cells control rheumatoid arthritis

31 July 2019



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Rheumatoid arthritis is a progressive autoimmune disease most frequently characterized by pain, swelling, and stiffness in joints. Increased numbers of follicular helper T cells have been observed in patients with rheumatoid arthritis, but the functions of these cells have been unclear.

In a new study published in *Annals of the Rheumatic Diseases*, a research team led by experts from the University of Tsukuba investigated the role of follicular helper T cells, a type of immune cell that guides the adaptive immune response, in the onset of [rheumatoid arthritis](#). They found that OX40-expressing follicular helper T cells contributed to inflammation by regulating a chemical modification of autoantibodies known as sialylation.

Rheumatoid arthritis is a chronic, systemic autoimmune [disease](#) that causes pain, reduced quality of life, and increased mortality in affected patients. Although the symptoms and [risk factors](#) for the disease are well-known, the underlying mechanisms that lead to initial onset of disease are unknown, and current effective treatments place

patients at risk of developing infections.

"Sialylation of autoantibodies has been shown to contribute to the onset of experimental arthritis in mice, through a subset of T helper cells," says Isao Matsumoto, corresponding author on the study. "Because OX40-expressing (IL-17-producing) follicular helper T cells are present at increased proportions in patients with rheumatoid arthritis, we suspected that these cells may play a role in sialylation that promotes the development of rheumatoid arthritis."

In the study, increased numbers of OX40-expressing (IL-17-producing) follicular helper T cells were found during the onset of experimental arthritis, accompanied by autoantibodies with an increased ability to activate immune cells in vitro. Analysis of the autoantibodies revealed that changes in sialylation caused this increased inflammatory potential, suggesting that the process of sialylation may provide a useful drug target for treatment of rheumatoid arthritis.

"Our analyses revealed that the interaction between OX40 on the surface of follicular helper T cells and OX40L on antibody-producing B cells led to reduction of sialylation on autoantibodies in mice," says lead author Izumi Kurata. "We found that blocking this interaction could reduce autoimmune inflammation while preserving immune interactions necessary for controlling potential infections."

In addition to blocking the interaction between cells that could lead to increased inflammation via autoantibodies, the researchers found that this blocking strategy could reduce the numbers of follicular helper T [cells](#) in mice, which may contribute to extended resolution of [arthritis](#) symptoms.

**More information:** Izumi Kurata et al. Potential involvement of OX40 in the regulation of autoantibody sialylation in arthritis, *Annals of the*

Rheumatic Diseases (2019). DOI:  
[10.1136/annrheumdis-2019-215195](https://doi.org/10.1136/annrheumdis-2019-215195)

Provided by University of Tsukuba  
APA citation: OX40-positive follicular helper T cells control rheumatoid arthritis (2019, July 31) retrieved  
1 May 2021 from  
<https://medicalxpress.com/news/2019-07-ox40-positive-follicular-helper-cells-rheumatoid.html>

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