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Protein's new role discovered in autoimmune disease

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Investigators at the University of Alabama at Birmingham (UAB) have identified the previously unknown role of a chemical 'messenger' leading to autoimmune disorders like rheumatoid arthritis and lupus.

The messenger is the naturally occurring chemical interleukin 17 (IL-17), an immunity protein.

UAB researchers pinpointed an unknown role IL-17 plays in autoimmune and inflammatory responses, aside from its commonly known effects within immunity. Future research will home in on IL-17's unwanted actions and preserve its benefits within the immune system.

The new findings are published in the journal *Nature Immunology*.

In the study, UAB scientists blocked messenger signals from the IL-17 protein to the immune system of mice. This disruption significantly reduced the number of white blood cells, namely disease-causing B cells, clustered in the mice's spleen.

The number of B-cell clusters dropped from 17 percent to 2 percent when the IL-17 protein signals were blocked, the study authors said.

The drop was a clear sign that IL-17 plays a major role on shaping B cells' ability to create more and more disease-causing antibodies.

"The effect of IL-17 to slow down B cells, thereby enhancing their

interaction with other immune regulatory cells a new and exciting discovery," said John D. Mountz, M.D., Ph.D., UAB professor of medicine and senior author on the study.

"This is surprising since previously IL-17 was thought to increase, but not decrease, cell motion. Now the effects of IL-17 on B cells can be explored more fully," Mountz said.

Many types of B cells make up the human immune system, which is regulated to sense and fight infection without attacking normal, healthy tissue. In autoimmune diseases that regulatory process becomes imbalanced.

"Knowing more about IL-17's ability to regulate unwanted B-cell migration will generate new ideas in the ongoing search for better drug targets in preventing and treating autoimmune disease," said Hui-Chen Hsu, Ph.D. an assistant professor in the UAB Division of Clinical Immunology and Rheumatology and lead author on the study.

Source: University of Alabama at Birmingham