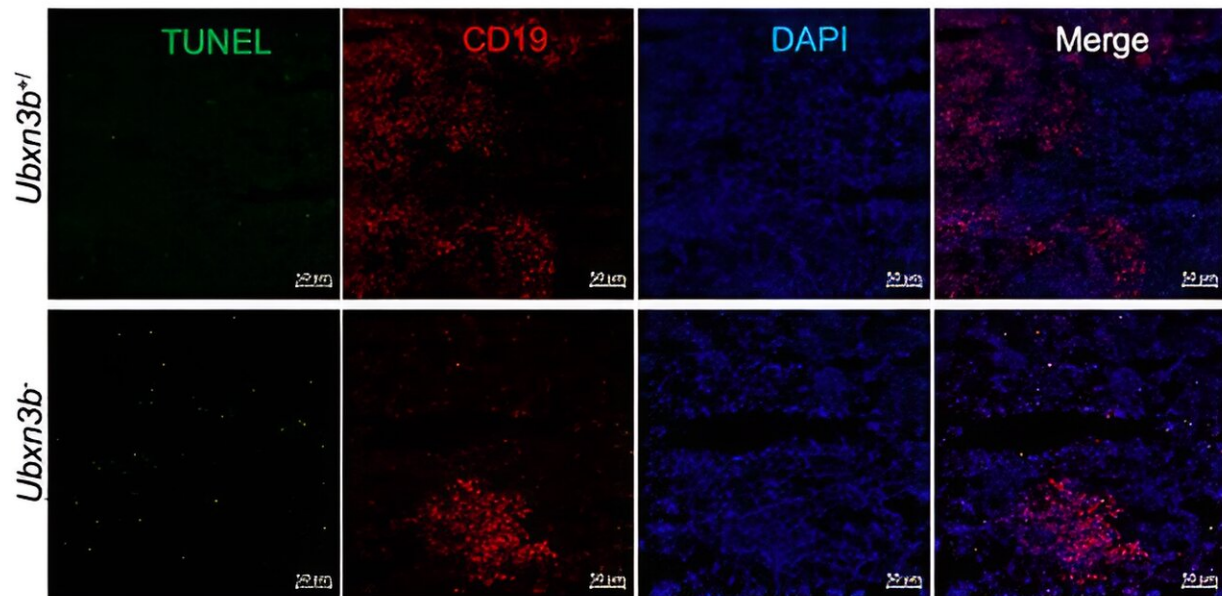


# Researchers find more effective way to deplete B-cells for the treatment of autoimmune disorders

August 14 2024, by Kim Krieger



UBXN3B is crucial for mature B cell survival. Immunofluorescence staining for apoptotic (TUNEL) and B cells (CD19) in the spleens of mice at 14 days after the first TMX/oil treatment. Credit: *eBioMedicine* (2024). DOI: 10.1016/j.ebiom.2024.105248

B-cells protect your body from infection. But sometimes, they misfire and cause debilitating disease. Now, University of Connecticut researchers have shown that a single protein might be able to defuse B-

cells gone bad. Their findings are [published](#) in the journal *eBioMedicine*.

B-cells are immune cells that make antibodies, the proteins that grab onto foreign invaders and mark them for destruction. B-cells are born in the [bone marrow](#) and then spread throughout the body, gaining experience against viruses, bacteria and other bad actors as they go. When a piece of an invader is presented to a B-cell, the B-cell copies it and then manufactures antibodies to combat it.

But sometimes B-cells get confused, and begin making antibodies against friendly cells in the body instead. This can cause [autoimmune diseases](#) such as multiple sclerosis or lupus. B-cells can also be involved in cancers such as lymphoma.

Certain treatments for lymphoma, lupus, multiple sclerosis, and other B-cell diseases try to deplete the body of the B-cells doing the damage. For some people, these treatments can be very effective. But not for all people; sometimes the treatments worsen the [disease](#). And sometimes the treatments work for a little while, but when stopped, symptoms flare up again.

Penghua Wang, an immunologist at UConn School of Medicine, was looking at something completely different when he stumbled upon a much more effective way to deplete B-cells. Wang and his colleagues Tingting Gang and Duomeng Yang, both immunology researchers at the School of Medicine, were investigating a protein called Ubxn3b and its role in COVID infection.

Mice missing the gene for Ubxn3b were known to be highly vulnerable to respiratory viruses. They found that Ubxn3b was important for viral immunity in mice because it encouraged the development of B-cells. Mice that lacked Ubxn3b lacked B-cells almost entirely.

Wang and his colleagues tried raising mice that developed a normal immune system in infancy, and then blocked Ubxn3b when the mice became adults. The mice stopped producing B-cells in their bone marrow, and existing B-cells in the spleen, [lymph nodes](#), and other peripheral areas of the body died without the protein. The mice lacking Ubxn3b were normal and fairly healthy otherwise.

"The rest of the immune system looked fine," in these Ubxn3b knockout mice, Wang says. "This gene function in cell survival is very specific to B-cells."

The researchers are now looking to find partners who study multiple sclerosis animal models, or other autoimmune disease models in animals, to test whether blocking Ubxn3b would truly be therapeutic. They also plan on developing a detailed molecular mechanism explaining how the gene regulates B-[cell survival](#).

**More information:** Tingting Geng et al, UB3B is crucial for B lymphopoiesis, *eBioMedicine* (2024). [DOI: 10.1016/j.ebiom.2024.105248](#)

Provided by University of Connecticut

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