

Secrets of immunologic memory

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Investigators at Sanford-Burnham Medical Research Institute (Sanford-Burnham) have discovered a new way the cell surface protein, CD44, helps specific T helper (Th1) cells develop immunologic memory. Linda Bradley, Ph.D., Bas Baaten, Ph.D., and colleagues determined that without CD44, Th1 cells died off during their initial immune response and were unable to generate immunologic memory.

This is the first time scientists have identified this unique CD44 function on Th1 cells, making the protein a potential target to treat a variety of diseases. The study was published online on January 14 in the journal *Immunity*.

CD44, a protein found on many cell types throughout the body, binds to the glycan hyaluronic acid (HA) in the extracellular matrix. When T helper cells are activated by infection, they upregulate (increase the activity of) CD44. Though CD44 is a marker for these "experienced" cells, its function has remained elusive. [T cells](#) are important components in the body's defense against diseases and, as memory cells, provide immunity to subsequent infections.

"In various infections and autoimmune conditions, Th1 cells are often the bad guys," said Dr. Bradley. "They can contribute to disease by overproducing [cytokines](#) and are often responsible for the disease pathology. Our findings reveal an opportunity to harness CD44 to control this pathogenesis."

The Bradley laboratory used T cells lacking CD44 that recognized a

protein fragment in the [influenza virus](#). Noting that the T cells did not survive and were unable to generate immunologic memory, the laboratory determined that CD44 protected the cells from programmed cell death initiated by the Fas receptor. This protective effect was specific to a subset of T cells, the Th1 cells, and mediated by the PI 3 kinase pathway. The researchers also demonstrated that survival of the cells could be controlled by antibodies capable of modulating CD44 signaling.

Provided by Sanford-Burnham Medical Research Institute

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