

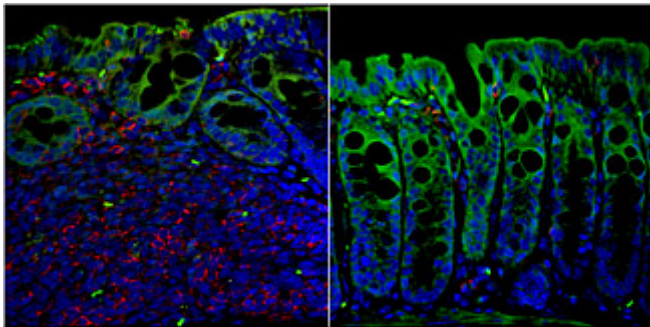
Scientists Discover Pain Receptor on T-Cells

October 05, 2014 |

Researchers at University of California, San Diego School of Medicine have discovered that T-cells – a type of white blood cell that learns to recognize and attack microbial pathogens – are activated by a pain receptor.

The study, reported online Oct. 5 in *Nature Immunology*, shows that the receptor helps regulate intestinal inflammation in mice and that its activity can be manipulated, offering a potential new target for treating certain autoimmune disorders, such as Crohn's disease and possibly multiple sclerosis.

“We have a new way to regulate T-cell activation and potentially better control immune-mediated diseases,” said senior author Eyal Raz, MD, professor of medicine.



An inflammatory response and damage to the intestinal wall (left) could be prevented by injecting TRPV1-deficient T-cells (right).
Credit: *Nature Immunology*, Bertin et al.

The receptor, called a TRPV1 channel, has a well-recognized role on nerve cells that help regulate body temperature and alert the brain to heat and pain. It is also sometimes called the capsaicin receptor because of its role in producing the sensation of heat from chili peppers.

The study is the first to show that these channels are also present on T-cells, where they are involved in gating the influx of calcium ions into cells – a process that is required for T-cell activation.

“Our study breaks current dogma in which certain ion channels called CRAC are the only players involved in calcium entry required for T-cell function,” said lead author Samuel Bertin, a postdoctoral researcher in the Raz laboratory. “Understanding the physical structures that enable calcium influx is critical to understanding the body’s immune response.”

T-cells are targeted by the HIV virus and their destruction is why people with AIDS have compromised immune function. Certain vaccines also exploit T-cells by harnessing their ability to recognize antigens and trigger the production of antibodies, conferring disease resistance. Allergies, in contrast, may occur when T-cells recognize harmless substances as pathogenic.

TRPV1 channels appear to offer a way to manipulate T-cell response as needed for health. Specifically, in in vitro experiments researchers showed that T-cell inflammatory response could be reduced by knocking down the gene that encodes for the protein that comprises the TRPV1 channel. Overexpression of this gene was shown to lead to a surge in T-cell activation, which in human health may contribute to autoimmune diseases. T-cells also responded to pharmaceutical agents that block or activate the TRPV1 channel.

In experiments with mice models, researchers were able to reduce colitis with a TRPV1-blocker, initially developed as a new painkiller. One of the promising discoveries is that colitis in mice could be treated with much lower doses than what is needed to dull pain. "This suggests we could potentially treat some autoimmune diseases with doses that would not affect people's protective pain response," Raz said.

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This study was funded, in part, by National Institutes of Health (U01 AI095623, P01 DK35108 and P30 NS047101), The Broad Foundation, Crohn's and Colitis Foundation of America, Canadian Institutes of Health Research, Fulbright Association, Philippe Foundation Inc. and Japan Society for the Promotion of Science.

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