MedicalXpress

Researchers turn off severe food allergies in mice

October 2 2010

Johns Hopkins scientists have discovered a way to turn off the immune system's allergic reaction to certain food proteins in mice, a discovery that could have implications for the millions of people who suffer severe reactions to foods, such as peanuts and milk.

The findings, published online in the journal *Nature Medicine,* provide hope that the body could be trained to tolerate food allergies that lead to roughly 300,000 emergency room visits and 100 to 200 deaths each year.

The research team, led by Shau-Ku Huang, Ph.D., a professor of medicine, and Yufeng Zhou, M.D., Ph.D., a postdoctoral fellow in the Division of Allergy and Clinical Immunology at Johns Hopkins University School of Medicine, discovered that one kind of immune cell in the gastrointestinal tract called lamina propria dendritic cells (LPDC) — considered the first line of defense for a body's immune system — expresses a special receptor, SIGNR1, which appears on the cells' surface and binds to specific sugars.

By targeting this receptor using sugar-modified protein, researchers were able to keep food proteins that would have induced a severe, even deadly, allergic reaction from causing any serious harm.

"There is no cure for food allergies, and the primary treatment is avoidance of the offending protein," Zhou says. "This could teach our bodies to create a new immune response and we would no longer be

allergic to the protein."

The researchers hope to confirm whether this promising process in mice can also occur in people.

Food allergies are triggered by the immune system and, in some people, can cause severe symptoms or even a life-threatening reaction known as anaphylaxis. In the United States, it is estimated that six to eight percent of children under the age of three and nearly four percent of adults have food allergies, and the prevalence is rising. Because of the extreme difficulty in avoiding all food allergen exposure and the lack of effective treatments, preventive and therapeutic strategies are urgently needed, Zhou says.

In the laboratory, Zhou and his colleagues took a food protein that causes allergies in mice and modified it by adding special sugars. They hypothesized that, when ingested by the mice, the modified proteins would be able to bind to what are known as the SIGNR1 receptors on the immune system cells. Bound in this way, the immune system would learn to tolerate the modified food protein — and the protein would no longer induce an allergic reaction, even when consumed in its unmodified form.

Zhou fed his mice the modified protein once a day for three days. Five days later, he tested them by feeding them the protein in its unmodified form. Another group of mice was not fed the modified protein at all. The severity of the allergic response to the unmodified protein — which in the control-group mice tended to be tremors, convulsions and/or death — was significantly decreased in those mice that had been pre-fed the modified protein. Some still had minor reactions like itchiness or puffiness around the eyes and snout, but none had serious ones. These mice appeared to be desensitized to the food protein, even when it was fed to them in its unmodified form, says Zhou. In this model, SIGNR1

plays a key role in shutting off some responses in the immune cells, but whether this is the only function of this receptor is, at present, unknown.

More information: www.nature.com/nm/journal/vaop … nt/full/nm.2201.html

Provided by Johns Hopkins Medical Institutions