# Researchers report antibody that prevents a broad range of allergic reactions

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Researchers have found a new mechanism in which an antibody can

prevent allergic reactions in a broad range of patients. Their discovery could pave the way for a far more effective allergy medicine. The team of researchers from the Departments of Engineering and Molecular Biology and Genetics together with German researchers from Marburg/Giessen has now described the molecular structure and mechanisms of action of the antibody in *Nature Communications*, and the results are surprising.

They were hoping to find new methods to improve existing treatment, but instead, they identified how a specific antibody completely inactivates the allergic processes. The antibody interacts in a complex biochemical process in vivo by which it prevents the human allergy antibody (IgE) from attaching to cells, thus keeping all allergic symptoms from occurring.

"We can now describe the interaction of this antibody with its target and the conformational changes very accurately. This allows us to understand how it interferes with the IgE and its specific receptors on the immune cells of the body, which are responsible for releasing histamine in an allergic reaction," says Edzard Spillner, associate professor at the Department of Engineering, Aarhus University.

## Allergic effects of birch pollen and insect venom eliminated

Generally, a person with allergies produces high levels of IgE molecules against external allergens when exposed to them. These molecules circulate in the blood and are loaded onto the effector cells of the immune system, which triggers the production of histamine and thereby an immediate allergic reaction in the body.

The function of the antibody is that it interferes with binding of IgE to

the two specific effectors (CD23 and FceRI) on the immune cells, thereby making it impossible for the allergy molecule to bind. Furthermore, the researchers have observed that the antibody also removes the IgE molecules even after binding to its receptors.

"Once the IgE on immune cells can be eliminated, it doesn't matter that the body produces millions of allergen-specific IgE molecules. When we can remove the trigger, the allergic reaction and symptoms will not occur," says Edzard Spillner.

In the laboratory, it took only 15 minutes to disrupt the interaction between the allergy molecules and the immune cells. The researchers conducted ex vivo experiments with blood cells from patients allergic to birch pollen and insect venom. However, the method can be transferred to virtually all other allergies and asthma.

Today, one in three Europeans suffer from allergic conditions, and the prevalence is steadily increasing. The treatment options are limited, but the researchers now expect that their scientific results will pave the way to developing completely new types of allergy medicine. "We can now precisely map how the antibody prevents binding of IgE to its receptors. This allows us to envision completely new strategies for engineering medicine of the future, "says Nick Laursen, assistant professor at the Department of Molecular Biology and Genetics.

The antibody is effective and considerably smaller than therapeutic antibodies currently used to produce allergy medicine.

"It is a so-called single domain antibody, which is easily produced in processes using only microorganisms. It is also extremely stable, which provides new opportunities for how the antibody can be administered to patients," says Edzard Spillner.

Unlike most therapeutic antibodies already available on the market, the new antibody does not necessarily have to be injected. Because of its chemical structure, it might be inhaled or swallowed, and these administration methods will make treatment easier, cheaper, and much and more comfortable for patients. However, before new allergy medicines can be produced, the researchers will have to conduct a wide range of clinical trials to document the effect and safety of the antibody.

**More information:** Frederic Jabs et al, Trapping IgE in a closed conformation by mimicking CD23 binding prevents and disrupts FcεRI interaction, *Nature Communications* (2017). DOI: 10.1038/s41467-017-02312-7

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