Do genes play a role in peanut allergies?
New study suggests yes
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Researchers have pinpointed a region in the human genome associated with peanut allergy in U.S. children, offering strong evidence that genes can play a role in the development of food allergies.

But in an additional finding that suggests genes are not the only players in food allergies, the Johns Hopkins Bloomberg School of Public Health-led research team found there may be other molecular mechanisms that may contribute to whether those who are genetically predisposed to peanut allergies actually develop them.

The findings are published online Feb. 24 in the journal *Nature Communications*.

"We always suspected it, but this is the first genome-wide association study (GWAS) that identified a genetic link to well-defined peanut allergy," says the study's principal investigator, Xiaobin Wang, MD, ScD, MPH, the Zanvyl Krieger Professor and Director of the Center on the Early Life Origins of Disease at the Johns Hopkins Bloomberg School of Public Health.

Food allergies have been rising rapidly in the U.S. and around the world over the past 20 years and now affect an estimated two to 10 percent of children in the United States. Food allergies have become a major clinical and public health problem due to their increasing prevalence, their potential to be life threatening and their enormous medical and economic impact. Peanut allergy is among the most fatal food allergies and is often a lifelong allergy, unlike milk or egg allergies which most children will grow out of.

In their study, Wang and her colleagues analyzed DNA samples from 2,759 participants (1,315 children and 1,444 of their biological parents) enrolled in the Chicago Food Allergy Study. Most of the children had some kind of food allergy. They scanned approximately 1 million genetic markers across the human genome, searching for clues to which genes might contribute to increased risk of developing food allergies, including peanut. They found that a genomic region harboring genes such as HLA-DB and HLA-DR and located on chromosome six is linked to peanut allergy. This study suggests that the HLA-DR and -DQ gene region probably poses significant genetic risk for peanut allergy as it accounted for about 20 percent of peanut allergy in the study population.

Not everyone with these mutations, however, develops peanut allergy, and researchers wondered why. One possible reason, they determined, was that epigenetic changes may also play a role. Epigenetic changes, in which a methyl group attaches itself to the DNA, alter the expression of a gene without altering its underlying code. The levels of DNA methylation regulate whether people with genetic susceptibility to the peanut allergy actually developed it.

While the study represents a "good first step," more research is needed. For example, a better understanding of genetic susceptibility will allow for early risk assessment and prediction of food allergies, perhaps as early as in utero, Wang says.

Unlike genes themselves, DNA methylation levels can change in response to environmental exposures, (in particular, in-utero and during the first few years of life) and the changes are potentially reversible. By identifying what environmental factors can alter DNA methylation...
levels in people with genes that make them susceptible to peanut allergy, researchers could potentially open a new avenue for prevention and treatment of peanut allergy.

"Hopefully, one day, we can manage or prevent food allergies in a safe, simple, effective way," Wang says. "We might be able to use pharmaceutical treatment, but if we can figure out whether a lifestyle, nutrition or environmental change could reduce allergies, that would be even better."

"One in 13 children in the U.S. have food allergies, and peanut is the most common food allergen among children," says James R. Baker, Jr., MD, CEO of FARE. "This important study adds critical insights that will help us understand the rise in the prevalence of peanut allergy, and we look forward to further exploration of these findings."

Provided by Johns Hopkins University Bloomberg School of Public Health


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