



## Diagnostic Measure for Defining Food Allergy using IgE Sensitization

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### DESCRIPTION

Environmental and genetic risk factors are likely to play a role in the etiology of food allergy, a complicated multifactorial disease. When exposed to specific dietary proteins, it causes an abnormal inflammatory reaction that leads to unpleasant clinical reactions, the most severe of which is anaphylaxis, which can be fatal. Existing twin and family studies have demonstrated that genetic make-up may significantly influence the emergence of food allergies. According to these investigations, genetic variations account for 15% to 35% of the reported individual variations in food-specific IgE. According to twin studies, monozygotic twins had higher concordance rates for peanut allergy sensitization than dizygotic twins. It was discovered that the heritability estimate for peanut allergy was between 82% and 87%, illustrating the importance of genetic influence because monozygotic twins were more likely to have a phenotype that was comparable than those with dissimilar genes. When compared to Asian countries, the prevalence of food allergy among newborns and young children under the age of five appears to be higher in Western nations compared to both Caucasian and Asian children born in Australia; Australian-born children of Asian parents have a higher rate of food allergy.

This shows that early environmental exposures may have a different impact on the influence of genetic predisposition on food allergy. The search for genes linked to food allergies has been conducted using both candidate genes and Genome-Wide Association Studies (GWAS). More and more GWAS are being conducted with a focus on peanut allergy and "any food allergy" results, discovering novel genes linked to these allergies. Yet, the majority of the populations in these study were Caucasian or European. Immune-related genes thought to have a role in the pathways behind food allergies have been the focus of candidate gene investigations. There has also been research to look at genes

previously linked to other allergic illnesses for a connection with food allergy given that asthma, allergic rhinitis, and eczema share genetic risk factors.

The genetic underpinnings of food allergy, however, are still less understood in comparison to other allergic disorders. This systematic review's main goal was to investigate the evidence supporting a link between genetic variants and food allergies and to pinpoint any gaps in our understanding. There hasn't been a thorough assessment of the hereditary factors that contribute to food allergies. Thus, we conducted a thorough evaluation of the literature on the genetic causes of food allergies. The genetic association studies of food allergies are being thoroughly compiled for the first time in this review. Overall, studies were of varying quality, and there was little consistency in the results for the same SNPs. Given that research on genetic associations in food allergies is still in its infancy, this is not particularly surprising. While some discovery studies omitted a replication phase, it is encouraging to see that more current research is beginning to acknowledge the value of replication in order to reduce the publication of false-positive results. Eight papers that were published over the last three years, all of which included a replication analysis, with the exception of two studies that were published in 2016. The majority of studies also included a statistical adjustment for population heterogeneity, excluding mixed and other ethnic groups from their statistical analysis, and included ancestry informative indicators as genetically inferred ancestry or were noted as a study constraint. Several though, did not touch on the need for any kind of population adjustment. Genetic research must evaluate population stratification since any detected allelic or genotypic frequencies may be connected with ethnicity rather than the outcome of the disease. Multiple testing adjustments are equally important to population stratification because the absence of multiple corrections may result in false-positive relationships with food allergies.

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